Chapter 16

Interpersonal Trauma

Everything can be acquired in solitude—except sanity.

—Friedrich Nietzsche

Our brains rely on other brains to remain healthy, especially under stress. When faced with illness, catastrophe, or loss, we turn to each other for comfort, regulation, and stability. Resiliency—our ability to cope with life's ups and downs—is closely tied to the extent and quality of our support systems. We appear to be capable of coping with just about anything when we are connected to those for whom we care and who care for us. But what happens when we experience trauma at the hands of those we love and depend on? Interpersonal trauma is an especially difficult challenge, particularly for children, because it creates competing responses within them: They simultaneously want to cling for support and push away for protection. It is precisely this approach- avoidance conflict that can make interpersonal trauma so damaging.

Learning to hold ourselves away from others cuts us off from the beneficial effects of relationships we need to assimilate trauma and move on with our lives. Thus, interpersonal trauma is more likely to be self-perpetuating and resistant to healing. When interpersonal trauma occurs early in life, this approach-avoidance conflict can become a consistent state of being around which our personalities are formed. We can witness this inner conflict in the behavior of children with frightened and frightening parents. When these children are stressed, they run toward

their parent while simultaneously averting their gaze, fall to the floor, or engage in other types of apparently irrational behavior. For a child, the experience of simultaneous impulses to run toward and away from a parent is crazy-making. Adults who find themselves in relationships that include physical and emotional abuse often turn to drugs and alcohol, become physically ill, or develop severe psychological problems while they struggle to stay close to those who dysregulate them.

Early interpersonal trauma in the form of emotional and physical abuse, sexual abuse, and neglect shape the structure and functioning of the brain in ways that negatively affect all stages of social, emotional, and intellectual development. Early trauma, especially at the hands of caretakers, begins a cascade of effects that result in a complex posttraumatic reaction. The effects often manifest in what we call personality disorders, which impact many aspects of a person's functioning and are resistant to change.

The tenacity of personality disorders rests in an approach–avoidance conflict. If you need to feel connected in order to heal but are too afraid to trust because you become fearful and dysregulated in relationships, you are stuck. This "Catch-22" keeps many people in a constant cycle of loneliness→approach→terror→avoidance→loneliness, and so on. Many people come to therapy for years but are too afraid to trust their therapists enough to share themselves openly. They desperately need to establish a relationship in order to heal, but their fears overwhelm them and they flee back into a safe isolation.

This basic biological principle is seen in cold-blooded animals that regulate their body temperature by changing locations. They lack the inner mechanisms that regulate body temperature so they are forced to use actions to survive. When we lack the ability to regulate our internal emotional state, we attempt to regulate by "acting out" or through geographical solutions. When we can't move away physically, we learn to dissociate our reality from consciousness. For example, abused children learn not to look at faces and are less skillful in decoding facial expressions (Camras et al., 1983). When they do look at faces, they are hypervigilant to any sign of negativity or criticism.

In the face of early interpersonal trauma, all of the systems of the social brain become shaped for offensive and defensive purposes. Instead

of the social synapse being used for free and flexible exchange of information between individuals, it becomes a "no-man's land" fraught with difficulties and dangers. We stare at others trying to predict when they will become dangerous and require us to fight or flee. Mirror systems are employed to defend instead of cooperate; attachment schemas are used as battle plans instead of ways of connecting. Faces are explored for signs of disapproval. Regulatory systems become biased toward arousal and fear, priming our bodies to sacrifice well-being in order to stay on full alert at all times. Reward systems designed to make us feel good through contact with loved ones are manipulated with drugs, alcohol, compulsive behaviors, and self-harm. When the brain is shaped in this way, social life is converted from a source of nurturance into a mine field.

Child Abuse and Neglect

Abused children have a high rate of disorganized attachment, demonstrate higher resting levels of stress hormones, and have strong physiological reactions in response to brief separations from their mothers (Carlson et al., 1989; Hertsgaard et al., 1995; Spangler & Grossman, 1993). By far, the most disturbed children examined in attachment research are those rated as having disorganized attachment. These children usually have mothers who suffer from unresolved trauma, passing it along through their actions and reactions. A powerful example of this intergenerational traumatic transfer is shown in the children of Holocaust survivors, who come to share their parents' trauma-based biochemistry in the absence of having any traumatic experiences themselves (Yehuda & Siever, 1997; Yehuda et al., 2000). A child with a traumatizing mother has no choice but to stay with, and depend upon, the source of the trauma. The safe haven is now a source of fear and emotional dysregulation, creating a new generation of victims.

The ability to link feelings and words does not come automatically but relies on relationships to build connections between separate neural networks dedicated to affect and language. A child needs to experience parents who connect their own emotions and thoughts and can help them do the same by asking questions such as, "Are you sad because Grandma

is leaving?" or "Are you worried that we won't get to school on time?" Questions such as these guide a child's attention to his or her inner thoughts and feelings. Parental concern and curiosity make children aware that they have an inner experience of their own and it can be different from what others may think and feel. Because this inner experience can be understood, discussed, and organized through a coconstructed narrative, it becomes available for conscious consideration. As one of my young clients once said, "Talking about stuff makes it kinda real."

Language, in combination with emotional attunement, creates the opportunity to support neural growth and network integration. When a child is left in silence due to parental inability to verbalize internal experience, the child does not develop the capacity to understand and manage his or her world. The ability of language to integrate neural structures and organize experience at a conscious level is mostly unavailable. When children are abused and neglected, both neural growth and integration are impaired.

Over the last decade, we have learned that multiple structures and communication networks within the developing brain are impacted by abuse and neglect. Most striking are the abnormal development of experience-dependent structures such as the cerebral cortex, corpus callosum, and hippocampus. These abnormalities are likely the result of the negative biochemical effects of severe and prolonged early stress combined with inadequate stimulation and regulation, required by the developing social brain. Table 16.1 summarizes some of these findings.

Parent–child talk in the context of emotional attunement provides the ground for the coconstruction of narratives. When verbal interactions include references to sensations, feelings, behaviors, and thoughts, they provide a medium through which the child's brain is able to integrate the various aspects of experience in a coherent manner. The organization of autobiographical memory in a manner that includes processing from multiple neural networks enhances self-awareness, increases the ability to problem solve, and allows us to cope with stress and regulate affect.

As you might expect, there is a relationship between caretakers' own attachment history and their parenting abilities. This relationship was shown clearly in rhesus monkeys who were raised in isolation and

Table 16.1. **Abnormal Brain Development Due to Child Abuse/Neglect**

Abnormal cortical development	Perry & Pollard, 1997
Diminished corpus callosum size	De Bellis et al., 1999b; Teicher et al., 1997; Teicher et al., 2004
Diminished left-hemisphere development	Bremner et al., 1997; Ito et al., 1998
Diminished left hippocampal volume and development	Bremner et al., 1997; Ito et al., 1998; Vythilingam et al., 2002; Brambilla et al., 2004
Decreased right–left cortical integration	Schiffer et al., 1995; Teicher et al., 2002
Increased EEG abnormalities	Ito et al., 1993

demonstrated neglectful or atypical maternal behaviors (Champoux et al., 1992; Suomi, 1997). As with monkeys, human mothers with atypical and conflictive parenting styles usually have traumatic histories. However, research also suggests that adults can create secure attachment for their children, despite their own negative experiences as children. Earned autonomy, or the attainment of secure attachment later in life, results in the ability to develop secure attachments and serve as a safe haven for one's children. Thus, the powerful shaping experiences of childhood can be modified through subsequent personal relationships, psychotherapy, and self-awareness (Siegel & Hartzel, 2003). The ability to consciously process stressful and traumatic life events creates the possibility for positive change via the growth and integration of neural networks.

Shame

While most of us aren't physically or sexually abused during childhood, all of us experience shame. During the first year of life, parent–child interactions are mainly positive, affectionate, and playful. The limited skills and mobility of infants keeps them in close proximity to the caretaker, who provides for their many bodily and emotional needs. As the infant grows into a toddler, increasing motor abilities and exploratory drives confront caretakers with new challenges. A parent's role comes to include protecting toddlers from themselves as they run with abandon in whatever direction their impulses direct them. The positive parent–child interactions of the first year of life give way by the beginning of the second

year to an almost constant cry of "No!" (Rothbart et al., 1989). Thus, the affection and attunement that were experienced as unconditional during the first year become tied to appropriate *behavior* as parents try to teach children to inhibit dangerous and forbidden impulses. The same face-to-face interactions that stimulated excitement, exhilaration, and brain growth during the first year now include information on the recognition of disapproval and disappointment. This is the context of the emergence of shame.

At its heart, shame is the visceral experience of being shunned and expelled from social connectedness. Social exclusion is painful and even stimulates the same areas of our brains that become active when we experience physical pain. In small doses, shame can be useful in the development of conscience and a sense of social responsibility. Because shame is powerful, preverbal, and physiologically based, the overuse of shame can predispose children to problems with affective regulation and self-identity. Schore (1994) rightly differentiates shame from the later-occurring phenomenon of guilt. Guilt is a more complex, language-based, and less visceral reaction that exists in a broader psychosocial context.

Guilt is more closely related to unacceptable behaviors whereas shame is an emotion about the self that is internalized before the ability to distinguish between the action and the self is possible. You can take action to alleviate guilt, but shame offers no redemption. At its core, shame is the emotional reaction to the loss of attunement with the caretaker. The power of shame comes from the experience of attunement as life sustaining, in part, because, for young primates, separation and rejection equal death. Prolonged and repeated shame states result in a physiological dysregulation that negatively impacts the development of networks of affective regulation and attachment circuitry.

The return from a state of shame to attunement with parents creates a return to a balance of autonomic functioning, supports affective regulation, and contributes to the gradual development of self-regulation. Repeated and rapid return from shame to attuned states consolidates into an expectation of positive outcomes during difficult social interactions. These repeated repairs are stored as visceral, sensory, motor, and emotional memories at all levels of the central nervous system, making the internalization of positive parenting a full-body experience.

Shame is represented physiologically in a rapid transition from a positive to negative affective state and from sympathetic to parasympathetic dominance. This shift is triggered by the expectation of attunement in a positive state, only to find disapproval and misattunement in the face of the caretaker (Schore, 1994). A person in a shame state looks downward, hangs his head and rounds his shoulders. This same state of submission is not unlike that of a dog when he hunches over, pulls his tail between his legs, and slinks away after a scolding for committing a canine faux pas. Similar postures reflect loss, helplessness, and submission in virtually all social animals.

Because shame is neurobiologically toxic for older infants, these early preverbal experiences can have lifelong effects. Prolonged shame states early in life can result in permanently dysregulated autonomic functioning and a heightened sense of vulnerability to others. When parents use shame as their primary socialization tool, children spend too much time feeling anxious, dysregulated, and fearing for their safety. When these children grow up, they can usually find criticism, rejection, and abandonment in every interaction. Their lives are marked by a chronic anxiety, exhaustion, depression, and a losing struggle to achieve perfection (Bradshaw, 1990).

Focus on the Hippocampus

Why discuss the hippocampus in a chapter on interpersonal trauma? Very simply, because research suggests that the hippocampus is extremely vulnerable to sustained stress (Benes, 1989; Geuze et al., 2005). The biological link between prolonged stress and hippocampal damage appears to be mediated via glucocorticoids such as cortisol. Glucocorticoids (GCs) are hormones secreted by the adrenal gland as part of the body's response to stress. Prolonged high levels of GCs result in dendritic degeneration, cell death, and inhibited hippocampal functioning (Sapolsky, 1987; Watanabe, Gould, & McEwen, 1992). GCs trigger hippocampal neurons to work harder and harder, until they eventually run out of energy, collapse, and die. Loss of volume in the hippocampus appears to be related to long-term, cumulative GC exposure (Sapolsky et al., 1990).

The quality and amount of maternal care, handling, and soothing touch stimulates the creation of GC receptors, allowing the hippocampus to downregulate GC exposure (Meaney et al., 1989; Plotsky & Meaney, 1993). The result is a decreased reaction to subsequent stress and greater protection of the hippocampus. If this is the case, one of the important biological effects of secure attachment would be to protect the hippocampus from stress. As we come to understand the processes that translate trauma into neurobiological structure, we will be able to move swiftly to treat individuals who have experienced—or are about to experience—trauma by blocking the impact of GCs (Cohen et al., 2002).

The hippocampus and related structures are essential for the encoding and storage of our explicit memory for spatial, semantic, and temporal information (Edelman, 1989; O'Keefe & Nadel, 1978; Selden, Everitt, Jarral, & Robbins, 1991; Zola-Morgan & Squire, 1990). The hippocampus is necessary for the consolidation and contextualization of new episodic and autobiographical learning (Eichenbaum, 1992; Squire, 1987). Severe damage to the hippocampus will result in anterograde amnesia, which is the inability to learn new information that is available to conscious awareness.

Because explicit memory requires networks involving the hippocampus and higher cortical structures, the development of conscious memory parallels the maturation of these systems over the first years of life (Fuster, 1996; Jacobs, van Praag, & Gage, 2000; McCarthy, 1995). Unconscious learning and conditioning that utilize alternate memory systems continue to occur, even though we have no conscious recollection of the experience. Take, for example, a fearful memory: Whereas the hippocampus is required for the conscious memory of the experience, the amygdala is necessary for the visceral response to fear (Williams, L. M. et al., 2001). The sight of a dog that once bit you might elicit a bodily fear response via the amygdala, but hippocampal damage would leave you with no conscious memory of *why* you were afraid.

The hippocampus and amygdala have an important relationship to one another. The amygdala has a central role in emotional and somatic experience, and the hippocampus participates in conscious, logical, and cooperative social functioning. Because both the emotional and cognitive components are vital to relationships, their proper development,

functioning, and reciprocal regulation are essential. Impairment of the hippocampus from early chronic stress can impact virtually every aspect of development. Decreases in hippocampal volume have been shown to correlate with deficits of encoding short-term into long-term memory (Bremner et al., 1993). Adult women who experienced childhood abuse have reduced left hippocampal volume and increased dissociative symptoms. This relationship suggests that the hippocampus plays a role not only in memory encoding but also in integrating our memories into a cohesive narrative about our past and personal identity (Stein et al., 1997).

Catherine—The Fabric of Life

Catherine was a woman in her late 30s who came to therapy complaining of anxiety and panic attacks. She was an accomplished writer who had published many successful novels, traveled widely, and lived an interesting and adventurous life. As she sat across from me, her posture overerect, never taking her eyes off of me, I became aware of how attentive she was to my every move, be it a shift in posture or reaching for my coffee. More than this, I noticed that her every hair was in place, her clothes were perfectly pressed, and each word was carefully chosen. Possessing none of these attributes myself, I smiled at the thought of what an interesting contrast we presented. Catherine made it clear from the outset that it was difficult for her to share her feelings and that she needed to proceed slowly. I assured her that I was in no hurry and that she could take all the time she needed. She decided to start by telling me about her family history, upbringing, and education.

The only child of an aristocratic English family, Catherine was sent off to a Swiss boarding school at the age of 6. She only saw her parents when they would meet at a resort for vacation or during an infrequent visit to their home in England. She was primarily raised by nannies, nurses, and tutors and had spent a good deal of time alone. She offhandedly mentioned that her parents didn't seem to like children, and that they couldn't wait to have her out of the house. She became interested in literature at an early age, and had loved to write "old-fashioned" stories in the style of Thomas Hardy. Even when Catherine discussed topics that she claimed

made her happy, she showed a noticeable lack of emotion. She acted like a disinterested reporter discussing the life of a stranger.

Over the next few weeks, Catherine described her many travels, her brief and superficial romantic relationships, the plots of some of her stories, and other facts of her life. She avoided talking of any of the things that had brought her to therapy, occasionally reminding me that she would get to them in her own time. Meanwhile, I began to notice something quite odd. From time to time, I found small pieces of material on the sofa, floor, or out in the waiting room. About 2 inches square, they appeared to have been cut out of some larger piece of cloth. I threw the first two away, but the third made me wonder whether someone was playing a joke on me, so I tucked it into my desk drawer. A few weeks went by as Catherine continued to provide me with factual information about her life. As she came in for one session, I held up one of the small pieces of cloth and asked her if she had ever seen anything like it. Abandoning her aristocratic stance, she lunged toward me, grabbed the material from my hand, and ran out of the room. I sat there stunned. She was gone before I could say anything. I looked down at my hand and saw that her nails had broken through my skin. What was the connection between Catherine and the fabric and why did she have such a strong reaction?

In the days that followed I left a number of messages for her, suggesting that she come in for a session so we could talk about what had happened. After avoiding me for a few weeks, she finally called back and we set up another session. Taking a seat across from me, she started out by apologizing for running away and said that she realized the time had come to talk about what was really bothering her. As it turned out, the small pieces of material were patches of a baby blanket that Catherine had carried with her for years. When she grew too old to carry her blanket, she carefully cut it into small pieces and carried those with her. She would always keep one in her pocket and rub it with her thumb and index finger to help remain calm. This had been a lifelong secret about which she was very ashamed. When she saw me holding a piece of her blanket, she felt completely exposed.

This disclosure helped us move forward into much darker childhood secrets. Her father had sexually molested her every time she came home from

boarding school. When she told her mother and asked for protection, her mother refused to believe her and told her she was only trying to make trouble. "From then on," Catherine said, "my mom always found a reason to keep me away from her and my dad." Catherine, desperate for help, approached one of the teachers at school. Again, she was rebuffed. Later, as a teenager, a female coach on whom she had a bit of a crush molested her. After they had sex, this coach also distanced herself from Catherine.

As with many children who are sexually molested by parents and teachers, Catherine felt deeply ashamed. In Catherine's case, the fact that her parents essentially abandoned her at such an early age left her feeling unloved and unlovable. She was overwhelmed by emotions and had no one to help her contain or process them. When she did ask for help, she was called a liar—another layer of victimization that compounded her shame. She had no place to feel safe, yet she longed for a relationship where she could discover what it would be like to trust someone. Through her novels, Catherine created worlds full of people who kept her company and of whom she was totally in control. Through our work, Catherine realized that she unconsciously tried to test me by dropping the pieces of cloth. She shared her fantasy that if she came to trust me, I would also molest her. Then she would lodge a complaint against me, but no one would believe her. After all, I was the "great doctor" and she was only a "crazy patient." Catherine had also created an even deeper fantasy that she would come to depend on me and I would send her away, banishing her to loneliness.

My primary job in therapy was to disconfirm her expectations. By being available, by listening, believing, and, most importantly, not exploiting or abandoning her, I created a safe haven for her to be able to become aware of and modify her interpersonal conditioning. Catherine had been abandoned and violated, a situation paired in her mind with anxiety, panic, and death. All she had to comfort herself for all of these years were squares of aging fabric. The countless nights she spent alone in a dark dormitory room in the Swiss mountains, overwhelmed with fear and loneliness, had imprinted her trauma deep into the wiring of her social brain. I explained to her the effects that this sort of conditioning has on neural systems related to fear, anxiety, and panic. Her symptoms

were no accident, nor were they a weakness of character. They were but a natural consequence of a child forced to grow up without loving parents in a frightening, overwhelming, and dangerous world. When your abuse comes at the hands of those who are supposed to protect and care for you, you truly feel adrift in an alien universe.

The depth of the harm caused by neglect, abuse, and inadequate nurturance rests on the fact that the human brain is a social organ. Relationships that cause pain teach children that their role in the group is tenuous, their existence is unnecessary, and their future survival is in question. Negative interpersonal experiences not only impact an individual's ability to relate to others, they also damage the body's ability to maintain and heal itself in response to physical illness and subsequent psychological stress. When stress is early and prolonged, neurons, neural structures such as the hippocampus, processes of immunological functioning, and the construction of the self can all be compromised. Raising a child is a daunting responsibility, one that includes building a brain that will last a lifetime—for better and for worse.

In the next four chapters we will look at different ways in which dysfunctions of social brain networks can result in well-recognized mental disorders. First we explore social phobia and borderline personality disorder to examine those who have a heightened sense of fear and shame when interacting with others. We then look at antisocial personality disorder and autism to examine two different ways in which a lack of social connection and attunement can manifest in human behavior. There is considerable debate concerning all four of these disorders over the relative influence of experience versus predetermined genetic programming that makes someone vulnerable to them. Although the answers to these questions remain to be discovered, what we do know about these social disorders can teach us much about the functions and dysfunctions of the social brain.

Chapter 17

Social Phobia: When Others Trigger Fear

Always expecting the worst, he was never disappointed for long. Never caught off guard either. All of which goes to show that . . . worrying works.
—Philip Roth, 1997, p. 385

Anxiety and fear are the conscious aspects of our body's ongoing appraisal of danger. They can be triggered by countless conscious or unconscious cues, and they have the power to shape our thoughts, behavior, and feelings. At their most adaptive, anxiety and fear encourage us to step back from the edge of a cliff, cross the street when unsavory characters are coming our way, or check to see if we signed our tax forms before sealing the envelope. At their least adaptive, they prevent us from taking appropriate risks, engaging in relationships, and exploring our world.

Just about everyone experiences some kind of social anxiety. A blind date, a job interview, or public speaking are all situations where it is normal to experience some level of anxiety. However, up to 12% of us experience social anxiety at some point in our lives that significantly impacts our functioning (Charney, 2004; Wittchen & Fehm, 2001). Social phobia appears to have both inheritable and learned components (Lieb et al., 2000; Mannuzza et al., 1995; Marcin & Nemeroff, 2003), and although a variety of differences in the brains of social phobics have been found, it is not known whether these differences are a cause, an effect, or simply correlates of social phobia (Li et al., 2001; Stein, 1998).

In this chapter we focus on ways in which our fear circuitry becomes activated in social situations. Keep in mind that what triggers social anxiety

is not a direct threat to our physical survival. Rather, it is usually related to the *anticipation of shame,* the social correlate of physical danger. As an infant, shame is an emotional disconnection with caretakers that is experienced as a threat to survival. The automatic and unconscious activation of shame continues to shape our self-image and social behavior into adulthood. For some, coping with shame and the anxiety it evokes is a crippling and lifelong struggle.

Samantha—In the Eyes of the Jury

Samantha, a woman in her 30s, came to therapy with the complaint that "my life is going nowhere!" At the beginning of our relationship, I had a difficult time following her. Her feelings, thoughts, and beliefs seemed so tenuous and vague that it was difficult to understand what she was saying. Even Samantha realized this, saying, "I don't know what to think, who I am, or what I'll do next." The one thing that seemed clear from her day-to-day life was that she was uncomfortable around people. Even though she insisted that she loved people, she did anything and everything to avoid them. The only people she felt safe and comfortable around were her immediate family and whatever man she was dating. She tried hard to go out into the world and do things, but usually something would trigger her to "get the hell home," and she would retreat to her bed and television.

Thinking back on Samantha, I'm still struck by her physical agitation, her furtive glances, and, most especially, her tendency to interpret nearly everything I said as criticism. There seemed to be no statement neutral enough to avoid making her defensive. If I asked her how she was feeling, her first response was,"Do you think I look bad?" When I commented on her arms and legs being crossed, her response was to apologize profusely, saying that she knew she had poor posture. She cancelled our initial appointment twice and when we finally did meet, I asked if she had been nervous about meeting me. Samantha apologized over and over again, pulled out her checkbook, and offered to pay for the two cancelled sessions. Her tension was contagious; I found myself growing increasingly hesitant to say anything to her.

My gaze made her uncomfortable. "People looking at me make me freeze. It feels like they can look right through me and see my thoughts. When I see someone looking at me, I begin to shake. Sometimes it feels like their eyes are hurting me. I'm like a scared rabbit; all I can think about is how to hop away." During high school, Samantha sat alone at a table in a far corner of the cafeteria. If someone sat near her, she would act as if she was finished eating, nervously gather up her things, and leave. She told me about a time when she had to make a presentation at work. She organized the chairs in the conference room, set up her materials, and put her first slide up on the screen. As everyone came in and seated themselves, she excused herself to go to the restroom. Samantha said that "I walked right out of the building, drove home, got in bed, and watched TV until I fell asleep that night. I just left them all staring at my first slide. I never even called to tell them what happened."

Even when seemingly relaxed, Samantha's anxiety always lurked just beneath the surface. "I have good people skills, " she told me, "and I'm good at fooling everyone, but they usually figure out that I'm stupid and have nothing to offer. I can tell by the way they look at me. If I say something and I see someone rolling their eyes, I just freeze and can't talk anymore. I can't tell you how many times I get up in the morning, get dressed, get in the car, and head for work. At some point during the ride, I just turn around and go back home. It's as if I'm possessed, like I'm not in control of my body. It just does what it wants to do." Unfortunately for Samantha, a steady supply of money from her family kept her from having to overcome these fears.

While growing up, Samantha's family never discussed any feelings except positive ones, as if putting words to negative feelings allowed them to become real. She remembered that when she was sad or afraid, her parents would tell her that she had nothing to worry about and the subject would be dropped. As a sensitive and shy young woman, this approach left her victimized by her own unspoken anxiety with no way of understanding or controlling it. All these years later, she experienced her emotions as alien forces that directed her actions.

The first focus of our treatment plan was to help Samantha become aware of her negative feelings and to describe them as clearly as she could. Once she was able to connect her inner emotional world with her conscious thoughts, we were able to explore and question them. It was

gratifying to be part of her growing ability to learn a language for her anxiety and fear, forming narratives about their origins, triggers, and ways of coping with them. During one session, we did a "psychological autopsy" on her flight from her presentation at work. Samantha came to realize that she hadn't run off because she was possessed by some alien force; rather, her anxiety had been triggered by noticing that someone had yawned. This yawn triggered shame and produced the thought that her audience was bored with her even before she began! This, in turn, led to a chain of thoughts and feelings that ended with the fantasy that she would be laughed at and humiliated—causing her to leave the room and not return.

Being shamed was, in fact, a common experience in Samantha's early life. Her father, an extremely successful businessman, had a sarcastic sense of humor that he often directed at his family. Samantha recalled many painful comments he made to her that made her flee to her bed in tears. Samantha came to see clearly how preparing to speak in front of her boss and coworkers created the same expectation of humiliation. She reacted the way she had as a child, retreating to her room and distracting herself with television, story books, or imaginary friends. This reflexive withdrawal, used as a defense to modulate anxiety as a child, continued seamlessly into adulthood. Sadly, without anyone to help her gain awareness of her emotions, her inner experience remained a painful mystery that resulted in her "going nowhere." Her thinking remained confused and vague, keeping her from focusing on the realities of her life.

Through the Eyes of a Social Phobic

Social anxiety disrupts our ability to have relationships through a variety of mechanisms. It alters how we gather and process information across the social synapse and how the networks of the social brain respond to experience. For example, social phobics have an attentional bias toward information that is socially evaluative in nature: They are biased toward anticipating, detecting, and remembering negative and angry responses by others (Amir, Foa, & Coles, 2000; Asmundson & Stein, 1994; Clark & McManus, 2002; Eastwood et al., 2005; Mogg et al., 2004; Musa & Lépine, 2000; Spector, Pecknold, & Libman, 2003; Straube et al., 2004).

They also tend to orient to anxiety-provoking stimuli and, once locked in, have a difficult time disengaging, even when it is irrelevant or harmful to their personal well-being (Amir et al., 2000; 2003; Fox et al., 2001; 2002; Georgiou et al., 2005; Yiend & Mathews, 2001).

Social phobics employ visual avoidance and elaborate processing strategies to modulate the fear evoked by being seen (Chen et al., 2002; Mansell et al., 1999; Van Ameringen et al., 1998; Wenzel & Holt, 2002; Wik et al., 1996). The combination of increased orientation to, and fixation on, social evaluation makes the experience of social phobics one of constantly being on trial: Every interaction is experienced as a negative evaluation of their worth. Thus, the memories and emotional impact of negative interpersonal responses cut deeper and last longer.

Children with social phobia have poor recognition of emotional facial expressions and experience greater anxiety while looking at faces (Simonian et al., 2001). On the other hand, socially phobic adults demonstrate a better memory for facial expressions than normal subjects, especially for those that are negative or critical (Foa et al., 2000; Lundh & Ost, 1996). It makes sense, then, that social phobics avoid looking directly at faces, especially eyes, preferring to focus on other aspects of those with whom they interact (Horley et al., 2003).

Turning away from others cuts down the amount of available information and results in the use of their own negative self-image when making self-judgments (Hirsch & Mathews, 2000; Hirsch et al., 2003). This is why social phobics come to conclusions faster in social situations—their social brains recycle old shame experiences and fail to gather new information. Based on their inner certainty of the inevitability of criticism and shame, they know that if they are "seen," they will be disliked. This interlocking set of thoughts, emotions, and behaviors maintains and reinforces their fear and avoidance of stress.

Orienting toward, and locking into, the negative expressions of others evolved due to its potential survival value. An angry look in our direction is a good indication that we need to pay increased attention to that person, and a frightened look in another direction suggests the presence of potential danger somewhere nearby. Social phobia, when not the result of sustained early stress or a specific traumatic experience, is most likely the result of an imbalance of the alarm

and feedback systems that regulate the approach and avoidance responses in social situations. This theory parallels our present understanding of obsessive–compulsive disorder, in which adaptive concerns about contamination and disorganization go unchecked, resulting in intense anxiety and disruptive rituals.

The Socially Phobic Brain

The triggering of these physiological and emotional reactions in response to other people leads to an experience of the social world as a dangerous place. Although people with social phobia may have an intellectual understanding that a situation is not dangerous, the power of their body's fear response makes it difficult for them to believe their own thoughts. When social phobics prepare to engage in public speaking, it is not surprising that they experience more anxiety and negative emotions than others without social phobia. They have an increased heart rate and right-sided electrical activation in anterior temporal, lateral frontal, and parietal lobes (Davidson, Marshall et al., 2000; Tillfors et al., 2002). Remember that right-sided bias correlates with emotional arousal and negative affect.

A right-hemispheric bias has also been found in other kinds of anxiety disorders (Nordahl et al., 1998; Rauch et al., 1995, 1997, 2003). Right-hemisphere activation has been shown to result in decreased metabolism in Broca's area, suggesting that the more anxious someone becomes, the more likely he or she will have difficulty speaking (Bruder et al., 2004; Rauch et al., 1997). This physiological pattern may explain why a fear of public speaking is particularly common. In fact, one group of researchers found that a combination of right prefrontal and left parietal activation was specific to social phobia, hypothesizing that this pattern of activation may be related to a unique interaction between the processing of autobiographical memory and social presentation (Bell at al., 1999). Perhaps such neural wiring makes it difficult or even impossible to overcome self-consciousness and the expectation of negative evaluation about the self. This theory is still highly speculative and we cannot yet say with any certainty that social phobia demonstrates a unique pattern of brain activation (Malizia et al., 1997).

For the most part, social phobics share the neurochemical profiles of those with other anxiety disorders: heightened (adrenergic) excitatory neurochemistry combined with decreased gamma-aminobutyric acid (GABA) inhibitory regulation (Argyropoulos et al., 2001; Bell et al., 1999; Papp et al., 1988). What distinguishes social phobia from other anxiety disorders is evidence of dysfunction in serotonin and dopamine systems (Nutt et al., 1998; Potts et al., 1996). Lower dopamine (D2) binding has been found in the striatum of social phobics, which in animals correlates with a lack of exploration and novelty seeking and lower social status (Schneider et al., 2000; Tiihonen et al., 1997). Remember, dopamine is an important neurochemical in the social reward system, related to approaching others, sustaining attachment, and creating a sense of well-being.

As with so many other findings, we don't know if biological differences are a cause or effect of social phobia. They could reflect an innate inability to compete socially or an experience-dependent shutdown of activity in the face of anxiety and shame. When parents dominate children through physical or psychological punishment, some young brains may be shaped to experience persistently lower social rank. The symptoms of hypervigilance and avoidance seen in social phobia may be an evolutionary continuation of the behavior of lower-ranking individuals in primate groups who "know their place" and stay in it. It would be an interesting and sad social reality if those of lower rank in the world came home and "took it out" on their children, only to create children shaped to remain of lower social rank.

The Role of the Amygdala in Anxiety and Fear

From an evolutionary perspective, many of our complex neural systems have evolved in the service of keeping us safe. The amygdala plays a central role in the evaluation and expression of fear and safety and has been conserved and expanded during evolution to accommodate increasingly complex cognitive, sensory, and emotional input into survival decisions. Its central role in appraisal and the triggering of the biochemical cascade of the fight/flight response involve it in networks of memory, affective regulation, and social relatedness. Electrical stimulation of the central

nucleus of the amygdala results in the experience of fear, whereas destruction of the amygdala results in an inability to acquire conditioned fear and possibly an elimination of fear reactions altogether (Carvey, 1998). It appears that the amygdala is indispensable for the association of sensory experiences with the fear response (Bechara et al., 1995).

As the center of our fear circuitry, the amygdala is capable of learning to pair any thought, feeling, or sensation with a fear reaction. Under the proper conditions, we can have a conditioned fear response to everything from lions, tigers, and bears to success, attachment, and even love. Social phobia, if nothing else, is a conditioned fear of social interactions that produces activation of the amygdala. Projections from the amygdala to the lateral hypothalamus result in sympathetic activation responsible for increased heart rate and blood pressure, and the amygdala's connection to the trigeminal facial motor nerve causes the facial expressions we identify with fear (Davis, 1992).

Although the amygdala has been the focus of intense study for many decades, another structure has attracted increasing attention in the neuroanatomy of anxiety. The *bed nucleus of the stria terminalis* (BNST) is a structure that is separate from the amygdala but appears to project to most of the same neural targets. Unlike the amygdala, it does not activate a fight/flight response to specific stimuli but results in a lower level of diffuse arousal we think of as anxiety (Davis et al., 1997). Whereas the amygdala is the center of fear conditioning to specific sensory cues, the BNST appears to be involved in activating a sense of concern over a longer period of time. Perhaps this is the structure responsible for a sense of foreboding, long-term anxiety, and making some of us "worry warts."

The BNST may be a later evolving companion structure to the amygdala, allowing us a longer event horizon for the prediction of trouble than the amygdala. The amygdala has been viewed as the center of both fear and anxiety. When we discuss the amygdala in humans, however, we may, in fact, be discussing the functions of both the amygdala and the BNST. Figure 17.1 shows just a few of the target areas that are activated by both of these structures.

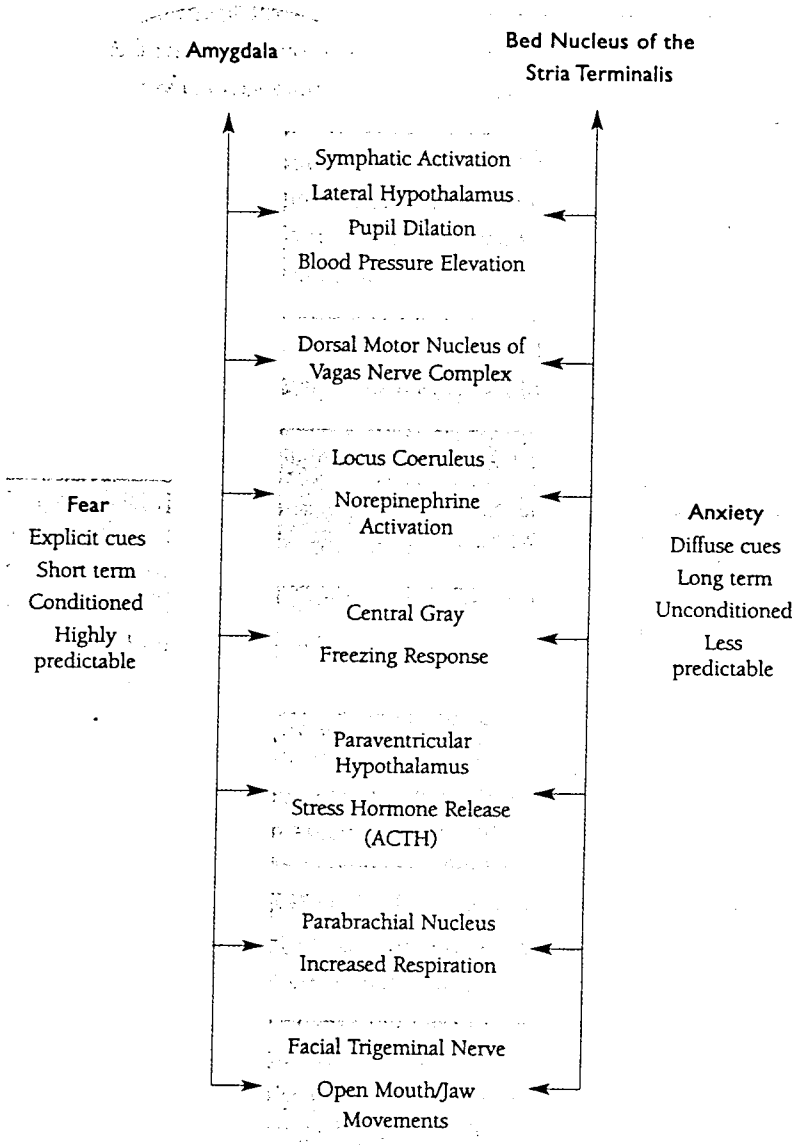

Figure 17.1. The amygdala and the bed nucleus of the stria terminalis: Target sites in the regulation of fear and anxiety. Adapted from Davis et al. (1997).

The prime directive of the amygdala is to protect us at all costs, and although it's good at its job, its successes come with some considerable downsides. The amygdala is stealthy in its appraisal, assessing situations based on past learning in small fractions of a second, way before conscious awareness. The tendency of the amygdala toward generalization, however, results in the triggering of panic by an ever-increasing number of internal and external cues (Douglas & Pribram, 1966). Aaron Beck put it best when he suggested that evolution favors anxious genes (Beck et al., 1990, p. 4). Unfortunately, that anxiety leaves us vulnerable to thoughts and feelings that are unnecessary and often destructive. Table 17.1 summarizes characteristic functionings of the amygdala.

Table 17.1. **The Amygdala**

- Appraises inner and outer environments much faster than conscious awareness.
- Does not require conscious awareness.
- Is well-formed before birth.
- Retains early learning and unconsciously weaves it into ongoing experience.
- Resists inhibition.
- Tends to generalize from specific instances.

The development and connectivity of the amygdala have many implications for both early child development and psychotherapy. Because the amygdala is operational at birth, the experience of fear may be the strongest early emotion. Without the inhibitory impact of the hippocampal–cortical networks, early fear experiences are likely to be overwhelming. Part of the power of early emotional learning may come from the intensity of these uninhibited and unregulated negative affects in shaping neural infrastructure. The infant is very dependent on caretakers to modulate these powerful experiences. What we call attachment schema reflect the power of early interpersonal experiences to condition the brain to approach or avoid other people as a way to regulate fear.

Amygdala dysfunction has been tied to all forms of anxiety disorders (Shekhar et al., 2003). Social phobics show greater amygdala activation during aversive conditioning to neutral faces and even greater activation in response to angry and contemptuous faces (Schneider et al., 1999; Stein et al., 2002). The amygdala is also essential in the reading of fearful facial

expressions; it may be that a hyperactive amygdala leads to sustained interpersonal vigilance and an overestimation of danger (Baird et al., 1999; Schwartz et al., 2003). When treated successfully with either medication or psychotherapy, a decrease in fear correlates with a decline in amygdala activation (Furmark et al., 2002).

One of the primary roles of the amygdala is to act as a social brake against random approach, inhibiting interaction with unfamiliar others until their safety can be evaluated (Amaral, 2002; Amaral et al., 2003b). In fact, it has been discovered that cells in the amygdala of macaque monkeys are selectively sensitive to approach movements (Brothers et al., 1990). When the amygdalas of macaque monkeys are destroyed, they begin to approach objects they should fear, including monkeys of higher rank. Human subjects whose brains were electrically stimulated in the region of the amygdala reported a sense of being reprimanded by an authority or of being an unwanted participant in a social group (Gloor, 1986). This finding certainly suggests that an emotional awareness of being where you are unwanted exists at a primitive level of processing and induces feelings of anxiety and shame.

The Fast and the Slow

In order to understand fear, the amygdala needs to be placed in the broader context of the human experience of fear. The response to, and evaluation of, fear involves two interconnected systems with different functions (LeDoux, 1996). So far, we have been discussing the fast system. With the amygdala at its core, this system makes rapid, reflexive, and unconscious decisions to provide for immediate survival reactions. This system develops first in the child and organizes learning related to attachment and affective regulation. The fast system sends information immediately and directly from the sense organs (eyes, ears, skin, tongue) through the thalamus to the amygdala. The amygdala then translates this "danger" information into bodily responses related to fight/flight reactions via its connections to the various structures described in Figure 17.1.

The slow system, involving the hippocampus and cortex, contextualizes and makes conscious what is being experienced. Its job is to balance

the reflexive activation of the amygdala with inhibitory input after the stimulus is judged to be safe. The slow system sends sensory information from the thalamus to hippocampal and cortical circuits for more detailed evaluation. This system is slower because it contains many more synaptic connections and involves complex conscious processing. Cortical circuits of memory and executive processing examine the information, compare it to memories of similar situations, and make voluntary decisions concerning how to proceed. This slow system in humans has the additional task of making sense of the behavioral and visceral reaction already set into motion by the fast systems. Figure 17.2 juxtaposes the fast and slow fear circuitry.

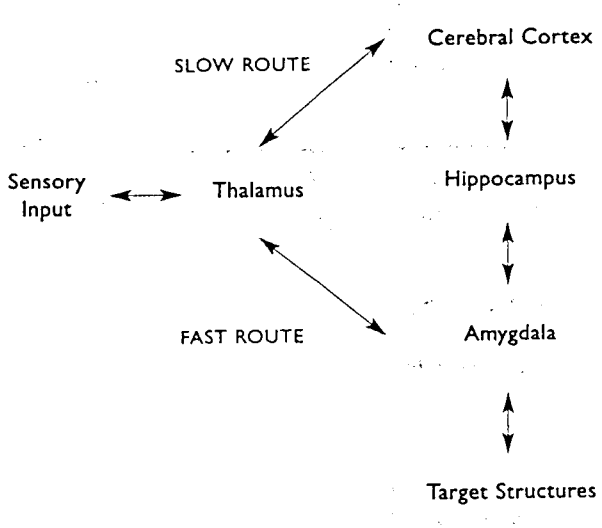

Figure 17.2. The fast and slow networks of fear processing. Based on Ledoux (1994).

From moment-to-moment, our fast system (organized by past learning) shapes the nature of our present experience. This is a key mechanism through which early social learning and attachment schema shape our experience. By the time we become conscious of others, our brains have already organized ways to think about them. At one extreme, "love at first sight" involves the triggering of positive associations and projection of them onto another person, thereby creating a sort of positive prejudice. In

the opposite case, as in situations of racial prejudice, salient aspects of a person's skin color trigger the fast system, which unconsciously shapes our experience as based in fear and other negative feelings.

Racism as a Form of Social Phobia

History is plagued with examples of racism, hate crimes, and "ethnic cleansing." An objective study of racism is difficult because emotions run high and usually cloud our judgement. And *why* do emotions run high? What would make hundreds of European-American adults threaten the lives of a handful of African-American children walking into a schoolhouse during the early days of integration?

What would allow concentration camp guards to order millions of innocent people into gas chambers? The cause of our inhumanity to others is an enduring and vital question. Racism seems so endemic in human history that it may not be "inhuman" but rather "all too human." On the other hand, countless examples of interracial cooperation and harmony seem to contradict the notion that racism is a basic human truth.

As we project our imaginations back to the prehistoric bands of our ancestors, we can easily see the benefit of quickly spotting unfamiliar others. Those from another tribe were potentially dangerous and not to be trusted. If this scenario of natural selection is true, it would make sense that the neurobiology of racism would be related to the fear circuitry in the brain. So is racism hard-wired into our brains, learned, or some combination of both? A number of studies have examined brain activation during exposure to pictures of same/other group faces in an attempt to identify the neural substrate of prejudice. (Because these studies have been conducted at U.S. universities, and our most salient racial struggle is between African-Americans and European-Americans, the studies reviewed here focus on these two populations.)

Recognition memory is superior for faces of same-race individuals and greater activation occurs in brain areas involved in the recognition (left fusiform cortex and right hippocampal area) for faces of our own race (Brigham & Barkowitz, 1978; Malpass & Kravitz, 1969). In these studies, African-American participants were better at recognizing European-American faces than European-Americans were at recognizing

African-American faces. This difference is likely due to greater exposure of African-Americans to European-American faces in the media and in most college environments (Golby, Gabrieli, Chiao, & Eberhardt, 2001). When European-American subjects are shown African-American faces, they demonstrate more left amygdala activation and enhanced startle response (Phelps et al., 2000). For African-American and European-American subjects, there is greater amygdala habituation to same-race versus other-race faces (Hart et al., 2000).

Because neither conscious awareness nor attention are required to condition a fear response, the body's reaction to individuals of other races is unrelated to the individuals' conscious experience or stated attitudes. Consistent with these neuroanatomical findings, social psychologists have found that stereotypes are automatically activated and require conscious inhibition to produce low-prejudice responses (Devine, 1989). Increased implicit prejudice is related to increased sensitivity to facial expression (Hugenberg & Bodenhausen, 2004). These emotional–visceral activations may relate to findings from other studies that suggest that other-race speakers are seen as more forceful or powerful than same-race speakers (Hart & Morry, 1996, 1997; Hass et al., 1991; Linville & Jones, 1980). In fact, anger results in greater expressions of prejudice even when the anger has nothing to do with a member of the other race, suggesting that negative affect can prime or activate prejudice (DeSteno et al., 2004).

Research with African-American children dating back to 1950 showed that they would choose white dolls over brown ones. When asked why, they described the brown dolls as "ugly," "not pretty," or labeled them with racial epithets. White dolls were described as "good," "pretty," and "the best looking because they're white" (Clark & Clark, 1950, p. 348). Given that the self emerges from relationships, cultural prejudice becomes woven into the core of the developing psyche. What a sad state of affairs.

So back to our question about whether prejudice is learned or innate. To explore this question, Phelps and her colleagues (Phelps et al., 2000) showed pictures of well-known and well-liked African-Americans to European-American subjects. These subjects demonstrated the same pattern of results as in previous studies except that amygdala activation was absent! These results suggest that exposure to, and knowledge about, a person of a different race can teach the brain to be less afraid.

Although the interpretation of these results has been contested (Dasgupta et al., 2000), it does make intuitive sense that positive exposure is capable of inhibiting the activation of fear circuitry. For European-Americans, amygdala activation may be reinforced by the consistent pairing of African-American faces with criminal behavior on the nightly news. The pairing of these images with criminal behavior may not have the same impact on African-American viewers because of their increased ability to differentiate same-race faces in conjunction with greater general exposure. For African Americans, real and suspected racial prejudice may consistently reinforce the brain to remain vigilant and suspicious of European-Americans.

Segregation and other forms of prejudice embedded in culture may keep us from becoming experts about the faces of those from other groups. Segregation may shape our brains to analyze the faces of people from other races using less efficient alternative circuitry, thus making us more vulnerable to cultural stereotypes and media representations. Like the social phobic, we may all employ avoidance strategies to modulate our anxiety with those of other races. We know from studies of affect regulation that labeling activates neural networks (dorsal lateral prefrontal/hippocampal) that inhibit emotional reactivity. So it may be that prejudicial labeling of others decreases our anxiety and creates the illusion of control. The lessons of history appear to support this interpretation.

Prejudice is so automatic and unconscious that its manifestations need to be continually uncovered right before our eyes. It does seem clear, however, that once stereotypical attitudes and beliefs are inculcated, change requires intention, attention, and time (Devine, 1989; Phelps & Thomas, 2003). People from other cultures, with different degrees of exposure, proximity, and prejudice, need to be studied before we can arrive at any conclusions about the origins of racial prejudice in the human brain. With the science of genetics revealing that race is more of a cultural construct than a biological reality, future study of how our brains react to one another can help to shed light not just on the nature and etiology of the neuroscience of prejudice, but also on how all of us who live on this increasingly crowded planet may learn to overcome our differences and discover the true wealth of human diversity.