Borderline Personality Disorder: An Overview

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Borderline personality disorder is a complex, disabling disorder. The chairperson for the American Psychiatric Association workgroup for the evidence-based practice guideline on its treatment gives an overview of this disorder's etiologies, neurobiology, longitudinal course and recommended treatments. Future directions for both treatments and research are also discussed.

Although the term *borderline* has been in clinical use since the late 1930s, it only became an official Axis II diagnosis in 1980 with the publication of *DSM-III*. Currently, *DSM-IV-TR* emphasizes that patients with borderline personality disorder (BPD) show a "instability of interpersonal relationships, self-image, and affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts," and any five out of nine listed criteria must be present for the diagnosis to be made.

**Classification and Diagnosis**

Gunderson (2001) portrayed *DSM*-defined BPD as a diagnostic category layered between neurotic and psychotic disorders, and he differentiated BPD from Otto Kernberg, M.D.'s, theoretical concept of intrapsychic structure referred to as *borderline personality organization*, an umbrella concept that encompasses a number of Cluster A and Cluster B personality disorders that are characterized by the presence of primitive defenses and identity diffusion, yet with the maintenance of reality testing (Kernberg, 1975). It is now clear that *DSM-IV* defined BPD is a heterogeneous construct that includes patients on the mood disorder spectrum and the impulsivity spectrum (Siever and Davis, 1991), in contrast to the original speculation that these patients might be near neighbors of patients with schizophrenia or other psychoses. Patients with schizotypal personality disorders are, instead, the genetic cousins of those patients with schizophrenia. Considerable controversy exists regarding the use of a categorical diagnostic system for the personality disorders because dimensional models of personality are widely utilized in personality studies and can accommodate style, traits and pathology (e.g., the five-factor model of neuroticism, extraversion, openness, agreeableness and conscientiousness [Costa and McCrea, 1992]). A reasonably robust literature supports the validity of a quantitative model (sometimes referred to as a "hypertension" model), implying that pathological functioning, disability and distress accompany the extreme exaggeration of an otherwise adaptive trait (Oldham and Morris, 1995). However, it has been difficult to achieve consensus on a single dimensional model, and the dimensional approach is not easily applicable to medical systems of classifying pathology.

**Etiology and Neurobiology**

A general convention that has been relatively universally accepted in personality studies is that personality itself involves two components: temperament (largely the genetic/constitutional component) and character (largely the component resulting from the molding and shaping influences of life events and development). There are a number of theories of the etiology of BPD (Oldham, 2002; Zanarini and Frankenburg, 1997), which need not be mutually exclusive. Due to the extensive heterogeneity within the *DSM-IV-TR* definition of BPD, there are inevitably multiple combinations of temperamental and environmental factors that lead to its development. A general stress/vulnerability conceptual framework is useful in considering varying combinations of predisposing genetic risk factors and stressful life experiences (Paris, 1999). Among the factors contributing to the etiology of BPD that have been suggested are:

- Affective dysregulation (Akiskal, 1981; Akiskal et al., 1985; Klein and Liebowitz, 1982)
- Deficit in impulse control (Hollander, 1993; Links and Heslegrave, 2000; Siever, 1996; Zanarini, 1993)
- Excessive aggression, either as primary temperament or secondary to severe and sustained childhood abuse (Kernberg, 1975; Zanarini and Frankenburg, 1997)
- Impaired development of autonomy, perhaps related to parental separation-resistant
Increasing numbers of studies are exploring the neurobiology of BPD, but findings to date are relatively nonspecific. It has been recognized that a complex process of gene-environment interaction is involved in the determination of personality types and disorders (Cloninger, in press; Siever et al., 2002; Torgersen, 2000). Endophenotypes are being studied, such as impulsive aggression in BPD, that are thought to reflect underlying genetic vulnerabilities (Siever et al., 2002). Reductions in central nervous system serotonin levels have been correlated with impulsive aggression in patients with BPD (Hansenne et al., 2002; New and Siever, 2002; Skodol et al., 2002b). Affective instability characterizes other patients with BPD, perhaps related to cholinergic irregularities (New and Siever, 2002; Pally, 2002).

In addition, patients with BPD who have experienced sustained childhood abuse may demonstrate hyperresponsiveness of the hypothalamic-pituitary-adrenal system (Rinne et al., 2002). Neuroimaging studies have suggested abnormalities in the prefrontal cortex in patients with BPD (Juengling et al., 2003; New and Siever, 2002), as well as reduced volume in the hippocampus and the amygdala (Schmahl et al., 2003). Further work is needed before the relevance and specificity of these findings for patients with BPD can be elucidated.

**Epidemiology and Course**

Borderline personality disorder is thought to occur in 1% to 2% of the general population, although there have only been a few large-scale, population-based epidemiological studies that included BPD and utilized structured interview methodology. In a careful analysis of the limited literature on the topic, Torgersen (in press) tabulated the prevalence of BPD in eight published studies, including his own Norwegian study (Torgersen, 2000). Across all eight studies, the median prevalence for BPD in the population was 1.42%, and the mean was 1.16%. The prevalence of BPD was estimated to be 10% to 20% in psychiatric outpatient populations and 15% to 20% in psychiatric inpatient populations (Gunderson, 2001).

Extensive comorbidity has been documented for BPD with other Axis II disorders (Oldham et al., 1992), as well as with Axis I disorders (Gunderson, 2001; Skodol et al., 2002a). Prominent among Axis I/BPD patterns of comorbidity are BPD and mood disorders (Skodol et al., 1999b), anxiety disorders (Skodol et al., 1995) and substance use disorders (Grant et al., 2004; Skodol et al., 1999a). The presence of comorbidity is reported to complicate the patient's treatment response (Skodol et al., 2002a). Evidence is persuasive that patients with BPD frequently have high levels of disability (Skodol et al., 2002a) and are high treatment utilizers (Bender et al., 2001).

All personality disorders, according to *DSM-IV-TR*, have their onset in adolescence or early adulthood and show an enduring pattern of dysfunction that is stable and of long duration. New data, however, are challenging this view of the stability of the personality disorders over time, and this is clearly the case for BPD (Grilo and McGlashan, in press). The multisite Collaborative Longitudinal Personality Disorders Study, funded by the National Institute of Mental Health, is a naturalistic study involving careful standardized sequential assessments over time. Recent data indicate that substantial percentages of patients who met semi-structured interview-based *DSM-IV* diagnosis of BPD at intake did not sustain that diagnosis at 24 months post-intake. Defining remission as two consecutive months during which no more than two BPD diagnostic criteria remain present, 42% of patients with BPD had remitted; if remission is defined as showing no more than two criteria for 12 consecutive months, the BPD remission rate was 28% (Grilo et al., in press). Clearly, then, the diagnosis of BPD is more variable over time than the *DSM-IV* generic criterion of longitudinal stability would imply. Other studies have shown similar results (Lenzenweger, 1999; Zanarini et al., 2003).**

**Treatment**

The evidence-based American Psychiatric Association practice guideline for the treatment of patients with BPD recommends psychotherapy as the primary treatment for BPD, combined with symptom-targeted adjunctive pharmacotherapy (Oldham et al., 2001). These recommendations were made with substantial clinical confidence, based on published randomized controlled trials of both psychotherapy and pharmacotherapy, as well as clinical consensus. The APA practice guideline does not recommend a specific type of psychotherapy, but cites two types of psychotherapy that have been demonstrated to be beneficial, using a randomized controlled design: dialectical behavior therapy (DBT) (Linehan et al., 1999, 1994, 1991) and psychodynamic psychotherapy (Bateman and Fonagy, 2001, 1999). Dialectical behavior therapy involves manual-based weekly outpatient...
individual psychotherapy, weekly group skills training and a number of other components (Linehan, 1993). The Bateman and Fonagy (2001) psychodynamic psychotherapy study also involved weekly individual and group sessions, in this case combined with other treatment activities in a partial hospital setting. Neither treatment is a quick fix: the DBT results were based on one year of treatment and showed reduced self-injurious behavior, greater treatment adherence and improved interviewer-rated social adjustment. The psychodynamic psychotherapy results followed 18 months of treatment and showed reduced self-injurious behavior, anxiety and depression. In both cases, follow-up studies reported maintenance of gains for at least one year (Bateman and Fonagy, 2001; Linehan, 1993). Subsequent to the publication of the APA practice guideline, there have been additional reports of controlled psychotherapy trials that also appear promising (Bohus et al., 2004; Clarkin and Levy, 2003; Linehan et al., 2002; Verheul et al., 2003).

In addition to psychotherapy, the APA practice guideline recommends symptom-targeted pharmacotherapy. In a review of the literature on pharmacotherapy for BPD, studies were categorized into randomized controlled trials, open-label trials and clinical reports (Soloff, 2000; Soloff, in press). Based on clinical judgment derived from a synthesis of the evidence, three algorithms were included in the APA guideline reflecting prominence of cognitive/perceptual symptoms, affective dysregulation symptoms or impulsive-behavioral dyscontrol symptoms (Oldham et al., 2001).

Summary
Borderline personality disorder is a disabling, complex, heterogeneous disorder characterized by variable combinations of impulsive self-injurious behavior, affective instability, cognitive/perceptual symptoms, interpersonal difficulties and other symptoms. Great strides have been made in understanding the etiologies, neurobiology and longitudinal course of BPD. An evidence-based practice guideline has been developed for the disorder, and a growing number of studies demonstrate that carefully planned and administered treatment can be effective for many patients with BPD.

References:


46. Skodol AE, Siever LJ, Livesley WJ et al. (2002b), The borderline diagnosis II: biology, genetics, and clinical course. Biol Psychiatry 51(12):951-963 [see comment].


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