CHAPTER 1

Making the Diagnosis

The general definition of a personality disorder in Section II of DSM-5 (American Psychiatric Association, 2013) requires long-term dysfunction in mood, impulsivity, and cognition, significantly affecting functioning, with an onset early in life. (The alternative system in Section III of the manual is discussed in Chapter 2.)

Notably, DSM-5 has dropped the distinction between Axis I and Axis II, which was originally intended to encourage clinicians to think about personality disorders. But that approach, while well-meaning, backfired. Clinically important disorders like BPD were placed in an "Axis II ghetto" where they could safely be ignored.

BPD is associated with a wide range of symptoms, including chronically low and/or unstable mood, a wide range of impulsive behaviors, severe problems in intimate relationships, and micropsychotic episodes (Zanarini et al., 1998a, 1998b). It is classified as a personality disorder because it leads to serious long-term problems in interpersonal relations and work, as well as an abnormal sense of self). But unlike other personality disorders, BPD is only partially ego-syntonic, since it is associated with many troubling symptoms that lead patients to seek help (Paris, 2015b).

BPD is common in the community. Estimates of its prevalence have varied, but most studies find that it affects about 1–2% of the population (Lenzenweger, Lane, Loranger, & Kessler, 2007; Coid, Yang, Tyrer, Roberts, & Ullrich, 2006; Torgersen, Kringlen, & Cramer, 2001). One study, the National Epidemiologic Survey on Alcohol

and Related Conditions (NESARC; Grant et al., 2004), found a much higher prevalence (nearly 6%)—but this estimate, while widely quoted in the literature, was an outlier. When the data set was reanalyzed by applying a stricter barrier to diagnosis, the prevalence went down to 2.7% (Trull et al., 2010).

BPD is the most familiar category of personality disorder in clinical practice. Its clinical prevalence is much higher than its community prevalence, reflecting the intense help-seeking behavior that characterizes these patients. Zimmerman, Rothschild, and Chelminski (2005) found that about 9% of all outpatients in a large clinical sample met criteria for this disorder. A study in the United Kingdom (Newton-Howes et al., 2010) found a similar prevalence (8.5%). These estimates correspond to my own experience running a hospital clinic that provides consultations to primary care providers. It has also been shown that BPD can be identified in clinical settings in countries all over the world (Loranger et al., 1994).

To treat BPD, you first have to recognize it. And to recognize it, you have to know what to look for. Since many of its features (anxiety, depression, mood swings, impulsive behavior) overlap with those of other mental disorders, the diagnosis of BPD is often missed in practice. Zimmerman and Mattia (1999) found that only half of patients meeting DSM criteria for this disorder are diagnosed by clinicians in outpatient clinics.

There is a common belief that you can't diagnose a personality disorder in the hour typically devoted to an evaluation. That is not true. You just have to ask the right questions and make sure you have taken a good life history. If you don't get enough information, you may need to see the patient again, and/or interview a family member or key informant. But most of the time, it is not that difficult to determine whether a personality disorder is present, and, if so, whether the patient's pathology falls into the borderline category. Experienced clinicians can often make a diagnosis in much less than an hour. Another misconception is that you cannot diagnose a personality disorder when a patient is depressed. But as we will see, careful history taking allows clinicians to separate mood states from personality traits.

One problem with the diagnosis of BPD comes from the way the construct has been defined. Like most categories in psychiatry, it has fuzzy boundaries and lacks precision. However, these problems are no worse than those for major depression, which is an even more heterogeneous diagnosis (Parker & Manicavasagar, 2005). In fact, the DSM-5 field trials found that BPD had a higher reliability between clinicians than major depression (Regier et al., 2013).

Another challenge to the BPD diagnosis is the view that personality disorders should be dimensional constructs, that there is no boundary between normal and pathological traits, and that the scoring of trait profiles is more scientifically sound than categorical diagnosis (Hopwood et al., 2018). This point of view is discussed in Chapter 2, where I examine its advantages and disadvantages.

Another reason why the validity of BPD has been challenged is the very term *borderline*. It is a misnomer. No one thinks any more—as did the psychiatrist who first described the disorder, Adolf Stern (1938)—that this form of pathology lies on a border between neurosis and psychosis. Moreover, the term *borderline* fails to describe the most salient features of the syndrome: unstable mood, impulsivity, and unstable relationships. This vagueness has contributed to the tendency for BPD either to be seen as something else, or to be ignored.

I agree with most of the criticisms of using the term *borderline*. The problem is that we don't have a better term to describe this important disorder. Many proposals focus on one aspect (emotional dysregulation, impulsivity, or interpersonal relationships), but do not do justice to the complexity of the syndrome, which describes the interaction of all three of these domains. While there is no doubt that emotional dysregulation is the most important feature of BPD, this pattern can be seen in other disorders (Schore, 2003).

At this point, if we don't understand the mechanisms behind BPD, renaming it could be premature, and the suggestions for a replacement would run into most of the same problems. We may come up with a better answer in the future. But until we know more, we may as well continue to use this admittedly imprecise diagnostic label.

A BRIEF HISTORY OF THE BPD DIAGNOSIS

A historical perspective can help to understand these problems. Stern (1938), the first to describe BPD, observed that such patients often became worse—not better—in what was then considered standard therapy. As noted above, he suggested that this group was unsuitable for psychoanalytic treatment because their pathology lay on a "borderline" between neurosis and psychosis. Stern documented these clinical features ("psychic bleeding," inordinate hypersensitivity, difficulties in both reality testing and relationships), and his description is as relevant today as it was over 80 years ago.

Yet following this article, there was only sporadic interest in borderline pathology over the next 30 years. The one exception, a paper by Robert Knight (1953), added little to what Stern had said, and had little impact beyond the psychoanalytic community.

Three psychiatrists were responsible for reviving and popularizing the concept of BPD. The first was Otto Kernberg, a psychoanalyst who has worked at the Menninger Clinic and Cornell University. Kernberg (1970) proposed that character pathology (or what we now call personality disorder) has three levels: one milder (close to neurosis), one moderate, and one severe (i.e., borderline). But there were two problems with this concept of *borderline personality organization* (BPO). The first is that the construct was entirely psychoanalytical, in that it was defined on the basis of theories about mental mechanisms rather than on observable behaviors. The second was that BPO lacked consistent diagnostic criteria, defining a very broad group of patients with personality disorder as "borderline."

The second pioneer was Roy Grinker, working in Chicago. Grinker, Werble, and Drye (1968) published the first empirical study of patients with BPD, which gave more weight to clinical observation than to psychodynamic speculation, and which subgrouped patients on the basis of observable symptoms. His group also conducted the first systematic follow-up studies of such patients (see Chapter 7).

The third (and ultimately most influential) pioneer was John Gunderson at McLean Hospital. Gunderson and Singer's (1975) seminal article in the *American Journal of Psychiatry* was a turning point for the acceptance of BPD. It showed that this form of psychopathology could be operationalized with behavioral criteria, and that a semistructured interview yielded a reliable diagnosis that could distinguish BPD from "near-neighbor" diagnoses.

I have vivid memories of reading this paper. Up to that point, under the influence of some of my teachers, I had rejected the validity of BPD. The concept seemed all too vague, and I refused to let my residents use it. But Gunderson and Singer convinced me (and many others) that it was both valid and clinically meaningful.

The work of these pioneers influenced the definition of BPD adopted in DSM-III (American Psychiatric Association, 1980). The BPD diagnosis was included in the manual for the first time, and personality disorders as a whole were given a separate axis (Axis II) to encourage clinicians to think about them. As a result, research took off. In 1987, the International Society for the Study of Personality Disorders (ISSPD) was founded, and it has sponsored biennial

meetings ever since. ISSPD also sponsors the *Journal of Personality Disorders,* first published in 1988. Three other journals are devoted exclusively to personality disorders: *Personality and Mental Health; Personality Disorders: Theory, Research, and Treatment;* and *Borderline Personality Disorder and Emotion Dysregulation*. Since BPD is by far the most important clinical problem in this area, most of the research has focused on that category.

Yet in spite of much research, there continues to be controversy about the best way to classify BPD. The system in Section II of DSM-5 (American Psychiatric Association, 2013), unchanged from DSM-IV, is the best known. It lists nine criteria, of which five must be present to make a diagnosis. But this use of a simple majority of criteria has not been shown to be the most accurate way of identifying a coherent disorder. Let us examine the history of BPD diagnosis in more detail.

BPD IN THE DSM SYSTEM

BPD in DSM-III and DSM-IV

When I went to medical school, I was taught DSM-I. As a resident, I learned DSM-II. Neither of these systems had the influence of DSM-III (American Psychiatric Association, 1980). Here was a manual that revolutionized psychiatry. The use of observable criteria and algorithms for diagnoses in DSM-III was a great advance over imprecise paragraphs of description. The DSM system made it more likely that when clinicians placed people in a category, they were talking about the same patients. From this point on, psychiatric diagnoses were expected, at least in principle, to be reliable. Finally, DSM-III brought psychiatry back into the mainstream of medicine. However, whereas most medical diagnoses are rooted in biological measurements, the categories in DSM-III, almost entirely based on clinical observation, can only be considered provisional.

The main advantage of DSM-III should have been its promotion of diagnostic reliability. But decades later, the reliability of important categories such as major depression remains low (Regier et al., 2013). By and large, the highest levels are found in research studies, where every observer is trained to rate phenomena in the same way. But years of teaching psychiatric residents have shown me that one cannot expect busy clinicians to take the time to use DSM criteria in the prescribed manner. It is all too easy to jump to conclusions from one or two features, rather than to open the book and count.

This problem applies to BPD. I have seen many clinicians make this diagnosis on the basis of a single feature: when patients present with overdoses, when they cut themselves, or when they show what Kernberg (1970) termed *splitting* (the tendency to see people as all good or all bad). But BPD is a complex disorder that cannot be defined by any one symptom or behavior.

While defining reliable criteria for diagnostic categories is a good thing, reliability does not establish validity. Psychiatry needs to develop diagnoses that are as valid as those used by other medical specialties. But as long as categories of mental disorder are based on clinical observation (as opposed to biological markers such as blood tests or imaging), their validity is bound to remain weak.

What are the best criteria for a valid diagnosis of mental disorder? 50 years ago, two psychiatrists from Iowa, Eli Robins and Samuel Guze (1970), wrote an influential paper on this subject. They proposed that diagnoses are valid if they are based on (1) a clear-cut clinical description; (2) laboratory studies; (3) delimitation from other disorders; (4) follow-up studies documenting a characteristic outcome; and (5) family prevalence studies.

BPD fails on most of these grounds. It greatly overlaps with other mental disorders. It lacks a specific biological profile. It does not have a specific family history. It is not a disease in the sense of general medicine. At best, BPD is a coherent clinical syndrome with a set of typical outcomes.

Yet if we were to apply the Robins and Guze criteria to most of the mental disorders listed in DSM, very few would be considered valid. Even the most intensively studied categories, such as schizophrenia and bipolar disorders, have serious problems with overlap, lack laboratory tests to confirm their presence, and do not conform to an expected family pattern. And major depression needs to be divided into subcategories to be made valid (Parker & Manicavasagar, 2005). All these diagnoses could turn out to be *syndromes* (i.e., symptoms that occur together, as opposed to true diseases with a common etiology and pathogenesis).

Although the proposals of Robins and Guze were sensible, diagnostic science is not advanced enough to apply such stringent criteria (and will not be for decades to come). Thus, even if BPD is short on validity, it is no better and no worse in this respect than other widely accepted disorders. In the meantime, we can look for ways to refine the diagnosis.

The eight diagnostic criteria for BPD introduced in DSM-III have not been changed, but a ninth criterion was added in DSM-IV

(American Psychiatric Association, 1994) to describe cognitive symptoms. This change added an important and characteristic set of clinical features, including paranoid trends and depersonalization.

Following the rules set out in the DSM definition, clinicians refer to a list of criteria, and must identify five of them in a patient to make a diagnosis. This *polythetic* approach is typical of the DSM system. But the manual fails to specify any core features without which a diagnosis should *not* be made. (Again, this problem is not specific to the BPD diagnosis, but applies to almost all mental disorders.)

DSM-5's Section II Definition of BPD

Let us examine how Section II of DSM-5 (American Psychiatric Association, 2013) defines BPD. As in DSM-IV (American Psychiatric Association, 1994), criteria are first established for an *overall* diagnosis of personality disorder. That definition consists of a pattern of inner experience and behavior that is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, leads to distress or impairment, and cannot be accounted for by culture or by other mental disorders.

BPD is then defined as a subcategory of personality disorder with a pervasive pattern of instability of interpersonal relationships, self-image, and affect, associated with marked impulsivity, beginning by early adulthood, and present in a variety of contexts. The nine criteria describe fear of abandonment, unstable relationships, unstable self-image, impulsivity, self-damaging behaviors, affective instability, emptiness, excessive anger, and paranoid ideas or dissociation.

These nine criteria fall into several domains: affective symptoms, impulsive behaviors, interpersonal sensitivity, and cognitive symptoms. (The identity criterion is rather vague and does not clearly fit into any of these domains.). Another problem is that cognitive symptoms fail to include a common feature in BPD: transient, stress-related auditory hallucinations (Zanarini, Gunderson, & Frankenburg, 1989).

The larger problem is that any combination of five symptoms gives you the diagnosis—even if not all domains are represented. And there are no core required symptoms. Within a polythetic system, patients with the same diagnosis can be very different (Clarkin, Widiger, Frances, Hurt, & Gilmore, 1983). There are just too many ways to reach the same conclusion. And the problem is even worse with nine criteria than it was with eight. Moreover, BPD is a complex syndrome that cannot be defined by a limited number of criteria. Any

expert in psychometrics will tell you that we need many more than nine items—either in questionnaire format, or as part of a semistructured interview. Clarkin et al.'s criticism is as valid today as it was in 1983. The DSM criteria that have been in place for the last 40 years were a good start, but cast much too wide a net.

TRAIT DOMAINS UNDERLYING BPD AND THEIR IMPLICATIONS

Although I am a defender of retaining the BPD diagnosis, I recognize that trait dimensions provide important additional information for clinicians that offer an in-depth view of any personality disorder. I therefore recommend that identifying these domains needs to be part of the assessment of every patient with BPD. In Chapter 2, I review several methods for doing so.

Patients with BPD have heritable trait vulnerabilities that can produce symptoms, but only under exposure to psychosocial stressors. Therefore, the disorder cannot be understood without considering its underlying structure of traits, which are related to high sensitivity to adverse life events. These traits, related to the domains of borderline pathology, should be present prior to the onset of symptoms.

The complication is that not one, but several, trait dimensions underlie BPD. They correspond to four domains: emotional dysregulation (or affective instability), impulsivity (or disinhibition), interpersonal sensitivity (often resulting in dysfunctional relationships), and cognitive dysfunction. While one or another of these has been thought to be primary, the evidence from studies using cluster analysis, factor analysis, or latent class analysis to examine which features hang together is not convincing. One large-scale study (Clifton & Pilkonis, 2007) concluded that a single factor fitted the data parsimoniously, reflecting the fact that all four domains are intercorrelated.

By and large, researchers on BPD agree that its clinical features can be understood in the light of these domains. Let us now examine the disorder in the light of each one.

Emotional Dysregulation (Affective Instability)

Emotional dysregulation (abbreviated in this discussion as ED) refers to a high intensity of emotional responses and/or a slow return to baseline following episodes (Putnam & Silk, 2005). *Affective instability*

(abbreviated here as AI) is a very similar construct, describing mood changes characterized by temporal instability, high intensity, and delayed recovery from dysphoric states (Koenigsberg et al., 2002). ED or AI is the most central feature of BPD (Linehan, 1993). Zimmerman, Multach, Dalrymple, and Chelminski (2017) found that one can screen for BPD with over 90% accuracy if one uses just this one criterion.

In classical mood disorders, one sees a consistently lowered (or raised) level of mood. You cannot cheer up a depressed person, and you cannot "bring down" someone in the midst of a manic episode. But with ED or AI, emotion is far from constant. Instead, affect is highly variable and shows a rapid and intense response to environmental triggers (Gunderson & Phillips, 1991). In BPD, patients can be in a different mood every day, or even every hour.

Linehan (1993) proposed the influential theory that the vulnerability to BPD arises primarily from an inborn temperament that makes people prone to ED. This model is based on a broad theory of emotion regulation as a function of the brain, which can differ between individuals on the basis of both temperament and life experience (Gross, 2014). A large body of empirical evidence supports the centrality of this trait in BPD. Patients with this disorder have more intense emotions to begin with, have difficulty regulating them, and rapidly shift from one emotion to another (Putnam & Silk, 2005; Henry et al., 2001; Koenigsberg et al., 2002). Livesley (2003) also postulated that the borderline pattern reflects abnormalities on a trait of emotional regulation, and developed a personality inventory with a specific subscale that can be used to assess affective instability (Livesley, Jang, & Vernon, 1998).

Since ED and AI are constructs that describe a tendency to respond to life events with unusually strong emotions, they have some similarity to the broad dimension that trait psychologists call *neuroticism,* a measure of negative emotionality (or, in common parlance, being "thin-skinned"). Neuroticism is one of the factors in the five-factor model of personality (FFM; see Chapter 2), and it can be assessed quantitatively by using self-report questionnaires. Costa and Widiger (2013) have argued that unusually high scores on trait neuroticism can help define BPD. However, this suggestion fails to distinguish levels of negative emotions (such as anxiety and depression) from *variability* in affect as well as unstable affect. Moreover, neuroticism is also high in anxiety disorders, as well as in personality disorders falling within DSM-5 Section II"s Cluster C, the so-called "anxious/fearful" cluster (Brandes & Bienvenu, 2006).

There are also methodological problems with "reducing" BPD to its trait domains. One is that self-report measures may not be the only or the best way to measure personality. People may not always remember how unstable their mood was, particularly when they were very upset. Instead of using questionnaires, researchers can assess moment-to-moment changes in mood by having patients score their reactions as these occur, using either pencil and paper or a cell phone. Several research groups have used this method, called *ecological momentary assessment*. Our group (Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007) found that patients with BPD experienced more unpleasant emotions, and also showed more variability in mood than normal controls. Similar findings have been reported by others (Ebner-Priemer et al., 2007). A review of this literature can be found in Santangelo, Bohus, and Ebner-Priemer (2014).

Thus far, no one has identified any consistent biological correlates of ED or AI. One research method is to expose patients to experimental settings in which affectively charged images are presented, after which one measures a range of psychophysiological responses. But this approach is limited by the artificial nature of an experiment. There could be large discrepancies between experimental models and real-life situations.

A related approach is to ask subjects to identify various emotional states from the observation of faces. In an early study, Frank and Hoffman (1986) reported that BPD patients are unusually sensitive to faces presented by a tachistoscope, and are particularly accurate in identifying negative emotions. Later, Wagner and Linehan (1999) found patients with BPD to be particularly hypersensitive to faces showing fear. This observation was confirmed by imaging data (Donegan et al., 2003). Thus patients with BPD tend to see neutral faces as threatening, and these responses are associated with increased reactivity in the amygdala. However, these are complex phenomena. Koenigsberg (2010) found that a very wide range of brain structures show activity changes in patients who present with AI.

Impulsivity

Impulsivity describes a set of psychopathological phenomena that share a common biological substrate. A biopsychosocial definition of impulsivity proposed by Moeller, Barratt, Dougherty, Schmitz, and Swann (2001) includes (1) decreased sensitivity to the negative consequences of behavior; (2) rapid, unplanned reactions to stimuli

before complete processing of information; and (3) lack of regard for long-term consequences. There are several other terms in the literature that describe similar phenomena: *disinhibition* (Clark, Livesley, & Morey, 1997), *low effortful constraint* (Nigg, Silk, Stavro, & Miller, 2005), *low conscientiousness* (Costa & Widiger, 2013), and *externalizing behaviors* (Achenbach & McConaughy, 1997; Krueger, Caspi, Moffitt, Silva, &, McGee, 1996). In longitudinal research, impulsive traits tend to follow a consistent trajectory over the course of childhood and adolescence (Masse & Tremblay, 1996).

Linehan (1993) has suggested that impulsive behaviors are largely responses to dysregulated affects. It is true that behaviors (e.g., self-cutting and substance misuse) can be used to deal with unpleasant emotions. However, some patients with chronic dysphoria do not have these features. In our own research (Zweig-Frank & Paris, 1995), we found that patients with Cluster C personality disorders also had high levels of trait neuroticism, but showed few impulsive behaviors. Also, patients with antisocial personality disorder (ASPD) have strikingly impulsive behaviors without being notably dysphoric (Paris, Chenard-Poirier, & Biskin, 2013). In contrast, BPD shows a wide range of both, so it makes sense to consider impulsivity as a separate underlying trait dimension.

Another problem is the ambiguous meaning of the term *impulsivity* (Whiteside & Lyman, 2001; Livesley, 2017). Some dangerous actions are not carried out on the spur of the moment; for example, self-cutting, particularly when addictive, can be planned in advance. Even so, the broad concept describes a tendency to carry out actions in response to stress—what therapists have traditionally termed *acting out*.

A large body of evidence supports the centrality of impulsivity in BPD. Standard self-report measures (such as the Barratt Impulsivity Scale; Patton, Stanford, & Barratt, 1995) show that patients with BPD score high on all subscales of this measure (Links, Heslegrave, Mitton, van Reekum, & Patrick, 1995; Links, Heselgrave, & van Reekum, 1998; Paris et al., 2004).

Impulsivity helps to explain why patients not only *feel* suicidal, but *act* on their thoughts by carrying out multiple suicide attempts or self-harm behaviors (Soloff, Lynch, Kelly, Malone, & Mann, 2000). Tellingly, impulsive spectrum disorders (such as ASPD and substance use disorders) are the most frequent disorders in the first-degree relatives of probands with BPD, and are much more common than mood disorders (White, Gunderson, Zanarini, & Hudson, 2003). In

addition, high levels of impulsivity are the most consistent predictor of clinical outcome in BPD (Links et al., 1998).

Impulsivity has consistent biological correlates (Zuckerman, 2005). Neurobiological studies have found that impulsivity in BPD has a consistent association with abnormalities in neurotransmitter activity. In contrast to the absence of consistent correlates for other trait dimensions, the biological correlates of impulsivity are robust, with consistent relationships to brain systems that modulate behavioral inhibition (Moeller et al., 2001) and are associated with serotonergic neural pathways (Siever & Davis, 1991).

Serotonergic dysfunction in BPD has been demonstrated by using neuroendocrine challenge tests that measure the brain's hormonal response to agents that increase serotonin activity (see review in Ruocco & Carcone, 2016). This relationship has also been confirmed by neuroimaging: positron emission tomography assessing serotonin activity in various brain regions (Siever et al., 1999; Leyton et al., 2001).

Interpersonal Sensitivity

The third domain is seen by Gunderson and Links (2008) as central to BPD. They describe *interpersonal sensitivity* (i.e., a temperamental tendency to respond strongly to real or imagined rejection). In other words, patients with BPD are thin-skinned, but most particularly in interaction with other people, particularly those with whom they are intimate.

It is not clear how interpersonal sensitivity differs from ED, given that problems with people is usually what produces unstable and intense emotions. However, there may be a separate biological mechanism related to this trait. Stanley and Siever (2010), as well as Herpetz and Bersch (2015), have suggested that interpersonal sensitivity could be related to abnormalities in neuropeptides such as oxytocin and vasopressin. Since oxytocin levels have been found to be related to love and attachment, this hypothesis makes intuitive sense. Yet much more research is needed, and we have no data showing that sniffing oxytocin is an effective treatment for BPD.

Cognitive Dysfunction

The cognitive symptoms in BPD are not accounted for by either AI or impulsivity. Hallucinations or depersonalization can be triggered

by periods of emotional dysregulation (Gunderson & Links, 2008). But patients with high neuroticism rarely hear voices. Nor do patients with other impulsive disorders.

Cognitive dysfunction is not a trait in the same sense as ED or impulsivity is, given that these phenomena are rare in normal people. At least half of patients meeting overall diagnostic criteria for BPD develop cognitive symptoms, and these features distinguish BPD from other personality disorders (Zanarini, Gunderson, & Frankenburg, 1990; Yee, Korner, McSwiggan, Meares, & Stevenson, 2005). These symptoms do not imply the presence of a frank psychosis: Patients may have paranoid feelings without interpreting them in a delusional way; may hear voices or see visions while understanding that these perceptions are imaginary; and may experience depersonalization without impaired reality testing. For this reason, the biological and psychological correlates of cognitive dysfunction in BPD may be entirely different from those of the psychoses.

Cognitive symptoms may reflect an entirely separate domain of trait vulnerability, but we know little about their biological correlates, or their sources in psychological development. Yet these are the phenomena that, even today, still seem to place these patients on a "borderline."

HOW TRAITS INTERACT TO PRODUCE DISORDERS

BPD is an outcome that reflects a combination and interaction of multiple trait dimensions. The two most important domains are ED and impulsivity, but they do not, by themselves, account for all clinical features of the disorder. It is their interaction that "cooks" the disorder. Moreover, trait dimensions can interact through feedback loops in which ED promotes impulsivity, while impulsive actions lead to further ED.

These problems often present clinically as a history of unstable relationships. Intimacy can be difficult for everyone; however, if people respond with intense emotion to every conflict, and if they act out impulsively when problems arise, their relationships are bound to be unstable. In patients with BPD, the way intimate relationships begin (with intense emotion and impulsive "jumping in") reflects their impulsive traits. Similarly, these traits influence the way intimate relationships end (with rage and impulsive breakups).

DIAGNOSTIC MEASURES

Although I am dissatisfied with the current DSM criteria for diagnosing BPD, I teach them to psychiatric residents. For now, it seems best for everyone to use the same manual.

But there are other ways to conceptualize the diagnostic process. As suggested by Zanarini (2005), BPD is a multidimensional disorder, so that patients have symptoms in multiple spheres (AI, impulsivity, interpersonal sensitivity, and cognitive impairments), the presence of *all* these features should be required to make a diagnosis. A narrower definition would describe a more homogeneous group of patients.

For my own research, I have used a system first developed at McLean Hospital by Gunderson—the Diagnostic Interview for Borderline Patients (DIB), later revised by Zanarini, Gunderson, and Frankenburg (1989) as the DIB-R. This semistructured interview assesses patients in the four domains of BPD pathology (affective, cognitive, impulsive, and interpersonal). Each domain is scored separately (0–2 for affective and cognitive, and 0–3 for impulsive and interpersonal). The maximum score is 10, and 8/10 is the cutoff for BPD.

The DIB-R scales parallel DSM but follow a more rigorous algorithm. The affective subscale taps AI and emptiness (DSM criteria 6 and 7), but to attain a full score of 2, the patients must have serious problems with anger (DSM criterion 8). The cognitive scale taps depersonalization, paranoid trends, and pseudohallucinations (a broader range than DSM criterion 9); if all these features are absent, then the other three domains will have to score fully. The impulsive scale taps suicidality and self-cutting (DSM criterion 5), as well as other self-damaging behaviors (DSM criterion 4). The interpersonal scale describes problems with abandonment, instability, and identity disturbance (DSM criteria 1, 2, and 3).

Patients with a score of 8 on the DIB-R will always meet DSM criteria. But quite a few who meet five of the nine DSM criteria will not be considered as having BPD according to the DIB-R criteria. These patients have borderline traits, but either lack the impulsive behaviors seen in the full syndrome, or do not have conflictual relationships (because they avoid getting involved with other people). This group has *subsyndromal* pathology; that is, some symptoms resemble BPD, but these patients do not meet full criteria for the disorder (Zanarini et al., 2007). They might be considered as lying on the "borderline" of BPD.

The criteria in DSM-5 could have been narrowed down in the

same way as in the DIB-R. For example, if DSM required seven criteria instead of five to be met for a BPD diagnosis, it would describe a more homogeneous population of patients that could be distinguished from those with other personality disorders. The patients I treat are hardly peas in a pod, but they are reasonably similar to each other.

Case Example 1: Wilma (Typical BPD)

Wilma was a 39-year-old illustrator who had been living with the same female partner for 15 years. This relationship had originally been sexual, but gradually evolved into a friendship. Wilma had an affair with a man, and then became involved with another woman. Telling her partner about this development led to a crisis, followed by two suicide attempts (followed in turn by hospitalizations). In the second attempt, Wilma went to a hotel to take an overdose, but called her partner to rescue her. At the time of evaluation, Wilma was still carrying out a secret affair with the lover, and was still having trouble making a decision. She was sleeping poorly, feeling empty, and experiencing mood swings with angry outbursts. She was also thinking of suicide, cutting herself regularly (something she had been doing for many years), and bingeing on alcohol. Other symptoms included depersonalization, paranoid trends, and visual hallucinations (Wilma would see people in her house, but know they were not real).

Wilma met all nine DSM criteria for BPD and scored 9/10 on the DIB-R.

Case Example 2: Samantha (Typical BPD)

Samantha was a 23-year-old student about to graduate from a university. Samantha's problems started in high school with severe bulimia nervosa; she still forced herself to vomit several times a day. She also cut herself regularly and often thought about suicide. Recently, Samantha had become involved with a boyfriend who was a drug dealer and who took cocaine daily. Samantha herself was drinking heavily and using marijuana on a daily basis.

Samantha had had many difficult and highly conflictual intimate relationships, with both men and women. She described feelings of emptiness and hopelessness. She also experienced several cognitive symptoms: depersonalization, paranoid thinking, and occasional auditory pseudohallucinations.

Samantha met all nine DSM criteria for BPD and scored 9/10 on the DIB-R.

Case Example 3: Sarah (Typical BPD)

Sarah was a 26-year-old nurse who, in spite of having had problems since her adolescence, was presenting for treatment for the first time. She had recently been seen in the emergency rooms of two hospitals for suicide threats. Sarah suffered from diabetes, but was noncompliant with treatment. Although she was a heavy user of alcohol and drugs, she had managed to do well in nursing school. Sarah was sexually promiscuous and had many relationships with individuals addicted to drugs and engaged in criminal behavior, whom she tried to save. Sarah had never made a suicide attempt, but had once hit her head with a rock to injure herself.

What had changed was that Sarah's difficulties began to affect her work. Sarah had angry outbursts with colleagues and on several occasions stormed off the ward. Similar problems had long occurred with boyfriends, usually associated with intense jealousy.

Sarah met all nine DSM criteria for BPD and scored 8/10 on the DIB-R.

However, some patients who meet DSM criteria do not score in all domains, as required by DIB-R.

Case Example 4: Melissa (BPD According to DSM but Not to DIB-R)

Melissa was a 19-year-old woman working part-time in a bakery. Since the age of 14, she had had seven hospital admissions for anorexia nervosa (without bulimia). Melissa had made a suicide attempt 3 years previously after a quarrel with her psychiatrist, and still had suicidal thoughts. Melissa also had been cutting herself since early adolescence.

Melissa was diagnosed in the course of an eating disorders program as having BPD, based on DSM criteria. She met criterion 1 (abandonment), criterion 3 (identity), criterion 5 (self-mutilation), criterion 6 (AI), and criterion 7 (emptiness), but did not meet criterion 2 (unstable relationships), criterion 4 (impulsivity), criterion 8 (emptiness), or criterion 9 (paranoia). On the DIB-R, Melissa scored 1/2 for affective symptoms, 1/2 for cognitive symptoms, 2/3 for impulsivity (based on self-mutilation and aggressive behavior), but only 1/3 for interpersonal relationships (only one friend, and most "borderline" behaviors occurred with professionals), giving her a total score of 5/10.

Some patients who have had BPD in the past will have recovered to the point where they no longer meet criteria (see Chapter 7).

Case Example 5: Nathalie (Lifetime but Not Current BPD)

Nathalie was a 36-year-old woman living alone. Recent symptoms followed a breakup with a boyfriend of 2 years. Nathalie was treated in a hospital for suicidal threats, but did not attempt suicide.

From adolescence, Nathalie had recurrently cut herself and had taken multiple overdoses. But she stopped these behaviors in her late 20s. Nathalie had been unemployed for 10 years, only had a few friends, and was estranged from her family. She had never had a successful intimate relationship. These problems went back many years. Nathalie had only completed high school and had never developed a career.

Nathalie had a lifetime diagnosis of BPD but not a current one, and scored 6/10 on the DIB-R criteria. These changes were mainly due to a reduction in the level of her impulsivity over time, as well as the absence of intimate stormy relationships.

Still other patients have features of BPD but have never met criteria and require a different diagnosis.

Case Example 6: Maureen (Personality Disorder but Not BPD)

Maureen was a 29-year-old woman who was being followed at a community clinic. She was seen after making a serious suicide attempt by ingesting 150 pills of various kinds.

Maureen's problems went back many years. She had graduated from a community college, but never held any job for long. She lived with her parents; she had no relationships with men, but retained some intense friendships with women. The breakup of a friendship, due to Maureen's excessive demands, was the precipitant for this overdose.

Although Maureen was referred with a presumptive diagnosis of BPD, she only met three of the nine DSM-5 criteria and scored 4/10 on the DIB-R. Even a lifetime BPD diagnosis would not fit, due to her low impulsivity and limited involvement in relationships. Given her long-term problems in work and relationships, Maureen met overall criteria for what DSM-5 now calls other specified (or unspecified) personality disorder, with traits lying mainly in Cluster C.

Diagnosis is important to the extent that it provides a guide to management. We aspire to treat patients on the basis of a reliable and valid categorization. An overly broad and fuzzy diagnostic construct fails to identify a core group of patients who show all (or most) clinical features associated with the disorder, and will include too many people with milder symptoms who need different methods of treatment.

The DSM system tends to overdiagnose BPD. Even so, many clinicians are reluctant to identify patients as having a personality disorder, categorizing them within other diagnoses. We need to improve the diagnosis of BPD and make it more valid to convince clinicians who doubt its validity. The best way to do so is to make the criteria more precise and more stringent. This could be accomplished if problems in the domains described by Gunderson and Zanarini were all required. Doing so would narrow the definition of BPD and describe a more homogeneous group of patients who are likely to require the same form of treatment.

Yet, in spite of all the work that has gone into developing interviews to measure BPD, they take too much time for routine clinical use. Thus there are now a number of self-report measures that can be used for rapid assessment. The best of these, in my view, are the Borderline Personality Questionnaire (Poreh, Rawlings, Claridge, & Freeman, 2006) and the Zanarini Rating Scale for BPD (Zanarini, Vulanovic, et al., 2006). These measures are also useful for assessing change during treatment.

BPD IN PRACTICE AND IN THE COMMUNITY

How common are patients with BPD in practice? Are there many cases, or does it just *seem* that way (particularly if each case feels like 10)? Research sheds light on this question. A large number of patients in a variety of clinical settings meet criteria for the BPD diagnosis (as currently defined in DSM).

Most patients with BPD in practice have had either emergency room visits or hospital admissions related to suicidality (Zanarini, Frankenburg, Khera, & Bleichmar, 2001). But their precise percentage on inpatient wards is hard to determine. The number of beds in most North American hospitals has been sharply cut back, and managed care, as well as crowded emergency rooms, discourages the admission of suicidal patients. Moreover, hospitals have different

thresholds for admission, depending on number of beds and size of catchment areas. Thus some estimates of BPD from the past, such as 25% of all inpatients at McLean Hospital (Gunderson, 1984), would not apply in the current clinical scene.

We have more recent information about the prevalence of BPD in outpatient settings. The largest sample derives from a study by Zimmerman et al. (2005) using a large practice affiliated with Rhode Island Hospital, in which 9% met diagnostic criteria.

Patients with BPD are also common in primary care settings. A study by Gross et al. (2002) found that 6.4% of patients met criteria for BPD (in a sample of 218 patients seeing a group of internists).

Needless to say, patients with BPD are particularly common in the emergency room. Forman, Berk, Henriques, Brown, and Beck (2004) found that 41% of 114 patients who had made repetitive suicide attempts, and 15% of 39 patients with single attempts, met criteria for this diagnosis.

But clinical cases are not necessarily representative of the frequency of mental disorders in community populations. Are there patients with similar problems who are not coming for help? If so, do they have milder or more severe symptoms? Research is needed to see how untreated patients differ from those who request treatment or who end up in hospitals.

Psychiatric epidemiology, which measures the prevalence of mental disorders in the community, provides data that can guide research, clinical practice, and planning for mental health systems. However, until the last few decades, few studies assessed the prevalence of any personality disorder diagnosis.

The Epidemiologic Catchment Area (ECA) Study (Robins & Regier, 1991) was a large-scale survey funded by the National Institute of Mental Health (NIMH) and conducted in the 1980s. It examined the prevalence and correlates of the most important DSM-III Axis I disorders. However, it failed to provide information about most personality disorders (only ASPD was assessed). One reason is that the ECA instrument (the Diagnostic Interview Schedule) was designed for use by nonprofessional interviewers. Personality disorder diagnoses require some degree of clinical experience for accurate assessment. Another reason was that the research base for the validity of most personality disorders was not considered to be good enough.

Another early report (Swartz, Blazer, George, & Winfield, 1989) reconstructed the diagnosis of BPD from ECA data on general symptoms, which is a questionable methodology. In the 1990s, the

next wave of NIMH-funded psychiatric epidemiology, the National Comorbidity Survey (Kessler et al., 1994), again limited itself to ASPD and did not try to measure BPD.

But in the next 20 years, several studies applied epidemiological methods to measure the prevalence of *all* personality disorders. A report from Oslo, Norway (Torgersen et al., 2000) examined the frequency of personality disorders in that city, and BPD was found in 0.7%. A second study, conducted at one of the original ECA sites in Baltimore (Samuels et al., 2002), also measured all Axis II categories, and BPD was found in 0.5%. A third study, the NESARC, designed to assess alcohol and substance abuse in the United States (Grant et al., 2004), examined the prevalence of all personality disorders, and BPD was found in as many as 5.9%; however, as noted above, the true prevalence was closer to 2.7% (Trull et al., 2010).

A longitudinal study of university students (Lenzenweger, Johnson, & Willett, 2004) examined all personality disorders (albeit in a relatively privileged population), and BPD was found in 1.6%. A prospective study of children followed into adulthood (Crawford et al., 2005) estimated the frequency of personality disorders in a community sample from self-report data, and estimated the rate of BPD as 3.9%. In the United Kingdom, Coid et al. (2006) published a study of all DSM-defined personality disorders in a representative community sample, and found BPD in 0.7%. In the United States, Lenzenweger et al. (2007) used a nationally representative sample, based on the National Comorbidity Survey Replication (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Their estimate of BPD prevalence was 1.6%.

Thus the research findings vary, but cluster around a prevalence of 1–2%. The much higher estimate in the NESARC (Grant et al., 2004), which found it to be nearly 6% (Grant et al., 2008), has been quoted in many research papers. But it is almost certainly an overestimate, as shown by later analyses of the same data (Trull et al., 2010). The high rate could have been due to raters who were too liberal about scoring clinical features that lie on a spectrum. The wide discrepancy reflects the uncertain boundaries of a condition that was rated by nonexpert research assistants. As we have seen, subclinical forms of BPD exist (Zanarini et al., 2007), although patients who only meet two to four DSM criteria for the disorder can still be seriously dysfunctional (Ten Have et al., 2016).

In a recent review, Ellison, Rosenstein, Morgan, and Zimmerman (2018) concluded that the point prevalence of BPD is roughly

1% in community settings, 12% in outpatient psychiatric clinics, and 22% in inpatient psychiatric clinics. Thus, while the results vary, it seems safe to conclude that BPD has a community prevalence similar to that of schizophrenia. It is also safe to conclude from studies of clinical populations that a fair percentage of the patients that clinicians see have BPD, whether they are recognized as such or not.

Yet, even though we see many patients with BPD, quite a few do not present clinically. Like other mental disorders, BPD varies in severity. Practitioners tend to think about diagnoses in terms of their worst cases. Even in schizophrenia, many patients live in the community without being followed in the mental health system (Harding, Brooks, Ashikaga, Strauss, & Breier, 1987). Similarly, patients treated for BPD, but with less severe symptoms, are more likely to recover (see Chapter 7).

Unfortunately, clinicians sometimes have the mistaken impression that all cases are like the most difficult ones, in which serious pathology continues unabated for years (Cohen & Cohen, 1984). This is one reason why clinicians stigmatize patients with BPD (see Chapter 12). They see cases in emergency rooms or on wards when the symptoms are at their worst. They do not consider that episodes marked by severe dysregulation are just that—episodes. In my experience, there are many patients with BPD whose problems are only known to their families and/or their intimate partners.

Gender is another reason for discrepancies between clinical practice and community prevalence. In the clinic, BPD is mostly a female disorder; up to 80% of clinical cases are women (Zimmerman et al., 2005). That may not be true in the community. Torgersen et al. (2001) found more women in their survey, but both Coid et al. (2006) and Lenzenweger et al. (2007) identified just as many men. By and large, women are more help-seeking than men: Clinics see more females with almost any psychiatric disorder, and fewer males go into therapy.

Some years ago, our group carried out a special study of men with BPD (Paris, Zweig-Frank, & Guzder, 1994a). But we had to advertise to find cases. We placed our advertisement in an "alternative" newspaper (read by many young males). We had expected to find a large overlap with ASPD. But although I have seen such cases in practice, none of the research participants had that comorbidity. In most respects, these men with BPD were identical to women with the disorder. Other recent studies (e.g., Goodman, Patel, Oakes, Matho, & Triebwasser, 2013) have reported similar results. Our group also

found that 10% of our male sample was actively homosexual (Paris, Zweig-Frank, & Guzder, 1995), but that was 25 years ago; since then, reported homosexuality and bisexuality may have become more common (Twenge, Sherman, & Mills, 2016).

The most probable explanation for gender differences in clinical samples is that women are more likely to develop the kind of symptoms that bring patients in for treatment. In one older report, twice as many women as men in the community suffered from depression (Weissman & Klerman, 1985). In contrast, a preponderance of men met criteria for substance abuse and psychopathy (Robins & Regier, 1991), and males with these comorbidities do not necessarily present in the mental health system, but they may be seen in forensic populations.

Men and women with similar psychological problems may express distress differently. Men tend to drink more and commit more crimes. Women tend to turn their anger on themselves, leading to depression, as well as the self-cutting and overdosing that characterize BPD. While ASPD and BPD may derive from similar underlying pathology, presenting with different symptoms influenced by gender, the differences in ED are too great to consider them as variants of the same disorder (Paris et al., 2013). On the other hand, men who have *both* disorders are more frequently violent (Robitaille et al., 2017).

We have even more specific evidence that men with BPD often fail to seek help. In a large study of completed suicides among people ages 18–35 (Lesage et al., 1994), 30% of the suicides had a diagnosis of BPD (as confirmed by *psychological autopsy*, in which symptoms were assessed by interviews with family members). Most of the suicide completers were men, and very few were in treatment at the time of their death. Similar findings emerged from a later study conducted by our group (McGirr, Paris, Lesage, Renaud, & Turecki, 2007).

This discrepancy applies to most of the patients clinicians see in the mental health system (schizophrenia is an exception, in that it has a preponderance of males). As I sometimes say to students, "Men just don't like to ask for directions."

That men can have typical BPD pathology is illustrated by the following case.

Case Example 7: Steven

Steven was a 28-year-old man working part-time who had recently finished a 3-year course in theatre at a community

college. Steven was living with a girlfriend he had been with for 2 years; this was a difficult and stormy relationship, since Steven could be very demanding. He was assessed after going into a frightening rage after a conflict with one of his teachers.

Steven had a psychiatric history going back to age 18, and had been admitted twice for suicidal threats. Although he had never made a suicide attempt, he often cut himself. The main problems were rages, with threats against others and occasional destruction of property, and a history of binge drinking. Other symptoms included "having a movie with sound and pictures" run in his head, associated with violent fantasies, as well as chronic feelings of depersonalization and derealization.

Steven met all DSM criteria for BPD and scored 8/10 on the DIB-R.

WHY MAKING A BPD DIAGNOSIS IS IMPORTANT

BPD is a diagnosis that makes a difference. If we don't recognize that our patients have this disorder, they can end up getting the wrong treatment (usually ineffective polypharmacy). Even *with* the diagnosis, patients may receive interventions that are misguided or counterproductive. But *without* the diagnosis, they are most likely to be treated with drugs of limited value. And in a therapy practice, missing BPD prevents clinicians from modifying their methods.

There are several advantages in making a diagnosis of BPD. The first concerns the recognition of a complex form of psychopathology with symptoms that do not occur in isolation. BPD is a construct that can account for the co-occurrence of a wide range of affective, impulsive, and cognitive symptoms in the same patient.

The second advantage concerns prediction of outcome. BPD has a characteristic course over time, beginning in adolescence, with symptoms peaking in early adulthood, followed by gradual recovery in middle age (Paris, 2003). This pattern provides an important frame for therapy.

The third advantage lies in predicting response (or lack of response) to treatment. For example, pharmacotherapy for depression is less effective in the presence of any personality disorder, and patients with BPD do not consistently respond to antidepressants (see Chapter 8).

The fourth advantage is that generic forms of psychotherapy do not work well in BPD. Instead, there is good evidence that specific

methods of psychotherapy are effective. If we do not make the diagnosis, patients may not be referred for these treatments.

Finally, failure to recognize BPD leads to mistaken expectations about course and treatment response. Making the diagnosis allows us to inform and educate patients and their families.

There are problems with the BPD diagnosis, but they are hardly unique. Unclear boundaries afflict most disorders in DSM. Examples include conduct disorder (which has an unclear boundary with misbehavior) or social anxiety disorder/social phobia (which fades imperceptibly into shyness). Major depression is fuzzy around the edges and lacks a clear boundary with normal unhappiness (Horwitz & Wakefield, 2007).

I once had a discussion with a prominent researcher in psychology about the problems with categories in the DSM system. He suggested that after DSM-III came out in 1980, academic psychiatry should have supported studies to determine the validity of every criterion for every diagnosis. (Technically, this would have required measuring *discriminant validity*—i.e., assessing the correlation of each criterion with a diagnosis, and the absence of correlations with other categories.) But no such studies were ever carried out. To this day, we cannot say that the criteria for major depression are the right ones, or that requiring five out of nine criteria to make a diagnosis is a valid procedure.

This work has never been done for BPD, but it could be. I recommend carrying out research to determine which criteria do the best job of establishing discriminant validity.

In summary, there are many problems with the BPD diagnosis, but I believe that it would be a mistake to dismiss or eliminate this category. And there would be clinical consequences if we did so.

SOURCES OF RESISTANCE TO DIAGNOSING BPD

Much progress has been made in the last decade in raising consciousness about BPD. This is partly thanks to the work of personality disorder researchers. Progress is also due to the work of psychoeducators (see Chapter 12).

Nonetheless, there have been and continue to be obstacles to recognizing BPD, leading to many patients' receiving the wrong treatment. And one source of resistance lies within my own profession of psychiatry. As I discuss in Chapter 3, these patients are all too often seen as having other disorders that overlap with some BPD

symptoms. Thus, if patients have mood swings, as most do, they may be diagnosed as having a bipolar disorder. If they are also depressed, as most are, they may be diagnosed with major depression. (Or, if the patients fail to respond to medication, they may be labeled as having "treatment-resistant depression.") If they have a traumatic past, as many patients do, they may be diagnosed as having posttraumatic stress disorder (PTSD).

One reason for the resistance to diagnosing BPD is that physicians prefer to put their patients in categories for which they believe they have effective tools. Thus psychiatrists may prefer to diagnose a bipolar disorder or major depression if they feel that they have medication that can control these symptoms. And psychotherapists who have learned psychological methods to help patients with trauma may be attracted to a diagnosis of PTSD. In contrast, feeling able to treat BPD requires a commitment to specialized psychotherapy that not everyone has.

Another source of resistance arises from how one perceives the more general construct of personality disorder. Admittedly, this is a concept that requires much more explanation than something like depression does. But it should not be too much of a stretch to understand that not "having a life" (as patients sometimes put it, and as I discuss in later chapters) can be the cause of serious unhappiness. This is why the treatment of BPD, which helps patients control emotions, avoid impulsive actions, and get along better with other people, is more efficacious than any existing medication.

This having been said, we need to keep in mind that diagnosis is not an exact science. At this point, categorization functions mainly as a way of communicating—describing clinical syndromes that hang together conceptually and that have implications for choosing options for therapy. Eventually, when we understand what causes mental disorders, we will have something better. But for now, making the diagnosis of BPD is important because it guides clinicians away from treatments that do not work to those that do.

CLINICAL IMPLICATIONS

- BPD is a diagnosis that describes a wide range of symptoms and that is rooted in personality traits.
- The best approach to refining the diagnosis lies in requiring more criteria and in requiring symptoms in more domains.

- Dimensional descriptions of traits are more useful for research than for clinical practice.
- There are more cases of BPD in the community than are seen by clinicians.
- The BPD diagnosis describes a group of patients who require a unique approach to treatment.

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