

The Frustrating No-Man's-Land of Borderline Personality Disorder

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Psychiatrists Larry J. Siever and Harold W. Koenigsberg argue that the complexity of borderline personality disorder may stem from the interaction among genetic vulnerabilities (such as extremes of temperament), early experiences, and vast differences in patients' coping patterns. Patients must be held responsible, they argue, but so must the mental health professionals whose role is to understand and help them.

For the young psychiatrist in training, the term “borderline personality disorder” conjures up images of that angry young woman who regularly calls the emergency room at midnight, telling him that she has swallowed rat poison but refusing to reveal her name or whereabouts.

For the boyfriend of the young woman who reacts to their arguments by slashing her arms, the term sums up a series of perplexing, profoundly disturbing behaviors. For the wife of the real estate developer, it evokes images of her husband's angry tirades after an evening of heavy drinking with his cronies.

For the person suffering from the disorder, the term may epitomize the bewilderment, bitterness, and sense of helplessness at the swirl of shifting emotions and insistent impulses that roil daily life.

Ask even the experts about borderline personality disorder and you will get an array of theories and interpretations different enough to remind you of the proverbial blind men examining the elephant, each convinced that a part is the whole. The psychoanalyst will talk of “splitting” and distorted “object relations,” the cognitive behaviorist of “faulty schema” and “an invalidating environment.” The psychopharmacologist may refer to imbalances of brain chemicals such as serotonin and dopamine, and the sociologist to “identity diffusion” promoted by a culture rapidly losing its cohesive social norms. Probably they will agree only on certain observations of behavior: that the person with borderline personality disorder experiences rapidly shifting emotions, is highly reactive to surrounding events, and has a short fuse for irritability, anger, and impulsive behavior.

At a time when psychiatry is grounding one severe mental disorder after another in brain biology, borderline personality disorder confronts us with an enigma—and a clinical dilemma. We have little trouble understanding how a man with a tumor impinging on his frontal lobes may become irascible and display poor judgment, or how someone with an abnormal organization of her brain may hear voices and act out of touch with reality. But we resist seeing the moody, irritable, apparently manipulative and willful behavior of “borderlines” in terms of the biology of the brain; it seems to absolve them of responsibility for their aggressive, antisocial, or even outright criminal acts. Thus we may dismiss them as “impossible” without comprehending the extent of their inner turmoil and pain.

Partly for these reasons, many people, among them many mental health professionals, think borderline personality disorder is far less common than it really is. Primarily manifested in irritating behaviors rather than signs more commonly associated with mental illness, the disorder frequently goes undiagnosed or misdiagnosed. The prevalence of borderline personality disorder has not been established systematically, but estimates are on the order of 2 to 3 percent of the general population and more than 10 percent of psychiatric outpatients. One in ten people with the disorder commits suicide. People with borderline personality disorder are frequently treated for conditions—such as major depression, anorexia or bulimia, or substance abuse—that can coexist with it. Also, many people with the disorder are in nonclinical settings, such as prison. The disorder is implicated in other public health problems, such as domestic abuse and compulsive gambling, in addition to suicide and substance abuse.

THINKING IN TERMS OF VULNERABILITIES

One way to think about psychiatric disorders of this kind is as neurobiologic vulnerabilities. Just as each of us differs in hair color, height, or eye color, we differ in subtleties of brain structure and function. These differences are genetic in origin, but they are elaborated by early biologic influences (starting in the womb) and all the experiences that mold us as infants and children. The end result is our own particular disposition, ways of behaving, and patterns of coping that are called our personality.

Sometimes, however, these individual differences are extreme enough to lead to significant psychological and social problems. Then we begin to think of them as potential vulnerabilities. A person's consistently extreme emotional reactions to simple daily disappointments and frustrations may make rational coping seem impossible. Where differences in temperament are modest, they can be either an asset—for example, the sensibilities and emotional reactions of an artist or writer—or a liability, such as a tendency toward “emotional storms” that disrupt relationships or even the continuity of sense of self. For example, it is counterproductive consistently to react to frustration with aggression rather than reflection on how to respond. The person who speeds, gets intoxicated, plunges into a promiscuous relationship, or recklessly gambles to drown out painful, desperate feelings of abandonment following the loss of a relationship may find temporary relief but is getting into some serious long-term problems. What in a milder form was a propensity for assertive action has become, in these extreme forms, a serious vulnerability.

Before we examine the evidence for the origins of these dispositions in the biology of the brain, how brain biology may shape an individual's development (and be shaped by it), and the resulting complexities of treating the patient with borderline personality disorder, let us share a clinical vignette to illustrate the complexity that clinicians face in drawing the line between willful behavior and biologically determined vulnerability.

MELANIE

Two friends had to carry Melanie into the emergency room. She kept dozing off from the overdose of sleeping pills she had taken. The psychiatrist on call noticed bandages on her left arm that barely concealed dried blood. Her eyes were baggy, the lids droopy, her complexion pale.

Her friends had found her in her apartment, unconscious but able to be aroused, and figured out that she had overdosed several hours earlier. They said Melanie had broken up with her boyfriend, a man often abusive to her, the previous night. She had called each of them in tears, feeling desperate and abandoned; they made plans to meet for coffee the next morning. Her friends became alarmed when she did not show up and went to her apartment. Melanie was admitted to the hospital for observation and a brief stay.

The resident physician who admitted her heard her story the next day, when she was more alert. She looked rested. She was fully made up and even cheerful. He elicited a long history of self-destructive behaviors that included drugs and alcohol, suicide attempts, cutting herself, and outbursts of temper, particularly with boyfriends. Her father was an alcoholic; her mother had been depressed. Growing up, Melanie had been sexually abused by an uncle and verbally abused by her father. As an adult, she had had a series of relationships with men she initially idealized, but who inevitably abused her. She was often moody and had had several episodes of depression, but more prominent was her emotional volatility, rapidly shifting from feelings of abandonment to rage. Her outbursts of temper made her personal relationships stormy and interfered with her effectiveness as a public relations consultant, although she showed a flair for her work when she was not irritable and easily offended by colleagues or clients.

In her episodes of despair, usually after a relationship broke up, she would abuse sedatives and alcohol or behave promiscuously. She often ended up unconscious, sleeping off drug-induced somnolence until she had to get up the next day for work. On some of these occasions, overwhelmed with rage and self-hatred, she cut her arms with a razor blade until she felt a sense of relief. This was not the first time such behaviors had led to admission to the hospital.

Melanie had pursued many treatment options, but would inevitably become disillusioned and abruptly end treatment. She had seen several psychotherapists and, at one time, a psychiatrist who met with her twice a week. She explored her feelings about her parents and childhood experiences and examined her rage, which frequently was directed at her psychiatrist. Her feeling of being exploited and abused by the psychiatrist (for example, when he went away on his planned vacation at times she felt she needed him) seemed to echo her feelings about her father's abuse and neglect. While at times she could see that anger at her psychiatrist was a distortion, based on her past experiences, rage ultimately overwhelmed her and she left treatment.

She then sought the advice of a psychopharmacologist, who suggested she might have a rapid-cycling affective disorder because her intense emotions changed so frequently. He prescribed mood stabilizers, which she abandoned because of the weight gain they caused. Next she sought treatment in a day program that offered cognitive/behavioral therapy, but she soon found daily attendance too demanding and also disliked being in the company of people who had "serious mental illnesses." She tried outpatient psychotherapy again, but abandoned it when her therapist showed up five minutes late for a session. The next counselor felt that her problems arose from repressed memories of sexual abuse at the hands of her father, and spent sessions talking about her childhood traumas.

During this odyssey of treatments sampled and abandoned, Melanie heard seemingly discrepant explanations of her condition. Although the psychiatrist did not

offer a direct explanation, his comments seemed to suggest that she had difficulty separating from her mother, whom she experienced as being inconsistently available to her, leaving her feeling furious. He suggested that much of her behavior was intended to make other people experience the rage that she found unbearable. The psychopharmacologist explained that low serotonin levels might underlie her propensity to anger and aggression; he prescribed a selective serotonin reuptake inhibitor (SSRI), an antidepressant that made more serotonin available in the brain. He later prescribed a mood stabilizer that he explained might help with her irritability. The cognitive/behavioral therapist emphasized that her parents had not validated her feelings, contributing to her difficulty in regulating her emotions and developing interpersonal skills that might temper her impulses. The last counselor traced her problems to her early abuse and suggested that she talk through those experiences. This catalog of explanations left her depressed and disillusioned.

SEEKING A WHOLE ELEPHANT

How do we make sense of Melanie's symptoms? Does she have a brain disorder to be treated with medications? A disorder arising from faulty learning? Are its symptoms a direct consequence of the trauma or abuse many people with borderline personality disorder have experienced? Are these explanations mutually exclusive, or do they all contribute to a full understanding of her problem?

The circuitous history of the concept of borderline personality disorder reflects these complexities. In the 1940s and 1950s, the earliest diagnosis that employed the term "borderline" was "borderline schizophrenia," a diagnosis that located the patient's problem somewhere between chronic schizophrenia and normality. (Today people with these mild psychotic-like symptoms and the social withdrawal characteristic of schizophrenia are diagnosed with "schizotypal personality disorder.") The psychiatrist Roy Grinker referred to a "borderline syndrome," which included the emotional turmoil and impulsiveness that we associate with borderline personality disorder, but also the psychotic-like symptoms associated with schizotypal personality disorders. Otto Kernberg used the term "borderline organization" to describe a psychological organization somewhere between psychotic, with fundamental alterations in reality testing, and neurotic, characterized by conflict and anxiety more than the tendency to behave impulsively. John Gunderson and Margaret Singer tried to define "borderline personality disorder" more precisely in terms of specific interpersonal characteristics such as unstable relationships and behavior such as suicide attempts and self-injuring. Their definition eventually was adopted by the American Psychiatric Association, with some modifications, for their *Diagnostic and Statistical Manual-III* (DSM-III), the handbook of psychiatric diagnoses, in 1980. While the term "borderline" has been criticized for not clearly reflecting the actual specific behaviors associated with the disorder, it remains in wide clinical use.

The complex personalities of people with borderline personality disorder cannot be reduced to a single, simple formula. It is more useful to parse the disorder into its components. When we do so, we see vulnerabilities of temperament that may well be rooted in the variations being discovered in key brain systems that regulate emotions and aggression. These individual differences, underlying and influencing a person's development, go a long way toward explaining the disturbed behavior and altered

psychology associated with borderline personality disorder. Here we will examine the neurobiology of the two essential components of the disorder: impulsive aggression and affective (emotional) instability.

THE NEUROBIOLOGY OF IMPULSIVE AGGRESSION

Although the propensity to act without foresight in an irritable or aggressive way is not unique to borderline personality disorder, it is integral to it. Studies of identical and fraternal twins and adopted children show that this propensity may be inherited. The genetic potential may be triggered by parents or peers who act aggressively; conversely, it may fade in a more supportive, caring environment. The threshold for aggressive acts is more easily crossed in a person of highly changeable emotions and moods—the other essential characteristic of the borderline patient.

Brain systems that suppress aggressive or socially inappropriate behaviors may be less effective in people with borderline personality disorder. The level of serotonin in their brains is a good place to begin an investigation because serotonin is a “modulatory neurotransmitter”: a brain messenger-chemical that regulates emotion, feeding, temperature, and appetite and can suppress aggressive or antisocial behaviors. The analogue of these human behaviors in animals, such as rats’ aggression toward mice, makes these animals promising models for testing the modulatory effects of serotonin. Rats with lesions of the serotonin system display markedly increased aggression in behavior such as killing mice, compared to rats without the lesions. Furthermore these rats have a hard time suppressing behavior once it has been punished. They continue pressing a bar that had been associated with a reward (food pellets) even after the pressing produces a shock instead of a reward. Their problem is not with discriminating between the reward and the shock but rather with suppressing behavior that previously led to reward. It is tempting to extrapolate from animals to humans, but the vast differences between them precludes direct comparisons. What we need are clinical studies of impulsive, aggressive people.

One method of studying the function of serotonin in humans involves measuring a breakdown product (or metabolite) of serotonin, 5-hydroxyindoleacetic acid (5-HIAA), in the cerebrospinal fluid (CSF) that bathes the brain. The concentrations of this waste product of serotonin give us an idea of the activity of the serotonin system in the brain. Concentrations have been found to be low in patients who are depressed, particularly those who seriously attempt suicide. Concentrations have also been found to be low in violent criminal offenders and armed services personnel (and others) with histories of aggression. All this suggests the possibility that low serotonin activity may be associated more with aggression, whether directed against oneself or others, than with depression or suicide per se.

Measurements of CSF 5-HIAA, the serotonin breakdown product, cannot tell us the responsiveness of brain cells that are affected by serotonin, but another study uses chemical agents that release serotonin near its targets of action—the receptors—and then measures responses by these receptors, such as the blood levels of hormones whose secretion they control. For example, the chemical fenfluramine causes release of the hormone prolactin, and the degree of prolactin release following administration of fenfluramine may give us an index of the responsiveness or capacity of the person’s serotonergic system. Studies using this strategy suggest that the serotonin system’s

activity has been blunted in patients with borderline personality disorder compared to normal controls, or even patients with other personality disorders.

This blunting is associated with angry outbursts, impulsive behaviors, and self-destructive behaviors—that is, impulsive aggressive symptoms—rather than with emotional instability. Blunted prolactin responses to fenfluramine also correlated with suicide attempts (particularly serious ones, involving injury) in both personality disorder patients and depressed patients. Personality disorder patients who had attempted suicide and engaged in self-destructive behaviors showed the most blunted responses. This is consistent with the hypothesis that both suicide attempts involving direct physical violence toward oneself and self-destructive acts, such as cutting oneself or burning oneself, represent self-directed aggression. Blunted prolactin responses to fenfluramine were also associated with high irritability and aggressiveness, as reported directly by the people affected. This result has been replicated several times and observed with other chemical agents that test serotonin system activity.

Measuring hormone responses, however, cannot help identify the specific brain circuits modulated by serotonin that are involved in inhibiting or releasing aggression. Imaging techniques such as Positron Emission Tomography (PET) scanning offer the possibility of studying the serotonin response of brain regions believed to be involved in controlling impulsive behavior. PET measures the activity of radioactively tagged glucose molecules, producing a picture of metabolic activity throughout the brain. Thus changes in brain activity can be seen directly following administration of chemical agents that enhance serotonin activity. Two such agents, fenfluramine and chlorophenylpiperazine (mCPP), the latter acting directly on serotonin receptors, cause increases in metabolism in the cortex—the part of the brain responsible for higher cognitive function, including modulating or inhibiting more primitive aggressive and sexual urges. The front of the brain behind the forehead and just above the eyes (called the orbital frontal cortex) is of particular interest. Lesions here can result in less inhibition of aggression.

A perfect example was found in Phineas Gage, a mild-mannered 19th century railroad worker who was injured in a miraculously specific way that destroyed much of his orbital frontal cortex but left him otherwise functioning. After the injury, Gage underwent a marked personality change, becoming irascible and impulsive and displaying poor social judgment. This famous historical case is consistent with other reports of people with injuries or lesions in this area who develop poor social judgment and antisocial traits. It appears that the orbital frontal cortex plays an inhibitory role, serving as the “brakes” for limbic regions involved in generating aggression. Since this region is heavily modulated by serotonin, one might think of serotonin as the fluid that keeps these brakes working properly. When the fluid is low, the brakes malfunction and impulses toward aggression are not inhibited. Indeed, people with borderline personality disorder who are notably impulsive in their aggression do not show the normal increases in metabolism following serotonin agents that normal volunteers do.

We do not know what is responsible for individual differences in serotonin system activity, but the differences are likely to be partly genetic. (Remember, there is good evidence for heritability playing a role in impulsive aggression.) One approach to identifying genetic factors involved in a trait or disorder is to select candidate genes: that is, genes that are likely, based on other evidence, to be associated with that disorder. For

example, genes that modulate the breakdown or synthesis of serotonin might be logical candidate genes. Thus we find that the gene controlling the enzyme tryptophan hydroxylase, which is responsible for the rate at which serotonin is produced, has been associated with suicide attempts in criminal offenders and impulsive behavior in personality disorder patients. Another candidate is a variant of a gene that controls the serotonin transporter, which inactivates serotonin by taking it back from the cleft between the neurons (the synapse), where it does its job, to the inside of the neuron, where it is broken down. Genes coding for other receptors that act like thermostats in modulating serotonin release have also been associated with suicide attempts in personality disorder patients.

There is evidence that trauma or abuse may modify serotonin system activity. People with borderline personality disorder often have histories of sexual or physical abuse. While this experience is not unique to them, it may help shape their personalities and leave its imprint on the brain. The serotonin system itself may be modified by these traumas and, of course, this plays a critical role in developing brain systems related to habits and coping skills. Complex relationships have been found among responses to serotonergic agents, cortisol (a major stress hormone), and a history of trauma.

The relationship between serotonin activity and impulsive aggression raises the possibility that drugs enhancing the activity of the serotonin system could alleviate impulsive aggression. The SSRIs, such as fluoxetine (Prozac) or sertraline (Zoloft), increase concentrations of serotonin at the juncture between nerve cells. These medications have helped in depression, and there is increasing evidence that they may help in impulsive aggression as well. Studies suggest that they reduce irritability and anger in patients with borderline personality disorder. Indeed, the effects on anger are more pronounced than the effects on depression itself. Unfortunately, people with borderline personality disorder are often very sensitive to the side effects of these medications. This sensitivity, or the likelihood of their not complying with the requirements, has meant that they often do not give the medication an adequate chance to work. This is particularly problematic because people who have reduced serotonergic capacity appear to require more SSRIs than others to achieve therapeutic affects. If used carefully, however, with incremental increases in dose, SSRIs can be brought to levels that reduce impulsive aggression.

THE NEUROBIOLOGY OF AFFECTIVE INSTABILITY

In addition to vulnerability to impulsive aggression, people with borderline personality disorder are unusually emotionally reactive. They may be content for a while, then become intensely angry or hopelessly depressed or unbearably anxious—each state, although intense, lasting only a few hours or a day. Contrast this with classic mood disorders like depression, in which the emotion, although it may wax and wane during the day, endures for weeks or months. Even in bipolar disorder, or manic-depressive illness, which is defined by the often-rapid succession of depression and mania or euphoria, the different mood states typically last weeks or longer.

To those who are close to them, borderline patients appear to have random and unpredictable emotions. On closer investigation, those emotions often seem to involve heightened emotional reactions to other people. Borderline patients may become distraught at ordinary criticism, which they experience as a blow to self-esteem; may

react with rage to a disappointment or minor slight; or may feel terror at a separation that they experience as virtual abandonment. Their emotional, or affective, instability may contribute to their turbulent, often unstable relationships and the inconstancy in their experience of themselves that leads to a confused sense of identity.

Less is known about the brain biology of this instability than about the basis of impulsive aggression, but the borderline person's overreaction to frustration and disappointment seem to be part of a heightened reaction to almost everything. A particular chemical system of the brain, the norepinephrine system, appears to be involved in regulating our level of arousal and vigilance in reaction to the environment. Neurons that release norepinephrine arise from a structure deep in the brain stem called the locus coeruleus, which acts as the brain's alarm center, and spread out widely throughout the brain. Substances that stimulate norepinephrine activity increase alertness and attention to the environment.

To figure out whether the norepinephrine system is involved in the emotional ups and downs, scientists administered amphetamine, a stimulant that causes extra norepinephrine to be released from the neurons, to people with differing degrees of emotional instability. They found that those least emotionally stable were most sensitive to amphetamine-induced shifts in emotion.

A second chemical system in the brain, the acetylcholine system, also appears to play a role in emotional reactivity. When substances that enhance acetylcholine are given to patients with depressive illness, they become more depressed; when these agents are given to patients in the euphoric phase of bipolar illness, they become depressed, as well. Patients with borderline personality disorder who receive physostigmine, a substance that activates the acetylcholine system, swing to depression; those borderlines with a history of extreme affective instability show the strongest reaction. Procaine, the local anesthetic dentists use to diminish pain, also stimulates the acetylcholine system. When borderline patients receive procaine intravenously, they show marked and variable emotional reactions, especially swings to depression and other unpleasant feelings.

The brain has receptors that might almost have been tailored to fit minor tranquilizers such as diazepam (Valium) or lorazepam (Ativan), like a lock fits its key. Since the brain could not have evolved a receptor in anticipation of a drug product, this intriguing discovery suggests that the brain has its own natural Valium-like substance. We have not yet found the natural Valium, but researchers have identified a natural brain substance called gamma-aminobutyric acid, or GABA, which enhances operation of these receptors almost like oil lubricating the lock. GABA receptors are found extensively in those parts of the brain most involved in processing emotion, particularly the amygdala—an almond-shaped structure located deep behind the temples on each side of the head. Because GABA may play a role in tranquilizing or damping down sudden surges of emotion, it seems possible that impairments in the GABA system may be involved in affective instability. One confirmation is that three medicines that act as mood stabilizers in borderline patients—lithium, depakote, and carbamazepine—all enhance GABA activity.

We can use brain scanning to observe the activity of brain structures that may be involved in emotional instability. When volunteers get shots of procaine, the substance that evokes intense emotional reactions in borderline patients, their brain activity

increases in certain regions of the amygdala, suggesting that those regions may play a role in emotional instability.

BRAIN, PERSONALITY, AND BEHAVIOR

We have seen considerable evidence that improperly regulated brain systems may give rise to impulsive aggression and affective instability in borderline personality disorder. But because these traits are crucial in setting the tone and quality of human relationships, they inevitably become entwined with a person's psychology and social functioning. In this way, a predisposition created by the brain becomes an important influence in the developing personality and contributes to the characteristics of borderline personality disorder.

Infants who are very emotionally sensitive may respond more intensely to the comings and goings of their mother or caretakers and show much greater distress at separating. This may lead to a more insecure attachment between infant and mother. If the infant is more impulsive and aggressive—that is, likely to express emotions forcefully—he may have crying spells and, later, temper tantrums when frustrated or left alone, which can wear down even the most supportive parents and overwhelm those who are depressed or who themselves have trouble with emotional reactivity and impulsiveness. Parents may become frustrated at their inability to soothe such a child and decide not to respond to its distress; at other times they may try everything to indulge the child to appease its upset and rage. These inconsistent (and, to the infant, unpredictable) responses may make it likely that the child will learn to deal with unpredictability by means of emotional storms or tantrums.

As the child matures, he may draw on these interpersonal strategies in order to regain emotional equilibrium. For example, when an upsurge of depression follows a blow to self-esteem, the borderline person may try to bolster her self-esteem by devaluing someone else. When feeling alone and abandoned, she may behave recklessly to stimulate the worry and involvement of others. To onlookers, these behaviors may appear manipulative because their purpose is to bring another person to attend to the borderline's needs. But because of their heightened sensitivity to the availability of others, people with borderline personality disorder often feel that they are not in charge of their own emotions—their emotions depend on the behavior of those around them. Attempting to control their own feelings, they find themselves trying to control the behavior of people they depend upon and care about. Repeated again and again, these patterns of behavior become ingrained. The borderline person experiences these styles of relating as the only way to survive emotional ups and downs and the feeling that others cannot be trusted to support her.

People with borderline personality disorder translate their anger or disappointment into impulsive action that they have difficulty reflecting upon or delaying. Their sense of abandonment by the ending of a relationship may make them feel desperate and enraged. To make themselves feel better, they act in ultimately counterproductive ways, using drugs or alcohol to soothe upset feelings, plunging promiscuously into sexual activity, turning their anger at themselves in self-destructive acts like cutting their arms or wrists, or indulging in impulsive gambling or binge eating. These measures may temporarily alleviate their distress, but they will bring destructive long-term consequences. The same behaviors often lead mental health professionals to

“rescue” them by intervening with hospitalization, giving borderline people the attention they crave.

THE TIGHTROPE ACT OF TREATMENT

Only by looking at the behaviors of someone with borderline personality disorder in that person’s social milieu do we fully understand their meaning. For although temperamental vulnerabilities of impulsiveness and affective instability may drive these behaviors, the interpersonal environment can buffer or provoke them. Some of the most effective treatment approaches address the interpersonal and the temperamental domains in tandem.

Early in treatment, the person with borderline personality disorder must be helped to recognize his tendency to become disillusioned with people, drawing others into intense involvements only to push them away when they disappoint even slightly. This recognition is a crucial first step, because the pattern inevitably will develop in the relationship with the therapist, threatening to end it before it starts. Unless the person with borderline personality disorder can examine this pattern, he will be unable to sustain a treatment relationship and will not be open to efforts, through either medication or learning new strategies to diminish his temperamental vulnerabilities.

Once a treatment alliance is established, the individual can begin to take responsibility for his behavior. Medications may then help reduce impulsiveness and emotional instability. Behavioral therapies may also help, teaching skills that reduce vulnerabilities. Unfortunately, the maladaptive interpersonal patterns that the borderline develops to cope with temperamental vulnerabilities become ingrained and typically do not lessen when impulsiveness or emotional overreactivity begin to diminish. He must learn what his characteristic maladaptive patterns are, when they are likely to be brought into play, what purpose they serve, and how to substitute more adaptive coping strategies.

This is the domain of psychotherapy. Some people learn how to identify and modify their behavior patterns in cognitive-behavioral therapy, which uses a step-by-step analysis of the triggers of their maladaptive behaviors and provides training in new coping skills. Others learn how their behavior patterns emerge, what purposes they serve, and how to defuse them by searching for and exploring how they show up in their ongoing relationship with their psychotherapist (called a transference-based psychotherapy). Researchers are seeking to learn what forms of therapy best serve which individuals with borderline personality disorder and are developing new medicating strategies to address the underlying vulnerabilities.

For example, Melanie was finally able to find a therapist who treated her with a form of cognitive-behavioral therapy and introduced her to skills training as part of a special approach called Dialectical Behavioral Therapy. Melanie was started on a selective serotonin re-uptake inhibitor (like Prozac) and a mood stabilizer by a psychopharmacologist. While her life is still somewhat unsettled, Melanie has not overdosed again and has started a relationship with someone who seems to respect her.

Some of the most effective therapies may be interpersonal, while medications may raise the threshold beyond which aggressive behavior or upsetting emotions erupt, making psychotherapy more effective. To ignore differences in the biology of the brain that make the person with borderline personality disorder susceptible to emotional and interpersonal turmoil is to repeat the lack of validation that they experienced growing up.

To absolve people with borderline personality disorder from responsibility for managing these vulnerabilities, however, is to license them to indulge their maladaptive predispositions because “they can’t help it.”

We can look at the notion of vulnerabilities in the biology of the brain as being similar to the vulnerabilities predisposing a person to hypertension or diabetes. Diabetic or hypertensive patients are responsible for managing these vulnerabilities, just as people with borderline personality disorder can take responsibility for their behavior while acknowledging the struggles they will face in managing their turbulent emotions and precipitate actions. The power of the mind can be brought to bear on managing the brain.

Courtesy of Larry J. Siever, M.D., and Harold W. Koenigsberg, M.D
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