Borderline Personality Disorder

HOW TO RECEIVE CREDIT

- Read the enclosed course.
- Complete the questions at the end of the course.
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Faculty Disclosure
Contributing faculty, Mark Rose, BS, MA, LP, has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

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Division Planners/Director Disclosure
The division planners and director have disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

Audience
This course is designed for physicians, physician assistants, and nurses who are involved in the care of patients with borderline personality disorder.

Accreditations & Approvals
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Course Objective

The purpose of this course is to provide health and mental health professionals with the information necessary to assess and treat patients with borderline personality disorder effectively and safely, while minimizing their own stress level and clinic disruption these patients are capable of producing.

Learning Objectives

Upon completion of this course, you should be able to:

1. Review the history of borderline personality disorder (BPD).
2. Describe the current and previous diagnostic criteria for BPD.
3. Outline the incidence and prevalence of BPD.
4. Identify common psychiatric and medical comorbidities of BPD.
5. Evaluate the pathophysiology and natural history of BPD in various patients.
6. Analyze barriers to the care of patients with BPD.
7. Discuss approaches to the assessment and diagnosis of BPD.
8. Describe conditions to consider in the differential diagnosis of BPD.
9. Outline the history of therapy for BPD and selection of the appropriate level of care forpatients with BPD.
10. Discuss approaches to identify and intervene to prevent self-harm, parasuicidal behaviors, and suicide in patients with BPD.
11. Assess the efficacy of available specialist psychosocial therapies used in the treatment of BPD.
12. Evaluate the efficacy of available generalist and primary care interventions used in the treatment of BPD.
13. Review the role of pharmacotherapy in BPD treatment, including contraindicated medications.
14. Describe the importance of involving the family in treatment approaches for BPD.
15. Discuss approaches to managing psychiatric comorbidities in patients with BPD.
16. Outline the prognosis of patients with BPD.

EVIDENCE-BASED PRACTICE RECOMMENDATION

Sections marked with this symbol include evidence-based practice recommendations. The level of evidence and/or strength of recommendation, as provided by the evidence-based source, are also included so you may determine the validity or relevance of the information. These sections may be used in conjunction with the course material for better application to your daily practice.

INTRODUCTION

Borderline personality disorder (BPD) is a severe, complex psychiatric disorder characterized by long-standing patterns of disproportionately intense emotions, impulsive and self-destructive behaviors, and chaotic relationships. BPD has a lifetime prevalence of 3% to 6% in the general population and a somewhat higher prevalence in primary care populations [1; 2]. Gender prevalence is comparable in community populations, but women with BPD are disproportionately represented in clinical populations.

The understanding of BPD is in the process of change, and knowledge from research and clinical practice is challenging many of the entrenched assumptions concerning the disorder and patients. Historically, causation has been widely assumed to be exclusively environmental, from trauma or abuse in early life, but it is now known to possess a strongly genetic component. The prognosis of patients with BPD has been uniformly bleak, and the disorder was thought to be chronic and resistant to treatment. However, research has revealed a substantially better prognosis and treatment responsiveness. Likewise, the long-held view that treating patients with BPD required significant commitment by highly trained, specialized mental health providers has also been disproven by numerous empirically validated interventions specifically designed for patients with BPD. These therapies can be implemented by mental health professionals with specialized training, mental health professionals with minimal training, or primary care providers with minimal training. Therapies that address the pathology of BPD can lead to significant and enduring patient benefit [3].

Perhaps the greatest barrier to effective care of the patient with BPD is the extent of stigma and negative attitudes toward patients with the disorder. The core features of BPD become activated and expressed in the interpersonal context, and only recently has the information required by clinicians to understand and address the behavioral expressions of core BPD psychopathology become available. Until the last decade, the absence of BPD-tailored therapies and effective strategies for providers to manage patients with BPD led to a lack of improvement in almost all patients, deterioration in some, and provocation of core pathologic features. This patient group is inherently challenging to work with, and lack of patient improvement and symptom exacerbation directed at therapist and clinic staff promoted negative and harsh attitudes toward patients with BPD.

Providers working with patients with BPD also require an understanding of the concept and diagnostic criteria in the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) and how these changes substantially depart from earlier DSM editions [4]. Such education on BPD can lead to greater provider confidence, more positive attitudes toward these patients, improvements in therapeutic progress in patients, and lower stress levels in providers [5].

HISTORY OF BORDERLINE PERSONALITY DISORDER

Unlike most personality disorders that were first described in Europe, the term "borderline personality" was introduced by American psychoanalyst Adolph Stern in 1938 to describe a patient group who did not fully fit the characteristics of psychotic or neurotic patient groups, thus existing on the "borderline" between the two. This concept of BPD persisted into the 1950s and 1960s. The identification and labeling of patients as "borderline" first arose during the era when psychiatry was dominated by the psychoanalytic paradigm.

The classification system for mental disorders was primitive and dichotomous, with classification tied to patient capacity for analysis. Patients considered analyzable, and thus treatable, were diagnosed with neuroses, while those considered not analyzable, and therefore untreatable, were deemed to have psychoses [6].

In 1975, Otto Kernberg introduced the term "borderline personality organization" in reference to a consistent pattern in some patients of disturbed psychologic self-organization, reflected by functional and behavioral instability and disturbance. Independent of a theory of causality, the cluster of symptoms and behaviors that characterize borderline personality became more widely recognized, as did the symptoms now known to characterize BPD, such as dramatic fluctuations from confidence to despair, markedly unstable self-image, rapid changes in mood, intense fears of abandonment and rejection, and propensity for suicidal ideation and self-harm. In 1978, Gunderson and Kolb described these characteristics that now define BPD and were instrumental in the inclusion of BPD as a formal psychiatric classification in the 1980 DSM-III [7; 8].

Despite the introduction of BPD as a formalized psychiatric diagnostic entity in 1980, multiple efforts have been made to recast BPD as an Axis I disorder, initially as a disorder on the schizophrenia spectrum, then as an affective or bipolar spectrum disorder, and finally as a variant of post-traumatic stress disorder (PTSD) [6]. The rationale for these efforts has been disproven, as will be discussed later in this course.

DIAGNOSTIC CRITERIA

The concept of BPD, and the signs and symptoms sufficient for a diagnosis of BPD, did not substantively change between the 1980 DSM-III and the updated and revised DSM-IV-TR release in 2000 [9]. According to the DSM-IV-TR, the diagnosis of BPD is attained by a pervasive pattern of instability of interpersonal relationships, self-image, affects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by five or more of the following [10]:

- Frantic efforts to avoid real or imagined abandonment (not including suicidal or self-mutilating behavior)
- A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation
- Identity disturbance (markedly and persistently unstable self-image or sense of self)
- Impulsivity in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating) (not including suicidal or self-mutilating behavior)
- Recurrent suicidal behavior, gestures, or threats or self-mutilating behavior
- Affective instability due to a marked reactivity of mood (e.g., intense episodic dysphoria, irritability, or anxiety usually lasting a few hours and only rarely more than a few days)
- Chronic feelings of emptiness
- Inappropriate, intense anger or difficulty controlling anger (e.g., frequent displays of temper, constant anger, recurrent physical fights)
- Transient, stress-related paranoid ideation or severe dissociative symptoms

The introduction of operationalized diagnoses for BPD and other disorders based on observable criteria in the 1980 DSM-III was considered a significant advancement in the field. However, the concept of and diagnostic criteria for BPD during and after the 2000 DSM-IV-TR became increasingly criticized on several grounds. For example, the description of BPD was non-specific. In the DSM III and the DSM-IV-TR, clinicians were instructed to diagnose BPD when five out of nine criteria were met. But, with this paradigm, individuals diagnosed with BPD could have as few as one criterion in common. This led to the same diagnosis given to patients with various criterion permutations, producing a heterogeneous patient group [11]. This issue is thought to have been largely resolved with the alternative DSM-5 criteria.

Research of BPD during the 13 years between the DSM-IV-TR and the DSM-5 clarified the understanding of BPD and prompted revisions to the diagnosis [4]. The greatest overall change between the DSM-IV and the DSM-5 has been the elimination of the multi-axial classification system, whereby BPD and other personality disorders were assigned a separate axis (Axis II). Several factors contributed to this change. The distinction between Axis I and Axis II disorders in earlier DSM editions received little empirical validation and increasingly became disputed in light of evolving research and clinical evidence. Personality disorders were traditionally conceptualized as the product of environmental factors, while Axis I disorders were viewed as having a biologic or organic cause. This dominant paradigm influenced the introduction of the multi-axial classification system in the DSM-III. While environmental stressors can contribute to personality disorder development, the same is also true with many Axis I disorders such as major depressive disorder (MDD) and PTSD. Also, BPD does not conform to traditional conceptions of personality disorders as ego-syntonic conditions; the symptoms of BPD are clearly ego-dystonic and lead patients to seek treatment for these symptoms [11; 12]. Another criticism of DSM-IV-TR criteria was the combination of unstable, stress-induced symptoms and stable personality characteristics, also termed dimensional traits [13].

DSM-5 ALTERNATIVE DIAGNOSTIC CRITERIA FOR BPD

In the 2010s, a new model for diagnosing personality disorders was presented to the DSM-5 Task Force, and it was strongly and unanimously approved. However, the American Psychiatric Association Board of Trustees voted to sustain the DSM-IV-TR diagnostic system for personality disorders, including unchanged criteria for BPD, in the main section of DSM-5 due to insufficient evidence to validate the new proposed model [14]. The proposed new model is maintained in the DSM-5 as an "alternative DSM-5 model for personality disorders," and professionals have reported good clinical utility. These proposed BPD criteria are organized into two sections: impairments in personality (self and interpersonal) functioning and pathologic personality traits [4]:

- Moderate or greater impairment in personality functioning, manifested by characteristic difficulties in two or more of the following areas:
  - Markedly impoverished, poorly developed, or unstable self-image, often associated with excessive self-criticism, chronic feelings of emptiness, and/or dissociative states under stress
  - Self-direction: Instability in goals, aspirations, values, or career plans
  - Empathy: Compromised ability to recognize the feelings and needs of others associated with interpersonal hypersensitivity (i.e., prone to feel slighted or insulted); perceptions of others selectively biased toward negative attributes or vulnerabilities

  - Intimacy: Intense, unstable, and conflicted close relationships, marked by mistrust, neediness, and anxious preoccupation with real or imagined abandonment; close relationships often viewed in extremes of idealization and devaluation and alternating between over involvement and withdrawal
- Four or more of the following pathologic personality traits, at least one of which must be impulsivity, risk taking, or hostility:
  - Emotional lability (an aspect of negative affectivity): Unstable emotional experiences and frequent mood changes; emotions that are easily aroused, intense, and/or out of proportion to events and circumstances
  - Anxiousness (an aspect of negative affectivity): Intense feelings of nervousness, tenseness, or panic, often in reaction to interpersonal stresses; worry about the negative effects of past unpleasant experiences and future negative possibilities; feeling fearful, apprehensive, or threatened by uncertainty; fears of falling apart or losing control
  - Separation insecurity (an aspect of negative affectivity): Fears of rejection by—and/or separation from—significant others, associated with fears of excessive dependency and complete loss of autonomy
  - Depressivity (an aspect of negative affectivity): Frequent feelings of being down, miserable, and/or hopeless; difficulty recovering from such moods; pessimism about the future; pervasive shame; feeling of inferior self-worth; thoughts of suicide and suicidal behavior
  - Impulsivity (an aspect of disinhibition): Acting on the spur of the moment in response to immediate stimuli; acting on a momentary basis without a plan or consideration of outcomes; difficulty establishing or following plans; a sense of urgency and self-harming behavior under emotional distress
  - Risk taking (an aspect of disinhibition): Engagement in dangerous, risky, and potentially self-damaging activities, unnecessarily and without regard for consequences; lack of concern for one's limitations and denial of the reality of personal danger
  - Hostility (an aspect of antagonism): Persistent or frequent angry feelings; anger or irritability in response to minor slights and insults

This proposed new model is not without criticism, such as the absence of clear delineation between disorders and between traits and disorders [15]. Some have argued that the criteria are too complicated for clinical use [14]. Either DSM-5 version can be used.

EPIDEMIOLOGY

INCIDENCE AND PREVALENCE

General Population

The results of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) study, the first large-scale, community study of personality disorders in the United States, were published in 2008. This study found the overall lifetime prevalence rate for BPD was 5.9% (18 million people), with similar rates in men (5.6%) and women (6.2%). The prevalence of BPD was higher in Native American men; younger adults who were separated, divorced, or widowed; and persons with lower levels of education and socioeconomic status. Those with lower BPD prevalence were Hispanic men and women and Asian women [1].

The NESARC data were subsequently analyzed using more conservative methods, which found a lifetime prevalence rate for BPD of 2.7% [2]. The findings of lower prevalence rates in earlier studies reflected self-report assessments for data collection and/or low response rates, and disparities between cross-sectional prevalence and lifetime prevalence reflect symptomatic recovery in some patients with BPD [16; 17; 18]. BPD prevalence is comparable to other major psychiatric disorders, such as schizophrenia or bipolar disorder, but lower than MDD or anxiety disorders [19; 20].

Clinical Populations

In clinical settings, BPD prevalence is considerably higher than in the general population, and BPD is the most common personality disorder across all levels of care. BPD has a prevalence rates of 20% to 22% among psychiatric inpatients, 10% to 12% among psychiatric outpatients, 56% among emergency room patients admitted for suicidal behaviors, and 6% among primary care patients [18; 21; 23; 24].

Persons with BPD and borderline symptoms are over-represented in civil, criminal, and child custody forensic settings, and individuals with BPD have an increased likelihood of legal system involvement as perpetrators and victims alike [25]. A study of 220 male and female offenders who recently entered prison found diagnosable BPD in 29.5% and the presence of at least one DSM-IV criterion for BPD in 93.2% [26]. This disparity between population and clinical prevalence indicates that many persons with BPD are undiagnosed and untreated [6].

DEMOGRAPHICS

In general, prevalence studies of BPD have found that prevalence in women is three times higher than in men in clinical settings, with similar gender prevalence in community settings [17; 27; 28]. Although epidemiologic studies have not formally assessed age of onset of BPD, data extrapolation from onset of self-harm (the most predictive symptom of BPD) suggests the onset of BPD occurs before 12 years of age in 32.8% of patients, begins between 13 and 17 years of age in 30.2%, and at 18 years of age or older in 37% [29; 30]. The association of higher BPD prevalence with lower education, income, and socioeconomic class suggests these adversity factors predispose to developing BPD, although this association is likely bidirectional; BPD symptoms may contribute to poor educational achievement, lower income, and social class [18; 27].

In demographic research, "fortunate circumstances" are defined as education above high school, living with a partner, and living in the outskirts of a city, and "unfortunate circumstances" are defined as the opposite. In one study, BPD was found more prevalent in unfortunate circumstances (1.2%) than in moderately fortunate (0.8%) or fortunate (0.4%) circumstances [18; 27].

BPD has been identified in every culture where it has been studied, including the United States, China, Japan, Brazil, Norway, India, and Kenya, although the prevalence rates reported in different countries have varied [18; 31].

RISK FACTORS

BPD is thought to result from the complex interaction between the early caregiving environment and innate temperament and emotion regulation factors. BPD-specific risk factors do not lend themselves to detection in population-level studies.

Risk Factors in the Early Environment

Prospective longitudinal studies have identified environmental and parental factors that significantly contribute to BPD development. The number of BPD symptoms at 28 years of age has been found to be significantly and directly correlated with early attachment disorganization or maltreatment; maternal hostility, inconsistency, and/or over-involvement; aversive or hostile parental behavior; low parental affection; family disruption related to the father's presence; and family life stress. Maternal hostility and early life stress contributed independently to the prediction of BPD symptoms at 28 years of age [33; 34]. Other studies

identified additional factors associated with BPD development, including childhood physical abuse or neglect, sexual abuse, maladaptive parenting, maladaptive school experiences, and the demographic characteristics of low family socioeconomic status, family welfare support recipient status, and single-parent family status [35].

Childhood Sexual Abuse

During the 1980s, the findings that childhood sexual abuse was prevalent in histories of patients with BPD led to the theory of childhood trauma as a primary etiologic factor in BPD development. Further research confirmed that while childhood trauma was highly prevalent in BPD, childhood sexual abuse was not necessary or sufficient for BPD development and did not account for much of the variance in causation [18]. In inpatient and outpatient settings, 40% to 70% of patients with BPD report childhood sexual abuse. Although traumatic childhood experiences, including childhood sexual abuse, are strong risk factors for later developing BPD, fewer than 10% of those with a history of childhood sexual abuse develop BPD, effectively eliminating childhood sexual abuse as a primary cause [36; 37; 38].

Adolescent Risk Factors

Specific adolescent risk factors for adult BPD have been identified. Substance use disorders, especially alcohol, during adolescence are a significant factor. In addition, depression and/or disruptive behavior disorders in childhood or adolescence, including conduct disorder, oppositional defiant disorder, and attention deficit hyperactivity disorder, are predictive of adult-onset BPD. A history of repetitive, intentional self-harm in childhood or adolescence is more common in adults with BPD than the general population [39].

COMMON COMORBID CONDITIONS

Psychiatric Conditions

Persons with BPD have high lifetime rates of other psychiatric disorders, including bipolar disorder (10% to 20%), MDD (71% to 83%), substance use disorder (50% to 65%), panic disorder (34% to 48%), social phobia (23% to 47%), PTSD (47% to 56%), and eating disorders (7% to 26%). Co-occurring personality disorders are also common in BPD, including avoidant (43% to 47%), dependent (16% to 51%), obsessive-compulsive (18% to 26%), and paranoid (14% to 30%) personality disorders [12; 41].

Medical Conditions

While several medical conditions are more common in patients with BPD than the overall population, exact figures on prevalence are difficult to find. Higher lifetime rates of diabetes, cardiovascular disease, sexually transmitted infection, and functional somatic symptoms appear in persons with BPD [42].

PERSONAL AND SOCIETAL COST

BPD exacts a huge toll on afflicted persons in the form of chronic emotional distress and functional impairment, and it imposes significant economic costs to the healthcare system, social services, and broader society [43]. Costs of the disorder include those related to persistent lack of productivity, and numerous behaviors more common among patients with BPD than in those without the disorder, such as reckless driving, domestic violence, incarceration, and pathologic gambling [44; 46]. Contributing to overall economic and personal cost is the negative impact on the clinical course and treatment response of medical conditions and other psychiatric disorders when BPD is present as a coexisting condition [49].

Patients with BPD are heavy utilizers of intensive healthcare services, resulting in higher related healthcare costs than patients with other personality disorders or MDD [50; 51]. Following suicide attempts or intentional self-injury, patients with BPD are typically hospitalized, and such episodes result in an average hospital stay of 6.3 days per year and roughly one emergency room visit every two years; these rates are 6 to 12 times higher than those of MDD [50; 52; 53]. Relative to patients with MDD, those with BPD are more likely to use almost every type of psychosocial treatment (except self-help groups) and most classes of psychotropic medications [50]. However, a prospective six-year study of patients with BPD found that while rates of hospitalization and day or residential treatment were high at study initiation, they significantly declined over time. Similar patterns were observed for intensive psychotherapy, while use of less intensive psychosocial therapy and polypharmacy remained stable during follow-up. At any time during the six-year period, 40% of patients took three or more concurrent medications, 20% took four or more, and 10% took five or more. Thus, outpatient utilization remained constant and inpatient utilization slowly declined over time [54].

Symptom severity in BPD is associated with healthcare use and costs. In primary care patients with BPD, severity of symptoms predicted increased use of primary care resources [43]. The number of borderline symptoms in male veterans with BPD is directly associated with levels of psychiatric comorbidity; suicidal and self-harming behavior; use of healthcare resources (including inpatient admission, outpatient visits, and emergency department visits); and rates of incarceration [56].

PATHOGENESIS AND PATHOPHYSIOLOGY

The understanding of BPD etiology has grown in complexity by incorporating the influences of biology, the environment, and their interaction. Earlier accounts of causation were based on prevailing psychoanalytic theory that emphasized early environment or experiences such as trauma in the development of BPD. With research identifying biologic factors associated with the BPD phenotype, modern conceptions of etiology have addressed constitutional factors associated to distinct genetic and neurophysiologic characteristics and how they interact with specific early life stressors to result in BPD [18].

PATHOGENESIS AND EARLY ENVIRONMENT

The following section addresses early environment factors that contribute to development of the core BPD features of disturbed attachments and interpersonal hypersensitivity. A later section will address the pathogenesis of emotional dysregulation.

Infant and Child Factors

It is essential to note that BPD is no longer viewed as solely the result of parental or primary caregiver behaviors that shape passive, inert children. It is now recognized that innate temperament and behaviors in a child influence parental behaviors by passively evoking parental behaviors and by actively soliciting certain types of parental interactions [57; 58]. This has been demonstrated in twin studies that found elicitation of maternal warmth was substantially controlled by child temperament [59].

A temperament that predisposes sensitivity to interpersonal stress contributes to the development of BPD. In these infants and children, heightened distress states may trigger fearful response in a vulnerable, depressed, anxious, ill, or traumatized caregiver, further diminishing his or her already compromised availability to the child. In particular, child traits of interpersonal hypersensitivity and stress reactivity evoke parental reactions of fearfulness or helplessness and withdrawal, which significantly affect the vulnerable child. Parents of a pre-BPD child are likely to exhibit adverse responses when confronted by increasing neediness or anger in the child, with child and parent factors both contributing to an escalating series of negative and difficult interactions that contribute to adult BPD [60].

Separation Distress and Ambivalent/Disorganized Attachment

Proneness to distress, particularly at separation, is a core feature of ambivalent and disorganized attachment patterns, the childhood counterparts of adult BPD attachment dysfunction. Infants with insecure attachments show greater distress-prone temperaments and irritability and are more likely to express the ambivalent form of attachment. Ambivalent infants engage in hyperactivation behaviors intended to elevate their visibility and increase engagement from an inconsistently attentive parent; these behaviors include clinging, anger, resistance to contact, and failure to soothe in the presence of their parents. Most ambivalently attached children also show the features of disorganized attachment, while a subgroup of infants with disorganized attachment exhibit the amplified distress and difficulty in soothing that is observed in ambivalent attachment. This latter infant subgroup has a heightened vulnerability for developing BPD. Thus, infants born with highly distress-prone temperaments and raised under non-optimal conditions of parental attention and interaction are at greater risk of evolving into ambivalent and/or disorganized attachment and BPD [63].

Disorganized Attachment

Disorganized attachment is the precursor to unresolved attachment, one of two attachment forms in BPD. Its expression mimics the relational style of adults with BPD and involves contradictory approach and avoidance with dissociative responses to caregivers. Roughly 15% of infants show disorganized attachment patterns by 1 year of age, which predicts controlling patterns of attachment relations by 3 to 6 years of age and behavior problems when entering school. Infant use of disorganized attachment strategies is heightened when raised in environments of low socioeconomic status (24%), parental psychopathology (30% to 60%), and infant maltreatment (60% to 70%). Higher cortisol stress responses are found in infants with disorganized versus organized attachment strategies, reflecting a genetic basis of the serotonergic abnormalities and high cortisol responses to separations found in adults with BPD [65; 66].

Parental/Caregiver Factors

Early psychoanalytic theorists identified problems with separation from caregivers as a developmental failure central to vulnerability for BPD, with insecure attachment and traumatic separation experiences the pathogenic factors that accounted for abandonment fears in BPD [68; 69]. Subsequent empirical investigation confirmed the association between dysfunctional early caretaking experience (e.g., frequent separations, parental over- or underinvolvement) and BPD diagnosis [70; 71]. These previous findings have been substantively enhanced and refined by more recent investigations, including the identification from multiple lines of evidence of specific caregiver contributions to childhood development of early attachment disturbance and interpersonal hypersensitivity.

Important contributions have come from reports by patients with BPD, who typically report experiencing very difficult primary attachment relationships in childhood. Common and recurrent themes include emotional neglect from both parents; parental invalidation of their thoughts and feelings;

parents or primary caregivers who were emotionally withdrawn, who were inconsistent, who failed to protect them, or who were over-controlling; and early separation from their primary caregiver [72; 73]. On the other hand, parents of patients with BPD often provide an account that is considerably less critical, and siblings of patients with BPD are commonly much better adjusted. The recollections by patients with BPD of their early family life should not be dismissed, but accepted with a degree of caution [60].

Caregiver Effects on Infant Attachments

Studies of twins have found that social adaptation in adulthood has a stronger association with infant attachment strategies toward their primary caregiver than toward other caregivers, and this finding is unchanged when the primary caregiver is not biologically related to the infant. Other research has shown that in 70% of cases, infant attachment pattern with the primary caregiver is highly correlated with caregiver attachment style before the infant was born. Attachment style is not heritable, and while caregiver behaviors do not sufficiently account for child attachments, they remain very important [65; 74; 76].

Parental Contributions to Disorganized Attachment

Disorganized responses of infants to parents are thought to indicate an approach-avoidance dilemma, or fear without solution, where the parent is both a source of fearful arousal and the only source of comfort from this arousal. Specific maternal responses to distress-prone infants engaging in the hyperactivation strategy have been identified. Referred to as aversive responses, these include withdrawal, fearful disorientation, role-reversal, negative-intrusion, and contradictory responses and bear great resemblance to the recollective accounts of adult patients with BPD [32; 60].

The infant reacts to this dilemma with contradictory attachment responses that include crawling toward the mother while crying and then collapsing on the floor midway, or calling out to the door during separation and then retreating away from the door at reunion. These contradictory infant responses substantially mirror the contradictory need/fear components expressed in the prototypical interpersonal behaviors of adults with BPD, and multiple studies have validated the strength of this association [65; 78].

Disrupted affective communication and emotional withdrawal in mothers with infants at 18 months of age have predicted the borderline personality traits of unstable relationships and self-harming behaviors in early adulthood, and disrupted maternal affective communication has been correlated with infant disorganization and maternal unresolved attachment [78; 79]. These associations remain after controlling for abuse exposure and suggest that parent-child affective communication, independent of abuse history, may play an important and independent role in the development of adult BPD [60].

Parental Psychopathology

Psychopathology is highly prevalent in the parents of patients with BPD, although it is difficult to determine the exact prevalence due to limited studies. The prevalence of maternal BPD is 10% to 15%. These mothers are more insensitive to their infants at 2 months of age, and their children are likely to show disorganized attachments at 1 year of age. Also prevalent in parents of patients with BPD are substance abuse, depressive disorders, eating disorders, and antisocial or other personality disorders [22; 80; 81]. An estimated 30% are negative for psychiatric disorder history [60]. The prevalence of affective, impulsive, and interpersonal phenotypes is increased in families of patients with BPD, including findings that 50% of relatives have affective instability, 33% have impulsivity, and 28% have disturbed relationship styles comparable to their borderline offspring with BPD [22; 82; 83].

Given the high frequency of psychiatric illness and familiality of borderline phenotypes, many parents of pre-BPD children are highly probable for under-reactive or hypersensitive predisposition in their response to their infant's predisposed temperament of distress proneness and interpersonal hypersensitivity. Development of insecure attachment in children is predictive of parental insecure attachment style, and maternal mental illness has been found strongly associated with insecure, disorganized attachments in their children [32]. These findings are consistent with the pathogenic effect of parental psychopathology on their pre-BPD children.

Evolution of Infant Disorganization in School-Age Children

The developmental pathways of BPD that evolve from early, disorganized attachment are complex, and the knowledge base is incomplete. However, some areas of research have been more thoroughly investigated, including controlling attachment strategies.

Controlling Attachment Strategies

Roughly 65% of children with disorganized attachment undergo a change between 18 months and 6 years of age, whereby attachment becomes organized around the goal of controlling interaction with the primary attachment figure. This is likely an adaptive response designed to increase dysfunctional parent involvement (i.e., alleviate parental inability to meet the child's comfort and security needs) [40]. After 18 months of age, dysfunctional parenting is more likely to be personalized in children with negative emotion as anger at the parent, which a hypersensitive parent may experience as personally rejecting. Controlling behaviors of these children toward their caregiver have been observed as young as 3 years of age [40].

The transition to controlling strategies involves two different forms. With controlling-punitive behavior, the child responds to attachment threats by attempts to control the parental relationship through hostile, coercive, or subtly humiliating behaviors. Controlling-caregiving behavior involves entertaining, organizing, directing, or giving approval to the parent. These two forms of controlling behavior are not mutually exclusive, and many children shift back and forth from devaluing, insulting comments to solicitous approval behavior toward the parent. The link between controlling parental strategies in preschool with teacher-reported behavior problems by 4 to 6 years of age is well-established, and controlling parental behaviors are associated with diagnosis of oppositional defiant disorder [32; 40; 65; 89]. Prospective data are lacking, but adult patients with BPD frequently report caregiving or punitive controlling behaviors toward their parents in childhood [40; 60; 90].

GENETICS AND HERITABILITY

A genetic basis of BPD was identified in several family studies that found family loading for the disorder and significantly higher prevalence of BPD in first-degree relatives of patients with BPD than in the general population [81]. Research from a 2019 total population study estimated aggregation and hereditability among family members, which showed a pattern of decreased familial association with genetic relatedness [47]. The concordance hazard ratio of BPD was 11.5 for monozygotic twins and 7.4 for dizygotic twins [47]. Among full siblings, the hazard ratio indicated a 4.7 times greater risk of BPD, compared with maternal half-siblings (2.1 times) and paternal half-siblings (1.3 times). Cousin relations were also part of the study, and it was found that the hazard ratio was 1.7 for cousins whose parents were full siblings, 1.1 for cousins whose parents were maternal half-siblings, and 1.9 for cousins whose parents were paternal half-siblings. Heritability has been found to range from 44% to 60%, with individual specific environmental factors accounting for the remaining variance.

This extent of genetic influence exceeds that of anxiety disorders and depression, but is less than that of bipolar affective disorder or schizophrenia [18; 47; 95].

Molecular genetics research has associated BPD with polymorphisms in genes involving serotonergic and dopaminergic systems. Although much research has focused on serotonin transporter gene (*5-HTTLPR*) polymorphism, often seen in individuals with anxiety and depressive symptoms, the evidence associating *5-HTTLPR* with BPD has been limited and inconsistent. However, one study involving children with BPD showed that carriers of the short allele of *5-HTTLPR* exhibited the highest levels of BPD traits, even after controlling for the substantial co-occurrence between BPD and depressive symptoms [18; 96].

The unique contributions of environmental and genetic factors to BPD etiology are important and their interactive effects essential. Specific predisposing stressful elements in the early environment interact with genetic factors to confer varying degrees of vulnerability to BPD development. The genetic influences that contribute to BPD development also increase the risk of exposure to specific stressful events [99]. For example, genetic factors influence stress reactivity, making some people more likely to develop symptoms following traumatic or chronic stress [100; 101].

NEUROBIOLOGY

Neuroscience research has identified specific brain and neurohormonal abnormalities that underlie core characteristics of BPD. The neural basis of emotional dysregulation in BPD involves hyperreactive limbic structures, primarily the amygdala, with a distinct dysfunction in prefrontal and frontolimbic activity as the larger mechanism [103; 105; 106]. Emotion intensity and dysregulation in BPD results from failures in top-down frontal control processes that would normally modulate the effects of bottom-up hyper-reactive limbic structures. A meta-analysis of neuroimaging literature concluded that subjects with BPD show [107]:

- Activation of a diffuse network of structures in negative emotion processing extending from the amygdala, anterior cingulate cortex, medial and dorsolateral prefrontal cortex, superior temporal gyrus, posterior cingulate cortex, and cerebellum
- Decreased activation of regions extending from the amygdala to the anterior cingulate and dorsolateral prefrontal cortices
- Increased activation of greater insula and posterior cingulate cortex

Reduced anterior cingulate cortex activation in negative emotion processing distinguishes BPD from MDD [108]. Insular cortex activity may underpin a range of symptom sectors in BPD, as this brain region is involved in the processing of emotional and physical pain, self-awareness, and social cognitive processes involved in empathy and adherence to or violation of social norms. Differences in neural activity in the insular cortex have been demonstrated in the context of difficulties among individuals with BPD to sustain and repair cooperation during social exchange [18; 109]. Neuropsychologic testing identifies functional alterations with brain region or neuropathway specificity and has confirmed functional impairment in the prefrontal, temporal, and parietal cortices [110; 111].

Neuropeptide research helps identify maladaptive brain processes involved in stress response and interpersonal sensitivity. As discussed, persons with BPD are typically exposed to high levels of stress during childhood from unstable and insecure attachments. Stress from fragmented insecure attachments interacts with and is amplified by genetic factors, and high stress levels continue through adulthood. Elevated cortisol response to psychosocial stress in subjects with BPD reflects physiologic alteration in stress management [112].

Endogenous opioid and oxytocin neuropeptide systems also mediate stress response and facilitate prosocial tendencies. Persons with BPD show functional alteration in both systems. In healthy individuals, oxytocin administration enhances interpretation of mental states from social cues or "mind-reading" and collaboration in social exchange tasks. But in people with BPD, oxytocin administration paradoxically increases mistrust and decreased cooperation in a social exchange, suggesting the oxytocin system in BPD increases mistrust and interpersonal instability in social activity [113; 114; 115].

Individuals with BPD often display both altered endogenous opioid function and non-suicidal self-injury behavior. Thus, investigation of a possible neurochemical basis of non-suicidal self-harm behavior in BPD was performed by comparing samples of cerebrospinal fluid from patients with BPD with or without a history of non-suicidal self-harm. The non-suicidal self-harm group showed significantly lower levels of the endogenous opioids β-endorphin and met-enkephalin, which bind and activate receptors that mediate stress-induced analgesia and physical pain analgesia, respectively. No differences were found in markers of serotonergic and dopaminergic function [116].

Endogenous opioid system dysregulation may result from chronic and severe childhood stress and trauma (from abuse, neglect, and loss) or from biologic predisposition. Chronic stress can blunt endogenous opioid response to acute stress, and severe physical or psychologic traumas may lead to permanent deficiency states or habituation to higher levels of endogenous opioids [117]. Childhood trauma and non-suicidal self-harm have a high co-occurrence [37; 120]. Patients with chronic stress response to early abuse or neglect may require elevations in endorphin levels for stress coping, and non-suicidal self-harm may increase endogenous opioids to restore homeostasis. The numerous reports that non-suicidal self-harm in persons with BPD is followed by mood enhancement with decreased negative affect, increased positive affect, and increased dissociative symptoms suggest that self-injury serves as self-healing through brief restoration of positive affect. While pharmacotherapy with the opioid modulator naltrexone has not shown consistent benefit, buprenorphine is an opioid modulator that differs from naltrexone in pharmacologic action and may be helpful in curtailing non-suicidal self-harm in patients with BPD [116].

Thus, early environment interaction with endogenous, heritable factors contributes to BPD pathogenesis and clinical presentation. While early childhood adversity, primary caregiver dysfunction, and overall early life stress are likely highly robust factors in the development of BPD, genetic factors and brain and neuropeptide system regulatory dysfunctions may represent the catalyst for susceptibility to pathologic responses to environmental life stress [18].

DEVELOPMENTAL NEUROSCIENCE

Emotion regulation is mediated by frontolimbic brain regions that include, among other structures, the amygdala, hippocampus, hypothalamus, dorsolateral and right dorsomedial prefrontal cortex, orbital frontal cortex, anterior cingulate cortex, and insula [121]. These and other structures are interconnected by function and structure and are recruited to modulate subcortical responses to emotional stimuli and inhibit behavioral impulses [122]. Dysfunction of this circuitry contributes to emotion dysregulation. Emotion regulation is best understood as an individual and interpersonal process that begins with early attachment and continues to later peer and romantic relationships. Early attachment and interpersonal relationships distribute the effort of emotion regulation through co-regulation [123].

Early attachment figures and relationships are the initial source of co-regulation. Bonding usually occurs quickly and unconditionally during a period of rapid development when neural links are being formed between the prefrontal cortex and structures that underlie emotion and memory including the amygdala, nucleus accumbens, and hippocampus [124]. These early relationships appear to have lasting effects on attachment style and emotion regulation. The caregiver-child relationship is the first experience in which the child learns to influence the caregiver's emotions and behaviors, and the child experiences self-regulation of behavior and emotions through caregiver actions [123].

This early attachment status predicts emotion regulation abilities and attachment style in adulthood. The developing frontolimbic system is sensitive to social inputs and structurally encodes expectations of distress alleviation and security provision from attachment figures [126]. During development, the amygdala tags emotional stimuli, while the hippocampus consolidates the associated contextual cues into long-term memory. Through this process, the behavior of attachment figures becomes stored as neural representation. The amygdala is also sensitive to signs of threat and, through input to the hypothalamus, functions to regulate stress hormones and facilitate social soothing [127].

Reciprocal projections between the prefrontal cortex and the amygdala, hippocampus, and hypothalamus contribute to memory formation and conditioned learning, including the appraisal of emotional stimuli and activation of appropriate motivated behavior [121; 128]. Through this process, conditioned responses to attachment figures are encoded within medial, orbital, and dorsolateral circuits of the prefrontal cortex to serve as markers of threat or protection. These associations are strengthened through dopaminergically mediated experiences of security [126]. Oxytocin activity in the hypothalamus, nucleus accumbens, ventral tegmentum, and amygdala influence attachment security, while increased endogenous opioid activity in the anterior cingulate cortex mediates sensitivity to and greater distress from social rejection, representing another mechanism by which attachment experiences are encoded within frontolimbic circuitry [124; 130].

Throughout childhood, the regulatory effects of the child-caregiver bond occur in the presence of the attachment figure and through the neural representation of caregiver availability in response to threat. A secure attachment between child and caregiver promotes reasonable assumptions of co-regulation and a confident sense of "the self" in relation to key attachment figures [123].

Abused or neglected children are more likely to display antisocial, aggressive, withdrawn, and disruptive behaviors during play interactions. Children with antisocial or aggressive behaviors are often viewed as mean or attention seeking and tend to be disliked by their peers [48]. The resultant rejection and enduring negative reputations within social groups produces functional changes in insular, ventrolateral prefrontal cortex, anterior cingulate cortex, and ventral striatum activation in adolescents. Thus, problematic behaviors and peer affiliations reinforce maladaptive attachment assumptions and decrease the likelihood of receiving effective co-regulation through friendships. Peer rejection may produce lasting biologic adaptations within these frontolimbic circuits. By late adolescence, BPD can be diagnosed reliably [123].

EMOTION DYSREGULATION

Emotion dysregulation is a core pathologic feature of BPD, and some researchers regard emotion dysregulation as the single foundational element. This section discusses the interactive effect of environmental, heritable, and neurobiologic factors that form the basis of emotion dysregulation development.

Emotion dysregulation is a complex process of multiple interactive genetic and environmental components that begins in infancy and develops over the lifetime. The widely used definition of emotion dysregulation as the inability to flexibly respond to and manage emotions is overly broad and non-specific to BPD, lacking in nuance, and a hindrance to BPD research and clinical practice. Emotion dysregulation is characterized by four components: emotion sensitivity, negative affect, deficient appropriate emotion regulation strategies, and maladaptive emotion regulation strategies [136].

Emotion Sensitivity

In BPD, emotion sensitivity is thought to have a biologic origin with presence in early life. It involves heightened emotional reactivity to environmental stimuli and is primarily associated with negative mood states such as anger, fear, and sadness. This heightened emotional reactivity also involves the emotions of others. Emotion recognition studies in BPD show negativity bias in emotion recognition (i.e., negative emotions in others are over-identified) and poor accuracy in correctly identifying facial emotions [137; 138].

Negative Affect

BPD is strongly associated with high levels of negative affect on dimensions of intensity and reactivity. High negative affect in BPD is considered secondary to emotion sensitivity such that hyper-reactivity to environmental stimuli triggers rapid changes in mood [140]. In BPD, the essential features of negative affect are instability over time and rapid intensification with little warning. Comparisons of BPD with healthy controls found that subjects with BPD showed significantly greater instability on measures of emotional valence and distress, greater propensity for large decreases in positive mood, and a substantially greater proportion of rapid decreases in positive mood leading to negative mood states [48]. Compared with persons with MDD, subjects with BPD show significantly greater levels of variability in positive and negative affect; instability in hostility, fear, and sadness; and extreme changes in hostility [143].

Deficient Appropriate Emotion Regulation Strategies

Persons with BPD did not learn the skills necessary to regulate emotion as emotionally sensitive children experiencing heightened negative affect. As a result, they face great difficulty in controlling their emotions and timing its expression [48].

Ability to identify one's emotions is important for emotion regulation. Related to emotion awareness is capacity to distinguish among emotional states, termed emotional granularity. Persons with high emotional granularity can reliably and accurately differentiate emotional states (e.g., sadness from anger); those low in emotional granularity often describe emotional states in global terms, such as feeling good or feeling bad. Persons with BPD have shown deficient emotion awareness, low emotional granularity (as evidenced by poor emotional clarity and mood and emotion labeling), and greater affect polarity ("all-or-nothing" thinking) [145; 146].

Poor distress tolerance, pervasive in BPD, is an artifact of deficient emotion regulation strategies, and both contribute to the development of BPD. Negative emotionality is the heritable tendency to experience negative affect and may be influenced by emotion sensitivity bias. Distress tolerance protects against developing BPD symptoms, to a much greater extent in persons with intense negative affect than with emotion hypersensitivity. Patients with BPD with poor distress tolerance and high levels of emotion hypersensitivity or intense negative affect are significantly more likely to exhibit impulsive and reckless behavior as maladaptive emotion regulation strategies [147].

Maladaptive Emotion Regulation Strategies

Maladaptive behaviors to regulate emotion can lead to emotion dysregulation problems very obvious to others. If negative affect becomes sufficiently intense, the person will likely choose maladaptive over adaptive behaviors. While maladaptive behaviors can produce immediate reduction in negative affect and are simpler to employ than adaptive behaviors, they have negative consequences and can become ineffective with

long-term use [148]. The maladaptive cognitive strategies of rumination and thought suppression, often used in BPD, actually increase negative affect in the long term [149; 150]. Experiential avoidance is also common in BPD and is characterized by behaviors to escape unwanted experiences [151]. The impulsive, suicidal, and self-injurious behaviors common in BPD are behaviors specifically used to regulate affect [136].

The highly prevalent impulsive behaviors in BPD, such as disordered eating, impulsive buying, and drug use, serve to reduce negative affect. Psychometric studies strongly associate BPD with urgency/impulsive actions during negative mood (to alleviate negative affect) [152]. BPD uniquely clusters in high levels of affect instability, urgency, and absence of premeditation [153]. Neurobiology studies show altered ventromedial prefrontal cortex function with BPD, consistent with impulsivity in the context of negative affect [106]. Affective instability with identity disturbance and impulsivity has predicted suicidal behaviors; affective instability with childhood sexual abuse predicts suicide attempts [154].

Various forms of self-injurious behavior are common in patients with BPD, with a prevalence rate of 20% to 90%, with the most common being self-injury by cutting or burning. It is almost always reported to reduce feelings of negative emotion and help control mood [55; 61]. Those with BPD report little or no pain during self-injurious behavior or during pain-induction tasks, an effect even more pronounced during distress [158]. Brain imaging has found a negative coupling between paralimbic and prefrontal brain regions in subjects with BPD, suggesting that prefrontal areas inhibit paralimbic regions following pain in this group [136; 159].

NATURAL HISTORY

Until recently, considerable pessimism surrounded the long-term prognosis of BPD and the capacity of patients with BPD to benefit from treatment and become less symptomatic and self-destructive. This negative impression of patients with BPD has been challenged by the findings of longitudinal studies [18].

EMERGENCE AND ANTECEDENTS OF BPD

While the exact age of BPD onset is uncertain, children younger than 12 years of age have met full criteria for BPD, and the incidence continues to increase up to early adulthood. The impulsive, aggressive, and self-destructive behaviors of BPD tend to emerge during or before adolescence and remain stable [18].

Borderline personality-related characteristics can be observed in children and are associated with increased risk of developing BPD. The extent that childhood borderline personality-related characteristics share etiologic features with adult BPD was studied in 1,116 pairs of same-sex twins, followed from birth through 12 years of age. The results found that borderline personality-related characteristics at 12 years of age were heritable within the range of estimates for adult BPD and were preceded by poor cognitive function and behavioral and affective dysregulation at 5 years of age. Exposure to harsh treatment (i.e., physical abuse or maternal negative expressed emotion) by 10 years of age predicted borderline personality-related characteristics at 12 years of age, and this was further heightened in children with a positive family history of psychiatric disorder. The authors concluded that inherited lability and harsh treatment both contributed to borderline personality-related characteristics, and each acted as a more virulent risk factor in the presence of the other [160].

ADOLESCENCE AND EARLY ADULTHOOD

Earlier in life, patients with BPD are likely to have been emotionally unstable, impulsive, and hostile. Although normal adolescence often involves rebellion or identity diffusion, the development and intensity of adolescent BPD traits may precipitate involvement in rebellious groups. These affiliations may heighten the risk of exposure to negative interpersonal elements and development of substance use disorder or PTSD. Adolescent BPD may also contribute to development of eating, mood, and/or anxiety disorders [161; 162].

As they enter adulthood, persons with BPD may undergo multiple hospitalizations resulting from poor impulse control, suicidality, or quasipsychotic and dissociative symptomatology. As discussed, BPD decompensation accounts for a sizeable proportion of psychiatric hospitalizations. Employment history is often characterized by multiple job losses or career changes, and interpersonal relationships are continually volatile and chaotic. Fluctuations in gender identity, sexual orientation, and personal values are common, reflecting cognitive distortions and fragmented sense of self. By their 30s, affective instability and impulsivity generally begin to lessen, and forming a relationship with a supportive and patient sexual partner or simply retreating to a more isolated lifestyle may promote earlier stabilization of disruptive emotional lability [161; 162].

COURSE OF PSYCHOPATHOLOGY

Earlier studies following the course of psychopathology in adult patients with BPD after hospitalization reported minimal improvement and a corresponding negative prognosis. However, these studies possessed flaws in study design, and several more recent studies provide a more accurate depiction of the long-term course of BPD [18].

The McLean Study for Adult Development (MSAD) and the Collaborative Longitudinal Personality Disorder Study (CLPS) found dramatic and unexpectedly greater remission rates than anticipated of BPD psychopathology, as measured by decreases in BPD criteria and by an operationalized definition of remission [163; 164]. Unexpectedly, patients with BPD who remitted were likely to remain remitted. In the CLPS, only 12% in remission relapsed [164]. Previous studies with shorter follow-up periods showed worse prognosis, perhaps because relapse rates are higher during the first few years of release from hospitalization [18].

Both studies examined the pattern of BPD symptom reduction. Four-year outcome data from the CLPS showed that certain criteria (e.g., self-harm) diminished more rapidly than other criteria (e.g., affective instability) [164]. Similar results were reported from the MSAD, in which impulsive characteristics and intense unstable relationships resolved earlier than loneliness/emptiness and intolerance of aloneness [163; 165]. It appears from these findings that symptomatology of BPD can remit but that disturbances in core personality functioning persist substantially longer.

COURSE OF SOCIAL FUNCTIONING

Findings related to the course of social adaptation in BPD natural history are more discouraging. The Global Assessment of Functioning scores (100 = optimal functioning) of subjects with BPD remained low (average: 65), with rates of disability support remaining stable. At 10-year follow-up, around 30% had full-time vocational activities and were married or had stable partnerships. However, the overall scores rose significantly on measures of self-satisfaction, recreation, and friends. A 16-year follow-up study reported that 60% of subjects had "recovered," defined by durable remission and sustained partnerships. This report gives a more hopeful perspective concerning sustained and substantive changes [18; 163; 164].

MEDIATORS AND MODERATORS OF COURSE

The neurophysiology of patients with BPD is primed to be stress responsive, which helps explain the findings that remissions frequently occur when people with BPD leave highly stressful situations and rapid improvements in composure and sociability when patients with BPD are placed in low-stress, asylum-like settings. Highly stressful events that are interpersonal in nature, such as rejection, precede and predict self-harm, suicidality, dissociation, and relapse [166; 167].

More severe BPD psychopathology, lower functioning, and a history of childhood sexual abuse were predictors of slower symptomatic recovery in the CLPS, while older age, childhood sexual abuse, family history of substance use, and greater vocational impairment were predictors of slower symptomatic recovery in the MSAD [168; 169]. Some of these predictors may have been moderators (e.g., childhood sexual abuse) and others mediators (e.g., low functioning) of natural history [18].

Longitudinal data also found high levels of healthcare service utilization, with gradual reduction in the use of emergency rooms, inpatient admissions, and other expensive services [50; 54]. Patients with BPD are heavy users of medications and polypharmacy, but a higher number of medications is correlated with worse clinical course. This may reflect over-reliance on medication as treatment despite modest (at best) benefit. Underuse of appropriate psychosocial therapies may contribute to a worsening clinical course, compelling the prescription of additional medications [18].

BARRIERS TO CARE

The full expression of BPD psychopathology occurs in the interpersonal context, and patients with BPD impose a legitimate and, on some dimensions, unique challenge to providers involved in their care. The propensity of patients with BPD to attempt suicide is probably the greatest source of provider stress. Patients with BPD are especially prone to feeling rejected and then reacting with rage, and the manner by which patients with BPD may endanger their lives can be unusually distress-provoking [170]. Difficult patient characteristics and prognostic pessimism have contributed to discrimination and bias in broader society and in the mental health system.

DISCRIMINATION AND BARRIERS TO TREATMENT

BPD is stigmatized to a greater extent than other psychiatric disorders, partially due to the misconception that BPD reflects a moral failing—a belief that the person with BPD should have control over his or her behavior. An extension of this misconception is that patients with BPD differ from those with "purely biologic" conditions, such as depression or schizophrenia. The stigma attached to BPD has also led to the use of therapy approaches non-specific to BPD that fail to address the unique psychopathology. Predictably, patients do not improve and may worsen, which reinforces negative clinician attitudes toward these patients [171].

The characteristic features of anger, suicidality, and vacillation between extremes of idealization and devaluation help contribute to the widespread attitude that patients with BPD are "difficult," "noncompliant," "manipulative," "troublemakers," "unresponsive," "impossible," and other pejorative descriptions [6; 172]. The symptoms that create difficulty and challenge for providers are the same that interfere with patient ability to maintain treatment relationships despite a desire to do so. Traits of BPD that lead to unstable and

stormy interpersonal relationships can have the same effect on therapeutic relationships, creating clinician difficulty in establishing rapport and alliance and resulting in early treatment termination by the patient [8].

Patient Experience After Diagnosis

BPD is almost universally described as isolating by patients and their families, and this isolation also extends into the clinical setting. An interview of patients with BPD was conducted to better understand patient experience in receiving the diagnosis of BPD. Five themes emerged [173]:

- Knowledge as power
- Uncertainty about what the diagnosis meant
- Diagnosis as rejection
- Diagnosis as "not fitting"
- Hope and the possibility of change

Some patients reported a feeling of clarity, focus for the future, and a sense of control, as the BPD diagnosis provided something tangible they could grasp. Others felt out of control with the diagnosis due to a lack of understanding when providers were not forthcoming with information and not communicating any hope for recovery. Relief or distress after receiving the diagnosis was related to the extent that patients felt empowered with the knowledge and what they could do to recover. Unfortunately, the subjects consistently reported feeling that receiving the diagnosis was quickly followed by a withdrawal of services [173].

The theme of rejection in the clinical setting is reported throughout the literature. A primary contributor may be the diagnosis itself. Many clinicians believe that treating the personality of a patient is impossible, resulting in persons with BPD receiving mental health care only during a crisis for exacerbated symptoms instead of ongoing treatment that addresses the full dimension of the disorder. Use of mental health services during crises is time-limited and brief, and the absence of lasting benefit from the short duration of therapy reinforces professional views that BPD is untreatable, which strengthens the associated stigma. Many patients diagnosed with BPD report feeling stigmatized during contacts with the mental health system, with staff assuming the patient is difficult, manipulative, and attention seeking. Many also report feeling blamed for their condition when the greatest message they seek is one of legitimacy and basic acceptance [8; 174].

Families often feel just as blamed by clinicians for their loved one's illness, and despite the growing body of evidence of substantial heritable and innate contribution to the development of BPD, there remains an unwavering view by some professionals that someone is culpable for a person's BPD [8; 174]. The misconception of BPD as solely the result of environmental stressors such as childhood abuse and trauma had been so persistent and salient that some therapists have used "recovered memory therapies" in treating BPD [12; 175].

A positive development in the United States has been the increased attention on and consideration of the perspective gained from those with psychiatric diagnoses and their families. Although not yet universally embraced or valued by mental health systems and professionals, many have come to appreciate the importance of their input when applied to treatment and service design, delivery, and evaluation. A byproduct has been the redefined relationships between mental health consumers/family members and professionals and the reduction in discrimination and bias [8; 176].

Combating Discrimination and Bias

The single greatest obstacle for people with BPD is overcoming pervasive misconceptions regarding the nature, causes, diagnosis, and treatment of the disorder. At the individual patient level, clinician knowledge of contributory environmental factors can offer important insight and guide treatment decisions, but it should not be the sole or even the primary determinant in treatment selection. Approaching the patient with BPD as someone afflicted with a brain disorder that requires fixing

obscures the important experiential factors of the patient, such as his or her attachment of meaning and importance to past events. Person-centered care has received increased attention as an element of overall healthcare reform and is essential in fostering recovery for BPD. A person-centered approach also interrupts the life-long pattern in which patient perception of experiences, and of self, has been defined and labeled by others. Providers should understand that even destructive behavior has meaning and represents communication by the only means the patient has. The most effective interventions are compassionate, reinforcing of patient dignity, and grounded by shared respect. While patients may require different approaches at different points in recovery, the most important approach at any stage is to view them as humans who need a compassionate relationship that embodies hope and healing [8].

Healthcare providers can create a more comfortable environment for a patient of another culture by acknowledging the impact of culture and cultural differences on physical and mental health. Symptom presentation is influenced by cultural factors, and this should be taken into account during the assessment process.

BARRIERS TO TIMELY AND ACCURATE BPD DIAGNOSIS

Recurrent suicidal threats or actions in response to fears of abandonment are by themselves strongly indicative of a BPD diagnosis. However, BPD remains underdiagnosed, and often misdiagnosed, in large part because the characteristic recurrent crises, emotional volatility, and self-injurious behavior are perceived as willful manipulative choices rather than expressions of illness [49; 62; 77]. A substantial gap exists between the education and practice of mental health care, and the current educational system for mental health professionals does not pay adequate attention to BPD or other personality disorders. One study found an average 10-year time gap between initial presentation for treatment and accurate diagnosis of BPD [178]. With this time delay comes unnecessary suffering, wasted treatment efforts, and, with a 10% mortality rate from suicide in patients with BPD, tragic and potentially preventable fatality [171; 179]. Several factors contribute to delays in accurate diagnosis of BPD, including stigma, reliance on pharmacologic treatments, desire for a clear-cut diagnosis, and costs.

Stigma

As noted, diagnosis of BPD can lead to rejection by the mental health system, resulting in clinician reluctance in making a diagnosis associated with stigma [180]. There can also be a general reluctance by patients and their families to explore the psychologic origins and conflicts related to personality disorders, and this may encourage a diagnosis of MDD or bipolar disorder because a "chemical imbalance" can represent a preferable and more palatable diagnostic conclusion [181].

Reliance on Pharmacologic Treatments

In psychiatry, the dominant emphasis on pharmacotherapy and focus on symptoms contrasts with the greater complexity of a biopsychosocial approach. Implementing the latter is more complicated, but neglecting to do so can lead to delivery of unnecessary treatment of marginal benefit, postponement of necessary treatment, problematic side effects or unanticipated treatment harms, and potentially tragic outcomes [171].

Desire for a Clear-Cut Diagnosis

Many of the former Axis I diagnostic entities, such as anxiety, bipolar, and mood disorders, are more familiar to healthcare professionals, while making an accurate personality disorder diagnosis requires more experience. Clinicians attempting to diagnose the patient with BPD may also tend to aim for a straightforward diagnosis. Simpler constructs, such as MDD, also lead to treatment options with greater clinician familiarity (e.g., pharmacotherapy) [182]. Evidence-based treatments for BPD are more complex, and many require training, skill, and patient involvement. They are also delivered

over extended periods, with recommended times of one year for dialectical behavioral therapy and transference-focused psychotherapy, 18 months for mentalization-based therapy, and three years for schema therapy [183; 184; 185; 186]. Although criticized for their cost and duration, the alternative can be a protracted series of acute treatments that may help the patient survive from crisis to crisis, but without durable long-term benefit [171].

Third-Party Reimbursement

Some insurance companies have had a policy of refusing reimbursement for care for a diagnosed personality disorder, often on the grounds that personality disorders are not conditions of medical necessity. This lack of coverage adds practical problems to the burden of suffering [171].

ASSESSMENT AND DIAGNOSIS

AGE OF APPROPRIATE BPD DIAGNOSIS

The DSM-IV-TR explicitly stated to exercise great caution when diagnosing BPD in patients younger than 18 years of age, largely from the belief that personality and behavioral patterns during adolescence are predominantly transient. In other words, adolescents may "outgrow" borderline symptoms, so diagnosing them before 18 years of age is premature [187; 188]. However, more recent research on BPD and personality development indicates that an adolescent diagnosis of BPD is valid, and ignoring BPD as a possible disorder in adolescents may hamper effective clinical intervention [189]. The typical onset of self-harm before 12 years of age suggests an important window to screen and provide early intervention for these children and their families [30].

In addition to self-harm, early indicators of BPD include body-image problems, shame, the search for exclusive relationships, and intense rejection hypersensitivity. While these signals can occur in adolescents without BPD, and even if these clinical features later attenuate, their presence predicts long-term social disability and a nine-fold increase in risk for adult BPD [191]. In these young patients, treatment should address symptom reduction and, most importantly, should aim to alter the life-course trajectory by promoting the development of alternate adaptive developmental pathways before the core features become intractable [39; 64].

CLINICAL FEATURES OF BPD

BPD is typified by significant impairments in identity, self-direction and interpersonal functioning, and pathologic overexpression of negative affectivity, disinhibition, and antagonism [4]. These pathologic deficits in personality functioning and pathologic personality traits are expressed as intense and disproportionate levels of anger, euphoria, depression, and anxiety, sometimes with rapid switching between mood states. This emotional intensity and instability leads to impulsive behavior, confusion, and shifting long-term goals, career objectives, friendships, gender identity, and values. Not infrequently, persons with BPD feel unfairly treated or misunderstood, bored, empty, and without a sense of who they are. Symptoms can be exacerbated by events in the environment that trigger emotional memory of past trauma or unresolved events [192].

EVIDENCE-BASED PRACTICE RECOMMENDATION

The National Health and Medical Research Council recommends that health professionals should consider assessment for borderline personality disorder (BPD) (or referral for psychiatric assessment) for a person with frequent suicidal or self-harming behavior, marked emotional instability, multiple co-occurring psychiatric conditions, non-response to established treatments for current psychiatric symptoms, and/or a high level of functional impairment.

(https://bpdfoundation.org.au/images/mh25_borderline_personality_guideline.pdf. Last accessed March 24, 2020.)

Strength of Recommendation/Level of Evidence: Practice Point (Point of guidance included in the guideline used to support evidence-based recommendations, where the subject matter is outside of the scope of search strategy, and which were formulated based on expert opinion using a consensus process.)

Their impulsivity often results in highly unstable relationships, with intense attachments and regard for their attachment object that can suddenly shift from great admiration to intense anger or hatred. With hypersensitivity to rejection, their perception of distance or imminent abandonment can trigger an angry reaction, threats, or possible attempts of suicide. These dynamics can also appear in the clinical setting triggered by therapist or provider shift change, sickness, holiday, or sudden change of plans [43].

In the absence of significant symptom expression in adolescence, the full onset of BPD can be triggered by events in adulthood considered normal developmental milestones, such as leaving home or starting an intimate relationship. Sometimes trauma is the triggering event, such as injury in a motor vehicle accident or sexual assault. Such events seem to precipitate the onset of BPD in predisposed persons such that BPD characteristics become fully expressed with attention to their condition for the first time [38; 192].

PSYCHOPATHOLOGIC FEATURES OF BPD

The new proposed diagnostic criteria for BPD in the DSM-5 are reliable and replicable and reflect the observable manifestation of dysregulated interpersonal, behavioral, identity, and cognitive domains [136]. However, the symptom criteria do not fully capture the foundational basis of the psychopathology. Abnormal personality traits in BPD have been attributed to four factors, with each factor representing an underlying temperament or phenotype:

- Interpersonal hypersensitivity
- Affect (emotional) dysregulation
- Behavioral dyscontrol (impulsivity)
- Disturbed self

Interpersonal Hypersensitivity

In the BPD domain of maladaptive and dysregulated interpersonal functioning, the fearful, highly reactive component combines abandonment fears, rejection sensitivity, and intolerance of aloneness to represent the most distinctive and pathogenic component of interpersonal pathology. This is termed the interpersonal hypersensitivity phenotype [60]. A strong genetic basis has been found for this phenotype [47].

The interpersonal hypersensitivity phenotype carries great clinical significance, as dysphoric negative emotional states and the absence of adaptive modulatory ability frequently lead to dissociation and self-injurious behaviors in response to interpersonal events such as rejection or aloneness. Patients with BPD are also hypersensitive to expressions of feeling they perceive in other's faces, with particular sensitivity and physiologic reactions to angry faces. Data from long-term studies show that symptom remission in patients with BPD is very often a response to positive interpersonal events—the characteristically negative emotional responses to interpersonal interactions convert to positive responses [60]. In these remitted patients, relapse is almost always the result of a negative event or outcome in a romantic relationship [60].

Several studies of adult subjects with BPD have found a greater than 90% prevalence of insecure attachment, with the preoccupied type of insecure attachment somewhat higher in prevalence than the unresolved type. The preoccupied type appears in the needy quality of attachments, and the insecure type captures the fearful and contradictory qualities. Hostile-helpless attachments have also shown high prevalence in BPD. With this attachment type, representations of attachment figures are perceived as hostile, untrustworthy, or abdicating of a parental role [40; 124]. These features of BPD have in common a highly negative meaning attached to real or imagined interpersonal slights or insults, especially in the context of important attachment relationships, and represent a core psychologic vulnerability in patients with BPD [60].

Abandonment Fear

Abandonment fears may be confused with separation fears, and patients with BPD also frequently have intense separation anxiety concerning attachment figures. The self-image, affect, cognition, and behavior of the person with BPD can abruptly and profoundly change when he or she perceives an imminent separation, rejection, or loss of external structure. Interpersonal hypersensitivity can result in intense abandonment fears and inappropriate anger, triggered even when confronted by criticism or with a time-limited separation. Abandonment fears stem from an intolerance of being alone and a need to have other people with them, and frantic, impulsive actions to avoid abandonment can include self-injurious or suicidal behaviors [3].

Related to chronic emptiness is the tendency to become overly attached to others and to desperately fear abandonment. This also relates to issues of trust and mistrust, an aspect of BPD that often manifests in family relations and confers an important rationale for family education and intervention. Family members find the intense attachment and fear of abandonment difficult to understand and stressful to deal with, and are often perplexed as to why their repeated reassurances of love and devotion have little effect on the fears of these patients [171].

Emptiness and Identity Disturbance

Chronic emptiness is described as a visceral feeling that is usually felt in the abdomen or chest and plagues the patient with BPD. It is not a feeling of boredom or existential anguish, but a feeling state associated with loneliness and neediness. Some have considered this experience an emotional state, while others describe it as a state of deprivation [3]. A sense of emptiness is often reported by patients with BPD as the very core element of their self-identity. It can be the most painful aspect of the disorder for some and can lead to drug use, promiscuity, or cutting in the effort to fill the void [171].

The characteristics of BPD self-identity, unique to the disorder, involve a distorted, unstable, or weak self-image that is reflected by sudden changes in goals, beliefs, vocational aspirations, and sexual identity. The absence of a clear, coherent sense of self leads to frequent and painful problems stemming from one's values, goals, likes, and dislikes being undefined. This often results in the person with BPD adopting and assimilating the values, habits, and attitudes of whomever they frequently associate. The inability to identify one's feeling states and motives behind one's behaviors (mentalization) are thought to account for the amplification of these identity disturbances in the interpersonal context [15].

Anger, Aggression, and Violence

Aggressive urges become problematic in persons who are unable to integrate aggression into their overall personality structure, usually reflecting modulatory failure by higher cortical structures. Many individuals with BPD report feeling angry much of the time, even when anger is not overtly expressed. Anger is often triggered when perceiving an intimate or caregiver as neglectful, withholding, uncaring, or abandoning. Expressions of anger may be followed by shame and contribute to a sense of being "evil" [3]. In some patients with BPD, innate temperament and environment interact to create an extreme loading of aggressive affect. The distinction between higher and lower level patients with BPD and excessive anger was one of several factors that predicted poor prognosis in a naturalistic longitudinal study of 500 patients with BPD. Factors predicting negative outcome include [171; 179]:

- Heavy loading of aggressive affect
- Antisocial features, including dishonesty
- Secondary gains ("fringe benefits") of illness
- Severely restricted interpersonal relations
- No love life, lack of physical attractiveness
- Low intelligence
- No steady work or study (shifting lifestyle)

- A pattern of negative therapeutic reaction, such as defeating therapists' efforts to prove one is stronger or to obtain gratification from frustrating the therapist (possibly driven by underlying envy)

Anger and aggression are likely to appear in any attachment relationship when abandonment is perceived, but most of all in the intimate relationships of persons with BPD. Perception of emotional distance or physical separation, coupled with intense fear of abandonment and loneliness, can provoke intense anger or rage. Studies of men and lesbians with BPD in treatment for domestic violence have found identical emotional and behavioral processes surrounding anger and violence toward intimate partners. For these patients, violence is used as a strategy to prevent abandonment by maintaining the connection to a partner through coercion and/or fear [194].

Little research has been published on the prevalence of intimate partner violence and BPD in non-clinical populations or in women in general, or comparing gender-specific rates. One study was conducted to explore the relationship between intimate partner aggression and borderline personality using the questionnaire responses of 14,154 college students in 67 colleges worldwide (19 in the United States) [194]. In this study, borderline personality referred to borderline personality organization, defined as similar but less severe core features of BPD, including emotional and behavioral dysregulation, disturbance in identity and self, and interpersonal hypersensitivity [195]. The prevalence of borderline personality was 11% to 15%, a rate three to four times greater than BPD. Borderline personality was used as a surrogate for BPD and was assessed by respondent answers to questions derived from DSM-IV-TR criteria for BPD [194]. The past-year rates of intimate partner aggression were generally greater for women, with the exception of sexual aggression (***Table 1***). These results are remarkable by virtue of the sample size and the extension of behavioral characteristics in clinical populations to community populations. Importantly, the results challenge long-held assumptions of gender differences in the expression of interpersonal intimate aggression. These findings also provide cross-cultural validity for the construct of borderline personality characteristics [194].

RATES OF INTIMATE PARTNER VIOLENCE PERPETRATED BY MEN AND WOMEN WITH BPD

Type of Aggression	Men		Women	
	Any	Severe	Any	Severe
Physical aggression	23.9%	8.0%	31.0%	11.3%
Psychologic aggression	65.7%	20.9%	75.3%	25.3%
Sexual aggression	29.3%	2.2%	21.2%	1.5%

Source: [194] *Table 1*

Splitting

Splitting is the psychologic construct describing the subconscious process that compartmentalizes bad, toxic, and/or terrifying representations of self and other from the good, rewarding, positive, and comforting representations [3]. This aspect of BPD is difficult for others, especially family members, to understand. A reality basis is rare; instead, patient perceptions are filtered by internal images of self and others that are exaggerated, distorted, and superimposed on relationships with others and on themselves [171].

Expression in Relationships with Others

As discussed, a distinctive characteristic of patients with BPD is a hypersensitivity to rejection and fearful preoccupation with anticipated abandonment. While these patients feel their lives are meaningless unless they feel connected to someone they believe truly "cares," their perception of "caring" usually imposes unrealistic expectations of availability and validation from the attachment figure. Within such relationships, an initial idealization can rapidly shift to devaluation when rejection is perceived. The near-universal inability of persons with BPD to perceive attachment objects in terms other than idealized (if gratifying) or devalued (if not gratifying) plays a large contribution to the stormy, chaotic, and unstable relationships of

these persons. It is considered a symptom of early insecure attachment characterized by both fearful distrust and needy dependency [3].

Expression in Relation to Self

In addition to this external "splitting," patients with BPD experience internal splitting. This typically involves vacillation between the viewing of self as a good person who has been mistreated, with anger the dominant emotion, and self as a bad person whose life is without value, with self-destructive or suicidal behavior the dominant expression. Splitting is also reflected in black-and-white or all-or-nothing dichotomous thinking [49; 60].

Psychotic-Like Symptoms

Patients with BPD can experience dissociation symptoms, whereby the feeling and perception of self and/or environment has an unreal quality. These symptoms often occur during situations of extreme stress. These patients can also be unrealistically self-conscious, believing that others are critically looking at or talking about them. These lapses of reality in the patient with BPD are distinct from other pathologies because, with proper feedback, they are usually able to correct their distortions of reality [3].

Impulsivity

Impulsivity in patients with BPD is frequently self-damaging in effect, if not by intent, and differs from the impulsivity found in other disorders, such as bipolar or antisocial personality disorder. As discussed, impulsive behavior is usually driven by the need to escape intolerable negative affect. Common forms of impulsive behavior include substance or alcohol abuse, bulimia, unprotected sexual promiscuity, and reckless driving [3; 171].

Suicidal or Self-Injurious Behaviors

Recurrent suicidal attempts, gestures, threats, or self-injurious behaviors are a hallmark of BPD, and this behavior is so prototypical with BPD that in the absence of other patient background information, recurrent self-destructive behaviors indicate a high probability of BPD. Self-harming acts often start in early adolescence or younger. Self-injurious behaviors or suicidal gestures are usually precipitated by interpersonal stressors, such as threats of separation or rejection, or by misinterpretation of emotion or communication in others as meaning that abandonment is imminent. When present, this clinical feature can greatly assist in the differential diagnosis in patients with dominant features of depression or anxiety [3].

Affective (Emotional) Instability

Early clinical observers noted the intensity, volatility, and range of emotions in patients with BPD, all of which contributed to the belief that emotional instability in BPD involved a variant of affective irregularity in affective and bipolar disorders. It is now known that although individuals with BPD display marked affective instability, these mood changes usually last only a few hours and the underlying dysphoric mood is rarely relieved by periods of well-being or satisfaction. These episodes may reflect extreme reactivity of the patient to stress, particularly interpersonal stress, and a neurobiologically mediated inability to regulate emotions [3].

Patient Depiction of Living with BPD

Description of BPD symptomatology in peer-reviewed published research is often lacking in dimension. The following passage captures the gravity of the experience when a person struggles daily with the symptoms of BPD [192]:

"It's like being on an emotional roller coaster—one minute I'm OK, the next I'm in an uncontrollable rage. I'm terrified that people I am close to will abandon me, and I feel that I will die without them.

My mum left me when I was 3 years old and my dad was an alcoholic who didn't have time for me. Inside I feel like…I must be a horrible person if my parents couldn't love me. The only way I can cope is to either hold on tight or push people away before they have the chance to hurt me.

Sometimes my emotions are so overwhelming that I cannot cope any longer and I either have to take an overdose or cut myself to get some relief from my thoughts. Events like my care coordinator being off sick or friends not making contact can push me over the edge."

GENERAL CONSIDERATIONS IN THE ASSESSMENT AND DIAGNOSIS OF BPD

The reliability of the diagnostic assessment for personality disorder has been considerably improved by the introduction of standardized interview schedules. There are two structured interview techniques widely used for diagnosing personality disorder with high specificity and reliability: the Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD) and the International Classification of Diseases, 10th Revision International Personality Disorder Examination (IPDE-ICD-10) [67; 75; 77]. The SCID-5-PD aligns with DSM-5 criteria for personality disorders diagnosis, while the IPDE-ICD-10 aligns with diagnostic criteria using the ICD-10 and DSM-IV-TR [67; 75; 77], One issue, common to many of the instruments, is the excessive length of time required for administration, with the interview for either method taking approximately one to three hours, depending on experience and skill level of the interviewing clinician [75; 77]. A patient questionnaire is also available that can be completed in approximately 20 to 30 minutes, and will lessen the time of the interview. It should be noted that despite strengths such as reliability and direct correlation with DSM criteria, structured interviews and questionnaires may not fully capture the complexity and dynamics of patient mental health status. The diagnosis of BPD is most easily established by asking patients whether they believe the criteria for the disorder fits them and by listening to patients describe interpersonal interactions. Patients with BPD may be more likely to accept the assessment process by participating in the diagnosis. As discussed, patients and their families often find it helpful to be informed of the diagnosis and are relieved to learn that others share similar symptoms for which there are effective treatments [49].

When BPD is diagnosed through unstructured clinical assessment in primary care and generalist settings, several potential drawbacks come with this approach, including poor clinician agreement in personality disorder diagnoses, interference in personality assessment by the presence of acute mental or physical illness, and BPD symptom mimicry by active affective and anxiety disorders, psychosis, or substance use disorder. Before making a definitive diagnosis of BPD, the clinician should consider speaking to a close family member or friend to better understand the patient's personality traits [43; 67].

At initial presentation, current psychosocial functioning and safety to self and others should be the focus, with additional attention to comorbid psychiatric illness, personality functioning, coping strategies, strengths and vulnerabilities, and the needs of any dependent children. Some patients with BPD experience distress during the assessment process, and steps should be taken to avoid re-traumatizing the patient from unnecessary history-taking if it can be obtained elsewhere or at follow-up [42].

Discussion of the diagnosis provides the opportunity for the patient to understand his or her illness, request treatment, and become involved in his or her own recovery. Effective intervention may be less likely if the diagnosis is not made or recorded. Professionals should take care to maintain a balance between validating the person's problems and experiences by placing them within the BPD framework and promoting a view that change is possible through a shared effort [42].

DIFFERENTIAL DIAGNOSIS

Correct primary and comorbid psychiatric diagnosis is essential for appropriate psychotherapy and pharmacotherapy selection, and many patients with BPD are initially misdiagnosed with MDD or bipolar disorder and treated with antidepressants or mood stabilizers [62; 161; 198]. In some cases, these failed differentiations occur during patient crises and dictate immediate interventions and treatment planning. The diagnostic error may become apparent only when the patient stabilizes.

Importantly, some patients with prior experience of multiple evaluations may provide a history consistent with manic or hypomanic episodes because of the formulaic, standardized (or "leading") nature of diagnostic questions [181].

Major Depressive Disorder

The differential diagnostic issues surrounding BPD have changed over time. The original question of whether BPD was an atypical form of schizophrenia was dismissed with findings of the modest overlap in phenomenology and absence of familial or genetic connection. However, a novel case-control genome-wide study of BPD and comorbid conditions demonstrated a genetic overlap of BPD and other mental disorders, especially with bipolar disorder, and to a lesser degree with MDD and schizophrenia [85]. More research is needed to further explore the potential genetic overlap and association between the conditions.

The question of whether BPD represents an atypical form of affective disorder has proven more difficult to dispel, one reason being that most patients with BPD have lifetime MDD and present with complaints of severe dysphoria. Interpersonal and behavioral characteristics differentiate BPD from MDD, and unlike MDD, depressive episodes in BPD are characterized by emptiness, shame, and a long-standing negative self-image [18; 49].

Major Depressive Episode

Patients with a major depressive episode show significant retardation in thought processes and psychomotor behavior and severe depression of mood ranging from profound sadness to the total unavailability of any subjective sense of feeling, with a sense of total freezing of all emotional experience in the most severe cases. The content of thought processes are severely self-demeaning and self-accusatory, rather than focused on blaming and accusing others. Possibly present in severe MDD are guilt feelings, ranging from intense exaggeration of real or perceived deficits or faults to extreme and delusional self-devaluations and self-accusations. This combination of behavior slowing, lowering in mood, and self-devaluation spanning a period of weeks to months, with consistent daily symptom fluctuations of feeling worse in mornings with mood improving gradually every evening, characterizes a typical major depressive episode [181].

Patients with MDD or BPD commonly report feeling chronically hopeless and helpless, and while this indicates depressive despondency, further questioning of what the patient feels hopeless about and helpless over will evoke in patients with BPD a response with accusations and rage against others, with affect more angry than depressed. This dominance of rageful reactions while professing total self-devaluating depression is characteristic of personality disorders and should call into question the assumption of MDD. Patients with MDD withdraw from social contacts and may worsen when premature efforts are made to encourage them to socialize. Depressive reactions in personality disorders are usually less severe, can shift abruptly from one day to the next or from one hour to the next, and are positively or negatively influenced by the immediate social environment. These are characteristic of a personality disorder with a characterologic depression (termed dysthymic disorder), not a major depressive episode [181].

Evaluation of Baseline Personality Structure

Patients with BPD may experience severe dysthymic reactions with frequent symptoms of depression but absent the intensity, consistency, and duration found in MDD episodes. These patients typically have a history of chronic minor depressive episodes or dysthymic reactions over many years without significant periods of remission. While they report having been depressed all of their lives, these symptomatic features require differentiation from the characterologic depressive personality [181].

Around 30% of patients with MDD develop chronic, treatment-refractory depression that shows a remarkable lack of response to pharmacologic interventions [200]. Some may significantly improve for several weeks with electroconvulsive therapy and then revert to chronic depression. Correct diagnosis is especially important in these patients, because some with refractory depression may have a characterologic depression that would benefit from appropriate psychotherapy, especially given that co-occurring BPD significantly reduces treatment response to therapy for MDD [181].

Environmental Triggers Preceding a Depressive Episode

Typically, in characterologic depressive reactions, environmental conditions may trigger depressive reactions, and while often remarkably minor, the patient pays undue attention to the symbolic value of the triggering event. MDD usually lacks this disproportionate reaction to environment as a primary episode trigger [181].

The more severe the psychic and neurovegetative symptoms, the more likely there is a major depression. Conversely, the more predominant the personality disposition and environmental triggers, the more likely there is a dysthymic disorder (characterologic depression). Some patients present a "double depression," with an acute episode of MDD in the context of a chronic characterologic depression. These cases require, first, the treatment of the episode of MDD. Only after the resolution of this episode by pharmacologic and/or other treatments will a complete and accurate diagnosis, prognosis, and treatment plan for the characterologically based dysthymic disorder become feasible [181].

Self-Destructive Behaviors in MDD and BPD

Issues common to MDD and BPD include suicidal tendencies and parasuicidal behavior. Acute or chronic parasuicidal behavior, such as repeated cutting or burning (particularly under conditions of intense emotional agitation, temper tantrums, or acute frustrations), typically reflects BPD. This can seem to happen "out of the blue" and correspond to an outburst of temper without the background of symptoms of MDD [181].

In contrast, suicide attempts in patients with MDD require careful diagnostic assessment of the conditions under which suicidal behavior occurred. The types of suicidality generally found in patients with BPD can most often be treated with outpatient psychotherapy. However, suicide attempts in patients with MDD have severe prognostic implications, require immediate, systematic pharmacologic treatment, and may require hospitalization; patients unresponsive to other treatments may require electroconvulsive treatment [181].

Bipolar Disorder

Distinguishing BPD from bipolar disorder, and especially bipolar disorder II, can present a diagnostic dilemma due to the shared, overlapping symptoms. Both disorders have in common a substantial risk of suicide or suicide attempt, impulsivity, and inappropriate anger. However, symptoms that differentiate BPD include self-mutilation, self-injurious behavior without suicidal intent, and a frequent history of childhood abuse. Insecure attachments, reflected by intense abandonment fears, are hallmarks of BPD and uncommon in bipolar disorder. Patients with BPD have higher levels of impulsivity, hostility, and acute suicidal threats relative to those with bipolar disorder. Careful history taking usually elicits a differing time course of mood lability. Patients with BPD are extremely sensitive to rejection and do not have episodes of mania. Mood lability is often triggered by interpersonal sensitivity; mood lability in bipolar disorder tends to be autonomous and persistent [49; 161; 203].

The most frequent diagnostic error is confusing the chronic emotional instability and affect storms of patients with BPD with true hypomanic or manic behavior. This differentiation is easier with bipolar I, while the assumption of hypomanic behavior can form the basis for a bipolar II diagnosis. The diagnosis of bipolar disorder requires at least one episode of a manic (bipolar I) or hypomanic (bipolar II)

episode. Accurate assessment of such an episode is essential and is done by patiently ascertaining whether the patient has one or several periods of three to four days (or longer) of dominant and unusually euphoric, angry, or irritated mood, with a sense of heightened energy, affective dyscontrol, little need to sleep, hyperactivity, and unusual behavior that contrasts with the patient norm. The behavior can involve inappropriate sexual exposure or behavior, gross recklessness with money or other properties, socially inappropriate approaches to others, and possibly elevated sexual drive along with overall expansiveness of mood and behavior. A full manic episode often involves loss of reality testing, grossly inappropriate social behavior with patient unawareness of the behavioral deviation, and possibly hallucinations or delusions that can lead to intervention by others [181].

Determining whether BPD is present involves evaluating the nature of interpersonal and significant relationships. Patients with pure bipolar disorder lack severe pathology of object relations in periods of normal functioning; even patients with chronic bipolar disorder with manic and major depressive episodes maintain the capacity for depth and stability in their relationships and for assessing themselves and significant others appropriately [204]. This contrasts with the pronounced and pervasive emotional immaturity; the absence of affective stability and significant and mature relations with others; chronic instability in work or profession, love relations, and self-assessment; and identity diffusion with lack of an integrated concept of self that typifies BPD [181]. However, roughly 19% of patients with BPD are comorbid with bipolar disorder. These patients show severe, chronic affective instability together with clear hypomanic episodes [85; 168].

The dramatic expansion in concept and diagnosis of bipolar "spectrum" disorders has contributed to patients with BPD receiving this misdiagnosis. The evidence that mood stabilizers provide modest benefit to some patients with BPD will probably continue to encourage undue reliance on medication treatments [18].

Post-Traumatic Stress Disorder

The high prevalence of childhood trauma in patients with BPD first reported in the 1980s led to a movement in the 1990s that argued BPD was not an entity unto itself, but a misunderstood form of PTSD [205]. However, the literature indicates that only one-third of the BPD population has a history of severe and extended abuse and only 20% of individuals with a history of serious abuse go on to have serious psychopathology as adults [206].

Potential confusion between BPD and PTSD also arises from repeated findings that ongoing, chronic sexual, physical, or psychologic traumatization, particularly in early childhood, constitutes an important etiologic factor in the development of a severe personality disorder, particularly BPD [181]. Additionally, there is a syndrome of severe early trauma leading to sequelae including a BPD-like syndrome, called complex PTSD, whereby trauma is the central concern and requires therapeutic priority. These patients experience great difficulty in trust and cognitive processing, rendering BPD treatments ineffective. However, as discussed, trauma in most patients with BPD is superimposed on a genetically determined pre-existing sensitivity, and although these patients experience psychophysiologic difficulty in processing trauma and communicating about these adverse events, as adults they benefit from therapies for BPD. Thus, patients with BPD, unlike those with PTSD, respond to treatments that focus on feelings and not trauma and challenge them to take control of their lives [18].

The typical symptoms of PTSD arise within the first six months after a traumatic event and may last up to two or three years following the event. Symptoms include insomnia, irritability, angry outbursts, difficulty concentrating, hypervigilance, exaggerated startle response, and intensive reliving of the trauma in the form of nightmares, "flashbacks," and repeated memories of the trauma. The development of further symptoms many years after the actual, real, or assumed trauma, including somatization symptoms, dissociative symptoms, emotional lability, impulsivity, self-destructive behavior, and,

particularly, chronic interpersonal difficulties with manifestations of emotional immaturity, is indicative of a structured personality disorder, which may derive from trauma or a combination of personality disposition and traumatic experiences [181].

Treatment of PTSD requires a psychotherapeutic approach that facilitates the controlled reliving and working through of the traumatic experience in the context of a safe and secure therapeutic relationship. In contrast, when traumatic experiences are at the origin of a personality disorder, the conflicts triggered by the trauma usually take the form of an unconscious identification with the traumatic relationship—that is, an unconscious identification with both victim and perpetrator of the trauma. This differentiation is important from a therapeutic standpoint. In the transference-focused psychotherapy of patients with BPD, they have to be helped to acquire conscious awareness of this double identification and resolve it in the course of transference analysis. This represents a very different psychotherapeutic approach than that required for the treatment of PTSD [181; 207].

Narcissistic Personality Disorder

In contrast to patients with BPD who present different aspects of their internal world from one moment to the next, patients with narcissistic personality disorder mask the fragmentation and weakness of their identity under a brittle and fragile grandiose self that they present to the world and to themselves [181]. Patients with a severe narcissistic personality disorder may present symptoms strikingly similar to those of patients with BPD, including general impulsivity, chaos in relations with significant others, severe breakdown in their capacity for work and emotional intimacy, and parasuicidal and self-mutilating behavior. These patients are also prone to antisocial behavior, which requires the differential diagnosis among different types of narcissistic pathology with different levels of antisocial features [181].

Important differential features include the patient with narcissistic personality disorder's difficulty accepting any dependent relationship, their severe lack of investment in relations with significant others except in exploitative or parasitic relationships, and an aloofness that contrasts with the highly ambivalent yet clinging and dependent relationships of patients with BPD. Patients with narcissistic personality disorder can show extreme fluctuations between feelings of inferiority and failure and corresponding depressive reactions and an inordinate sense of superiority and grandiosity reflected by contemptuous and dismissive behavior toward others, including therapists. Patients with BPD may alternate their relationship between clinging dependency/idealization and angry rejection and dismissal, but do not show the chronically contemptuous and dismissive attitude of narcissistic patients. Resulting from these characteristics, patients with narcissistic personality disorder are usually isolated socially, even if they are externally part of a social network. They lose their friends and do not maintain relationships over an extended period of time, and their objective loneliness contrasts with the complicated, contradictory, yet enmeshed relationships of patients with BPD [181].

Antisocial behavior may be a complicating symptom of BPD, but it may be more central in lower levels of narcissistic personality disorder; it is always a negative prognostic factor. This is particularly true for antisocial personality disorder and for the syndrome of malignant narcissism, the most severe form of the narcissistic personality characterized by ego-syntonic aggression, paranoia, and antisocial traits. These are important differential diagnostic considerations when the clinical picture appears to be, at first sight, a BPD. They should be considered in the differential diagnosis of all patients within this spectrum of pathology who present with chronic antisocial behaviors [181].

GENDER DIFFERENCES

Although BPD does not differ by gender in prevalence, notable gender differences in BPD have been found in personality traits, comorbidity, and treatment utilization. In men with BPD, explosive temperaments, high levels of novelty seeking, substance abuse, and antisocial personality characteristics are more frequent. In women with BPD, current or past eating disorder, mood disorder, anxiety, and PTSD are more likely. Gender differences also appear in service utilization, with men with BPD more likely to have received substance abuse treatment, and greater use of pharmacotherapy and psychotherapy services more likely in women with BPD [208]. In clinical settings, 80% to 90% of patients with BPD are female, despite comprising roughly 50% of BPD in the general population. Some have speculated this clinical over-representation by women results from greater tendencies for inward expression of aggression (self-harm) that leads to medical intervention, while men with BPD are more likely to express aggression outwardly against others, leading to incarceration [171]. Although research shows comparable rates of intimate partner violence perpetration in men and women with BPD, the consequences of violence are gender-asymmetric [194; 209].

GENERAL TREATMENT CONSIDERATIONS

HISTORY OF THERAPY FOR BPD

As mentioned, the term "borderline" was introduced in 1938 to identify a patient subgroup with tendencies for regressing into a "borderline schizophrenic" mental state in unstructured situations. Over the next several decades, the primary psychiatric condition these patients were believed to "borderline" was schizophrenia. In 1967, psychoanalyst Otto Kernberg introduced the construct of "borderline personality organization" to describe patients with personality organization on the boundary between patients with psychotic personality organization (considered more severe) and patients with neurotic personality organization (considered milder) [210]. Borderline personality organization was defined as a broad form of psychopathology characterized by the primitive defenses of splitting and projective identification, identity diffusion, and lapses in reality testing [6].

During the 1970s, psychoanalytic psychotherapy was virtually the sole therapy approach addressed in the literature on BPD treatment. BPD was conceptualized in terms of specific structural deficits in the personality, requiring long-term, individual, intensive treatment aimed at restructuring the personality and eliminating BPD symptoms [18]. Psychoanalytic therapy for BPD was the focus of numerous conferences and many books. Well-known psychoanalytic psychotherapists gave compelling accounts of serious problems they encountered in treating patients with BPD, and the concepts of "countertransference hatred" and "negative therapeutic reactions" became recognized as uniquely applying to patients with BPD [211; 212; 213].

Kernberg stated that the adverse reactions to therapy that were highly commonplace in patients with BPD resulted from unconscious guilt (as an element of masochistic character structures), unconscious envy that underlies patient need to destroy what is received from their therapist, and unconscious identification with a primitive and sadistic object that underlies patient need to destroy the therapist as a good object [212]. Patient failure to improve with psychoanalytic therapy was solely attributed to pathologic motivations in the patient with BPD [6]. Improvements were rare exceptions rather than the rule. Although unknown at the time, in many patients with BPD traditional psychoanalytic therapy promoted symptom exacerbation from unintended toxic interaction between therapist approach and core BPD psychopathology [214].

Specifically, therapist neutrality encouraged patient projection and fueled abandonment fears, and therapist passivity promoted patient fears of disinterest and neglect. Therapist interpretations of negative motivations were experienced by patients as blaming and invalidating.

Despite the mismatch between therapy and patient pathology, important and enduring contributions to the borderline construct came from psychoanalytic observations. These include recognition of "stable instability" in patients with BPD, their desperate need to attach to others as transitional objects, an unstable and often distorted sense of self and others, reliance on splitting as a defense mechanism, and intense abandonment fears [6].

During the 1980s, biologic psychiatry began to replace psychoanalysis as the dominant therapeutic paradigm and approach. The validity of psychiatric condition criteria in the DSM-III was measured by investigations of discriminating descriptors, familiality, longitudinal course, treatment response, and biologic markers [215]. Research showed that BPD was internally consistent, showed a differing course from schizophrenia and major depression, had a familial basis, had a modest and inconsistent response to multiple medication classes, and was not a variation of depression [6].

Increasing attention became drawn to a possible relationship between BPD and PTSD, on the basis of presumed causality. Studies showed a high prevalence of childhood physical and sexual abuse in patients with BPD. During the same time period, feminists increasingly criticized DSM-III diagnoses, including BPD, for pathologizing women or covertly blaming victims [6]. Descriptions of BPD psychopathology were viewed by some feminist clinicians as byproducts of male anger, with male clinician diagnosis of female patients as BPD based on negative gender bias [216; 217]. Based on these theories, BPD was believed to disguise the underlying condition of PTSD [205; 218].

While beneficial treatment approaches remained absent, accumulating knowledge informed clinicians of what not to do. The adverse and harmful effects of therapist neutrality, passivity, poor maintenance of boundaries, and countertransference enactment were increasingly recognized. Empathy and support became widely appreciated as essential therapist approaches with these patients. Hospital-based clinicians understood that patients with BPD were not feigning symptoms to gain admission; the symptoms were genuine and usually remitted in response to the "holding" and supportive environment of the hospital. The shifting presentation of patients with BPD became coherent by understanding their expression varied by patient perception of feeling "held" (e.g., depressed, cooperative), rejected (e.g., angry, self-destructive), or alone (e.g., impulsive, brief psychotic experiences) [6; 219].

In the 1990s, psychiatry became dominated by the biologic paradigm. Biologic psychiatry challenged the diagnostic integrity of BPD on the basis that BPD lacked both a unifying neurobiologic organization and specific pharmacotherapy response. Resolution of this criticism began with the introduction of a pathophysiologic model based on impulsive/behavioral dyscontrol and affective/emotional instability, explaining the psychobiologic basis of common BPD symptoms [6; 220]. During the late 1980s, psychologist Marsha Linehan began to notice that many patients with BPD treated with cognitive-behavioral therapy (CBT) developed worsening of symptoms. Patients with BPD felt invalidated by their CBT therapist from the constant push for change in the absence of feeling that distress and suffering they experienced was acknowledged and appreciated by the therapist. Patients responded by shutting down or becoming agitated or suicidal. This clinical observation contributed to the development by Linehan of dialectical behavior therapy, a groundbreaking approach in general and the first therapy tailored to the needs of patients with BPD. Dialectical behavioral therapy also challenged the perception of therapeutic nihilism [18; 221].

Introduced in the early 1990s, dialectical behavioral therapy was the first of a second wave of therapies developed specifically for patients with BPD. Following dialectical behavioral therapy, schema-focused therapy, transference-focused psychotherapy, and mentalization-based therapy were introduced. Despite differing theoretical orientations, all possess common elements, such as a goal- and symptom-oriented approach, long-term patient involvement (usually at least 12 to 18 months), and high levels of structure with background and implementation described in treatment manuals [3]. Comparison of outcomes in patients with BPD shows that all of these approaches produce similar improvements in suicidality, intentional self-harm, and depression, and reduction in emergency room, hospitalization, and medication use [18].

A third wave of psychosocial approaches for BPD emerged with therapies intended for delivery by generalist mental health or primary care providers (e.g., internists, nurses) and developed to overcome implementation barriers encountered with specialist therapies. Their initial use as control group therapies in clinical trials led to unanticipated findings of efficacy. With refinement and empirical confirmation, they became introduced as primary therapies. Generalist approaches include structured clinical management, good clinical care, supportive psychotherapy, and general psychiatric management [18].

Thus, therapy approaches for patients with BPD have undergone an evolution that began with the introduction of second-wave therapies based on greater understanding of the underlying psychopathology, identification of previous ineffective approaches, and the tailoring of new therapies informed by neuroscience and clinical observation. The core effective elements of second-wave therapies, such as coping skills, problem solving, psychoeducation, validation, and an active therapist role, were incorporated into third-wave generalist therapies. Effective therapy also facilitates development of feelings of trust and closeness with the therapist (which may have been previously absent from the patient's life) and the expectation that learned skills will be applied to relationships outside of treatment to facilitate improvement. Patient progress is promoted with validation, as this helps develop patient recognition and acceptance of self as unique and worthy [3]. Two essential provider skills in working with patients with BPD are the ability to remain calm and the ability to remain accepting of the patient during extreme affects. Facilitating the learning of emotion-regulating skills requires sitting with the patient as he or she experiences these emotions and helping the patient identify and integrate them into the full range of emotional life. This ultimately leads to emotional integration and an ability to adaptively navigate intense emotions [171].

Psychotherapy is the current foundation of BPD treatment. Development of a secure attachment to the therapist is generally essential for patient improvement, but this does not come easily given the inherent intense needs and fears of attachment relationships. Patient symptoms can be difficult for professionals to manage, as they may assume the role of protective caretaker and can become angry and fearful when the patient suddenly reverts to dangerous or maladaptive behaviors. Patients with BPD may also abruptly terminate even highly skilled therapists. While this may be experienced as a failure by the provider, even brief therapy exposure is often later shown to have served a valuable purpose in helping the patient through a difficult period and in helping remove patient resistance to seeking and engaging subsequent therapists [3].

COMMON TREATMENT MYTHS

Provider reluctance to work with patients with BPD is often fueled by common myths, now dispelled. Common myths about treating patients with BPD, and their correction, include [214]:

Myth: Patients with BPD resist treatment.

Truth: *Most actively seek relief from emotional pain; treatment of their personality disorder requires psychoeducation by clinicians.*

Myth: Patients with BPD angrily attack their providers.

Truth: *Excessive anger and fearful wariness toward others, especially caregivers, are symptoms (instinctive transferences) of their disorder.*

Myth: Patients with BPD rarely improve.

Truth: *Roughly 10% significantly improve or remit within six months, 25% by one year, and 50% by two years. Once remitted, relapses are unusual.*

Myth: Patients with BPD improve only with extended, intensive treatment by experts.

Truth: *Such treatment is only required by a subgroup. Most do well with intermittent treatment by non-experts with some training. Intensive therapy can actually promote regression.*

Myth: Recurrent suicide risk invariably burdens providers and carries serious liability risks.

Truth: *Excessive burden or fears of litigation are often symptoms of inexperience and of poorly structured treatments.*

Myth: Recurrent crises require providers to be available at all times.

Truth: *Such a requirement is rare and means a different level or type of care is needed.*

THERAPY STRUCTURE AND MODALITY

Multimodal Treatment

Multimodal treatment, the use of two or more treatment modalities, has been affirmed by several lines of evidence as effective in the treatment of BPD. Different modalities of treatment can complement and augment the benefits of each other. The inclusion of multiple providers in a treatment team elevates the level of mutual support. The use of multiple modalities and providers allow the patient with BPD to express anger and disappointment without leaving treatment [18; 222].

Group Treatment

Group therapies are either led by professionals who select enrollment or by peers as self-help groups of people who assemble to discuss common issues. Both are effective in BPD.

Dialectical behavioral therapy skills training groups resemble classrooms in structure, with focus, direction, and homework between sessions provided by the group leader. Mentalization-based therapy groups assist in recognizing misattributions and how one's actions affect others. Patients with BPD may resist interpersonal or psychodynamic groups in order to avoid the required expression of strong feelings or personal disclosures; this is why such groups are beneficial. All professional-led groups can significantly enhance the treatment course by allowing patients with BPD to learn from persons with similar life experiences [3].

Patients with BPD may also join self-help groups addressing specific problems, such as Alcoholics Anonymous, Overeaters Anonymous, or Survivors of Incest. These and other self-help groups can provide a network of supportive peers that is beneficial as an adjunct to treatment, but they should not be used as a sole intervention [3].

Elements of group therapies designed for BPD include [214]:

- Self-assessment: Situational adaptations, problem solving
- Dialectical behavioral therapy skills training: Emotion regulation, impulse control, agency
- Mentalization-based therapy: Self-other awareness, psychologic-mindedness
- Interpersonal: Self-disclosure, assertion, anger management

Group therapy for BPD can help in developing the following skills [214]:

- Social skills (e.g., listening, sharing, competing)
- Self-disclosure (e.g., reduces shame, isolation)
- Assertiveness (e.g., self-respect, self-care)
- Self-other awareness (e.g., mentalizing)

LEVELS OF CARE FOR PATIENTS WITH BPD				
Level of Care	**Goal**	**Length**	**Clinical Tasks**	**Treatment Modalities**
Inpatient hospitalization	Making therapy possible	1 to 2 weeks	Safety/crisis stabilization, assessment, treatment planning	CM, medication, psychoeducation
Residential or partial hospital (10 to 20 hours/week)	Basic socialization	1 to 6 weeks	Daily living skills, social skills, impulse control, assist with community living, alliance building	CM, groups (DBT, self-assessment), psychoeducation
Intensive outpatient (4 to 10 hours/week)	Behavioral change	3 to 12 months	Further socialization, impulse control, alliance building	CM, groups (skills, interpersonal), individual psychotherapy
Outpatient (≤4 hours/week)	Interpersonal growth	As long as needed	Introspection, agency, skill generalization, intrapsychic change, alliance building	CM, groups (interpersonal, mentalization), individual psychotherapy
CM = case management, DBT = dialectical behavioral therapy.				
Source: [18]				*Table 2*

Levels of Care

Most providers working with patients with BPD will at some point need to determine the appropriate level of care for patients in crisis (***Table 2***). This decision should be made balancing the important principle of keeping treatment at the least restrictive level of care while maintaining patient safety. In other words, it is necessary to maintain sufficient structure to keep the patient safe but enough exposure to problems to maintain engagement in working toward treatment targets. Higher levels of care will provide safety and crises containment, but unnecessary placement in a higher level of care in response to dramatically expressed distress is an over-reaction that will reinforce the recurrence of crises and avoidance. Conversely, placement in an inappropriately low level of care will likely result in the patient spiraling into panic and desperation, an escalation of impulsive behaviors, and halting of treatment progress. The optimal level of care is based on clinical judgment and experience, because research guidance in this area is not of sufficient quality [3; 18].

Hospitalization

For patients with BPD, hospitalization is usually restricted to the management of crises (including, but not limited to, situations in which patient safety is precarious) and is short in duration. Hospitals provide a safe place where the patient has an opportunity to gain distance and perspective on a particular crisis and where professionals can assess the patient's psychologic and social problems and resources. It is not uncommon for medication changes to take place in the context of a hospital stay, so professionals can monitor the impact of new medications in a controlled environment [3].

However, hospital admission carries liabilities unique to patients with BPD. The American Psychiatric Association's recommendation of hospitalization whenever patients with BPD are suicidal has been criticized by experts for several reasons [86; 223]. Patients can internalize the invalidating message of their inability to get through a crisis without hospitalization. Progress in therapy can be rendered impossible by repeated hospitalization, as this becomes the learned coping strategy for distress. Hospitalization also prevents addressing the

interpersonal problem that triggered the crisis in the first place and can reinforce pathologic behaviors and make the patient worse [225].

SUICIDAL BEHAVIOR AND NON-SUICIDAL SELF-INJURY

Suicidal Behavior

The most dangerous features of BPD are self-harming behaviors and suicide risk. Suicidal ideation (i.e., ruminating and fantasizing about suicide) is pervasive in the BPD population [3]. Up to 10% of persons with BPD complete suicide, a rate 50 times greater than in the general population. More than 70% of those with BPD attempt suicide at least once, and patients with BPD attempt suicide an average of 3.3 times in their life [86; 226]. Suicide attempts in BPD tend to peak when patients are in their 20s and 30s, although suicidality can occur in any age group [8].

This led researchers to investigate whether a high lethality subtype of BPD could be identified. One study found that patients at highest risk for suicide had greater illness severity, vocational failures, and estrangement from family and friends. Low lethality subjects had better overall psychosocial functioning but more negativism, lifetime substance abuse, and histrionic and/or narcissistic personality disorder comorbidity. Suicide attempts of this latter group may reflect dramatic "communicative gestures," which show little change in medical lethality with repeat attempts [227]. Long-term longitudinal data have identified factors with greatest prediction of suicide attempt: diagnosis of MDD, substance use disorder, or PTSD; non-suicidal self-harm behavior; sexual assault as an adult; caretaker death from completed suicide; affective instability; and more severe dissociation. Prediction of suicide attempts in patients with BPD is complex and involves assessment of co-occurring psychiatric disorders, prominent BPD symptoms (i.e., self-harm, affective reactivity, and dissociation), adult adversity, and family history of completed suicide [228].

Studies of completed suicide in BPD found that duration of the "suicidal process," spanning from the first unequivocal suicidal communication by verbal threat to first suicide attempt and ultimately to death, may be as brief as 30 months or as long as 10 years. Suicide completion in BPD tends to occur relatively late in the course of the illness. In a 27-year follow-up study, the average age of those who completed suicide was 37 years. Younger patients with BPD tended to make frequent low-lethality attempts as communicative gestures, while older patients completed suicide after years of illness [86; 229; 230].

Approximately 50% of people with BPD experience an episode of major depression when they seek treatment, and about 80% have a lifetime prevalence of a major depressive episode. When depression coexists with an inability to tolerate intense emotion (as seen with BPD), the urge to act impulsively is exacerbated. It is imperative that providers carefully evaluate patient mood and appreciate the severity of the patient's unhappiness, but also recognize that antidepressant medications usually have only modest effects.

Patients with BPD often abuse alcohol or prescribed/illicit drugs for the short-term benefit of diminished social anxiety, distance from painful ruminations, or diminished intensity of negative emotions. While drug or alcohol use to achieve these effects can be viewed as self-medication, this use of substances is problematic because the disinhibiting effect from many substances elevates the risk of self-injury, suicide attempts, and other self-endangering behaviors [3].

As discussed, the propensity of patients with BPD to attempt suicide can often be intensely stressful for providers caring for them. As these patients are prone to feeling rejected and then reacting with rage, the manner in which they endanger their lives can be intended to inflict emotional agony on the provider(s) the patient perceives as uncaring or rejecting. As an example, a case was described in which a patient was discharged from an inpatient unit, walked to her car, and ingested

all of her prescribed medications. She then called the inpatient ward, told staff she had just overdosed on all her medications, but would not reveal where she or her car were. In this case, the patient was found by hospital personnel in time to intervene and save her life [5].

Non-Suicidal Self-Injury

Intentional self-harm behaviors (also termed parasuicidal acts or non-suicidal self-harm) are a common feature of BPD, occurring in 75% of those with the diagnosis and in an even higher percentage of those who have been hospitalized. Physical scarring and potentially disabling physical handicaps can result from this behavior [3].

Self-harm behavior assumes many forms, and patients with BPD often self-injure without suicidal intent. Most self-injury involves cutting, but it can also involve burning, hitting, head banging, or hair pulling. Some self-destructive behaviors are not perceived by patients as self-harming; among these are unprotected promiscuous sex, driving under the influence, and binging and purging [3]. Non-suicidal self-harm can also occur during hospitalization and may be expressed by treatment-sabotaging behavior. In one study, 63% of psychiatric inpatients who exhibited medically self-sabotaging behaviors had BPD. Behaviors and motivation included purposely avoiding needed medical treatment and/or prescribed medication(s) to hurt oneself; gravitating toward a dangerous situation hoping to be physically hurt; and damaging oneself on purpose to seek medical treatment [231].

Motivations for non-suicidal self-harm differ between individuals and within individuals across situations. As many as 40% of non-suicidal self-harm acts occur during dissociative periods, when numbness and emptiness dominate; as discussed, the most common motive for non-suicidal self-harm is to temporarily alleviate numbness or excruciating emotional pain. Suicide attempts are sometimes made when feeling alone and unloved or when life feels too painful to endure. Suicide attempts can occur with a vaguely conceived plan to be rescued, indicating a motive that relief from intolerable feelings of aloneness will occur when connection with others is established with "discovery" of the patient [3].

Clinicians can become constantly fearful of the suicide potential of patients with BPD, and managing this risk is of the utmost importance to maintain client safety. Intentional self-harm, impulsive behaviors with potentially self-destructive consequences (e.g., driving under the influence), and recurrent suicidal threats are probably the most difficult and stressful aspects of BPD for providers [8]. The patient with BPD may plead with his or her provider to keep communications or behaviors secret, but safety must be the priority. The patient, providers, and family cannot work together effectively without candor, and self-destructive threats or acts should never be kept secret for the benefit of all concerned. Family members/friends do not have the capacity to live with the specter of self-destructive behaviors in their lives, and patients will not progress in their treatment until these behaviors are eliminated [3].

Providers may find it difficult to distinguish between suicidal and non-suicidal self-harm, complicating selection of the safest and least restrictive intervention to implement. Some patients with non-suicidal self-harm may require hospitalization if the non-suicidal self-harm behavior is life-threatening. Providers may underestimate the severity of intent in chronically suicidal patients and forego the decision to hospitalize; alternatively, a decision to hospitalize may adversely impact a therapeutic relationship if the patient disagrees with the need for hospitalization [161].

Distinguishing suicidal from non-suicidal self-harm can be assisted by asking the patient about his or her intention. If suicidal intent is confirmed, patient safety is the priority. This may require hospitalization and usually necessitates contact with the patient's family members, often despite the patient's protests. In cases in which suicidal intent is denied, self-harming behaviors or threats may be

effectively managed by concerned attention from family/friends and the provider and by establishing a plan for crisis management. In clinical trials of various types of psychotherapy, emergency department visits have been used as needed in such situations, but alternative distress-coping strategies, such as telephone or e-mail contact or use of an Alcoholics Anonymous sponsor, should be encouraged. With repeated or escalating self-destructive behavior, a mental health professional with BPD experience should be involved in patient care [49].

Suicide Risk Assessment

Assessment of self-harm or suicidal behavior should be ongoing, with differing focus as dictated by patient status or clinical setting (***Table 3***). As noted, patients with BPD are often serious about suicide and have a high lifetime risk of completed suicide. Many overdoses later show ambivalent suicide intent, and episodes of non-suicidal self-harm are predictors of suicide risk and future suicide attempts. Suicide attempt or non-suicidal self-harm seldom reflects conscious attention-seeking behavior. However, some patients may use this as a motive in order to try to minimize the seriousness of their intent. Patients with BPD can also make suicide threats to lengthen their hospital stays. One study of 100 female inpatients with BPD found that subjects commonly expressed suicidal ideation or made suicidal gestures or threats around the time of discharge [232; 233].

Because patients with BPD are known as extensive users of healthcare services, with multiple presentations for crises, self-harm, or suicidal threats, healthcare providers can become desensitized and doubtful over the seriousness of patient intent and may feel manipulated [235]. Affective instability is the BPD feature with greatest association to attempted suicide. The affect, mood, and behavior of patients with BPD can rapidly change, as when shifting from very depressed and suicidal to angry and entitled, leaving the provider feeling frustrated and baffled [238].

Self-destructive behavior with suicidal intent may be planned or impulsive in persons with BPD. Some patients with BPD use threats of suicide to communicate distress to their provider or other people with whom they feel a close attachment. With a trusting therapeutic relationship established, patients with persistent suicidal thoughts may disclose risk factors that require intervention, such as stockpiling medication intended for overdose [42].

Many clinicians have found concepts from dynamic deconstructive psychotherapy useful in understanding affective instability and suicide risk in patients with BPD [239]. This model frames the emotionally labile reactions to the environment as a switch between different states of being, or pseudo-personalities, such that patients can alternately present as helpless and childlike (helpless victim state), angry and self-righteous (angry victim state), or depressed and suicidal (guilty perpetrator state). These states reflect different sets of polarized and poorly integrated attributions of self and others, and not strategies of manipulation [235].

In the angry victim state, patients see themselves as heroic victims, do not accept responsibility for failures, and blame others for their setbacks and problems. They feel justified in their actions and behaviors, including anger, manipulation, or violence. The angry victim state protects the patient from feelings of shame or humiliation and is triggered by interpersonal stimuli. In this state, patients are at low risk for suicide because they externalize the source of their problems [235].

Patients with BPD in the guilty perpetrator state are at significantly elevated risk for suicide [240]. Patients assume complete responsibility for every setback or misfortune, view their lives as an unending series of failures and bad decisions, and feel ugly, worthless, and evil. The guilty perpetrator state preserves an idealized image of others by devaluing oneself and is often triggered by perceived rejection, abandonment, separation anxiety, or any situation that prompts ambivalence toward major attachment figures [235].

ONGOING SUICIDE RISK ASSESSMENT IN PATIENTS WITH BPD
Perform Risk Assessment in these Patient Circumstances
First mental health services contact
Initiation of structured psychologic therapy during a crisis
Onset of additional psychiatric condition such as substance use disorder, depression, or psychosis
Change in psychosocial status
Transition between services or discharge from treatment
Predictors of Suicide Attempt
A clear plan for suicide
Intention of using a lethal method
Access to intended means and feasibility of executing plan
Absence of hoping for rescue during planned suicide attempt
Expressed hopelessness about their future
Delusions convincing patient they must die
Co-occurring depression or substance abuse problem
Lack of strong supportive social network
Assess Risk by Identifying These Changes in Patient
Pattern of suicidal behaviors
Changes in lethality of method or pattern of non-suicidal self-harm behaviors
Co-occurring mental illness or substance use
Psychosocial support resources
Mental state (especially depression, hopelessness, and suicidal thoughts)
Assess Immediate Risk Following Non-Suicidal Self-Harm or Suicidal Behavior
Assess triggers for suicidal ideation or behavior, such as abuse, separation, or loss
With triggers present, determine if time-limited or ongoing
Determine whether patient is blaming self or others for current problems. Self-blame strongly suggests higher suicide risk; blaming others suggests lower suicide risk.
After medication or drug overdose, assess what has changed between the time of overdose and the present moment that may lower suicide risk. If no changes, consider hospitalization.
Patient release home may be perceived as abandonment, but hospitalization can promote regression, and either decision may increase suicide risk. To help mitigate the dilemma, communicate this dilemma to patient and ask for his or her input. Patients with BPD are more likely to assume an adult, responsible role when they feel involved in decision making.
Approach for Patients at High Acute Risk of Suicide
Do not leave the patient alone. If required, invoke the powers of local mental health statutes, such as a 72-hour hold.
Prevent or reduce access to the means of suicide.
Consult senior staff.
Contact other providers involved in the patient's care and family, partner, or other close supports.
Find out what, or who, has helped in the past.
Clearly explain your actions.
Do not agree to secrecy over a suicide plan.
Make a management plan.
Consider psychiatric inpatient admission.

Table 3 continues on the next page.

ONGOING SUICIDE RISK ASSESSMENT IN PATIENTS WITH BPD (*Continued*)
Risk Assessment in the Inpatient Setting
The goals of inpatient admission are stabilization, preparation for outpatient treatment, and linkage or consultation with an outpatient therapist. To minimize regression, keep hospital stays brief. Inquire about the nature of relationships outside of therapy to assess the presence of issues related to abuse, rejection, loss, or separation. Use motivational interviewing to help the patient gain inner motivation for inpatient and outpatient treatment. Meet with family to help identify precipitating factors for suicide attempt or non-suicidal self-harm that led to admission. Undue attention to co-occurring affective, anxiety, or bipolar (formerly Axis I) disorders and pharmacotherapy can encourage a passive sick role and worsen regression. Avoid benzodiazepines for anxiety problems. Patients with BPD often like benzodiazepines and describe them as the only helpful medications, but these agents can worsen behavioral and affect dysregulation. Educate inpatient staff to watch for patient tendencies to split providers into opposing camps with behavior that provokes opposing reactions.
Approaches to Improve Patient Safety and Decrease Risk
Stress the need for patients to take responsibility for their own safety and commit to working toward recovery. This expectation can be delivered early in treatment as one of the written treatment goals and expectations. Safety improves as dissociation and core BPD symptoms improve, and the most effective technique to facilitate this improvement is to have the patient articulate recent provocative destabilizing interpersonal experiences, create sequential narratives of these experiences, and label the associated emotions. Structured, manual-based therapies are more effective and stabilizing than reliance on clinical intuition and judgment while using an eclectic, unstructured approach.
General Suicide Risk Management Concepts
Always take suicide threats by patients with BPD seriously, but managing risk differs with chronic suicidality and acute suicidality. In chronically suicidal patients, active suicide prevention efforts (e.g., hospital admission, close observation) can be unhelpful and may escalate risk; tolerating long-term suicide risk is often necessary for patient and provider. Chronic suicidality improves as patients become less symptomatic and quality of life improves. However, in patients with chronic, high suicide risk, emergence of new symptoms, behaviors, or psychiatric comorbidity may indicate the immediate risk of suicide has increased. Short-term inpatient psychiatric admission may be appropriate to manage acute risk, while plan modification to ensure immediate safety while continuing BPD treatment and managing comorbidity can manage long-term risk. When managing non-suicidal self-harm-prone patients, frequently assess changes in risk pattern, such as frequency, type, and level of risk. Patients with persistent low-lethality non-suicidal self-harm may be at low present risk of suicide. Escalation in non-suicidal self-harm lethality potential can indicate high long-term risk. Structured outpatient therapy is more suitable than inpatient admission in these cases, as greater patient benefit is gained from forming therapeutic relationships and stable social supports. Suicide may follow patient disengagement from therapy and giving up on trying to receive help, although unintended fatality from self-harm occasionally does occur.
Source: [42; 86; 234; 235; 236; 237] *Table 3*

Circumstances that intensify attachment wishes and fears can induce or amplify states of being and rapidity of switching. These can include abusive relationships, prolonged hospital stays, or poor patient-therapist boundaries, such as physical touch, multiple contacts per week, or extended sessions. These conditions can result in the patient with BPD regressing and becoming moody and childlike, grossly over-reacting to slight provocation, and rapidly shifting between states of being [235].

Most importantly, clinicians should know their ambivalence and conflict over whether or not they should take the suicidal behavior and threats of a patient with BPD seriously actually mirrors the same internal conflict within the patient. This inner conflict involves whether or not their illness is legitimate or whether they are fully to blame for all their problems, attention-seeking, or simply needing to "clean up their act." As the patient takes one side of the inner conflict or changes from one side to another, he or she can rapidly switch states as blame is switched from self to others. Determining immediate risk of self-harm is informed by assessing the patient for current state of being, recent stressors, alcohol misuse, and support system integrity [235].

EMOTIONAL HYPER-REACTIVITY

Patients with BPD are likely to be consistently hyper-reactive on an emotional level, and providers should understand that this reflects patient psychopathology and not necessarily the environment or clinician behavior. This pathologic feature requires therapeutic modification in order for patients to effectively function in interpersonal relationships. It is effectively addressed by didactic instruction, skills development, and cognitive-behavioral approaches—elements common to several psychosocial therapy approaches [241].

Emotional hyper-reactivity is most likely to manifest when limits are set or when a patient (mis) perceives an attachment relationship is about to dissolve. In both contexts, patients are likely to over-react in an emotionally volatile, angry, and possibly regressive manner. In the primary care setting, the patient with BPD may encounter the experience of refusal, or limit setting, in numerous situations, for example, clinician refusal to order a particular laboratory study, prescribe a requested medication or drug class, or make a particular referral. Refusal dynamics can also emerge with patient requests for unnecessary time-off-work excuses, automobile handicap flags, or disability status [45].

Another manifestation of emotional hyper-reactivity involves intense reactivity to touch and physical examination in some patients with BPD, possibly from strong association with negative childhood experiences. The emotional hyper-reactivity should be anticipated, the patient prepared by the impartial presentation of the medical stimuli, and the clinician should avoid personalizing patient response [45].

PSYCHOSOCIAL THERAPIES

THERAPY SELECTION

Therapy selection should best match the characteristics of the patient with BPD [161]. Psychodynamic therapy is suggested for patients with:

- A chronic sense of emptiness and underestimation of self-worth
- Loss or prolonged separation in childhood
- Conflicts in past relationships
- Capacity for insight
- Ability to modulate regression
- Access to dreams and fantasy
- Little need for direction and guidance
- A stable environment

Cognitive therapy is the best option for patients with:

- Obvious distorted beliefs about self, world, and future
- Pragmatic (logical) thinking
- Real inadequacies (including poor responses to other psychotherapies)
- Moderate to high need for direction and guidance
- Responsiveness to behavioral training and self-help (high level of self-control)

Interpersonal therapy is best suited for patients with:

- Recent, focused conflict with spouse or significant other
- Social or communication problems
- Recent role transition or life change
- Abnormal grief reaction
- Modest to moderate need for direction and guidance
- Responsiveness to environmental manipulation

Supportive therapy approaches should be selected if the following patient characteristics are present:

- Failure to progress in other therapy modalities
- Suicidality
- Cognitive impairment and illogical thought
- Acute or chronic medical illness
- Presence of somatization or denial of illness
- Necessity of high levels of guidance
- Responsive to behavioral methods

Psychosocial interventions in BPD are grouped according to the required level of provider education and training. These include specialist therapies delivered by psychiatrists, psychologists, counselors/therapists, or social workers with extensive specialized training; generalist therapies delivered by psychiatrists, psychologists, or other mental health professionals with minimal additional training; and interventions by primary care providers that require minimal additional training.

When planning structured psychologic therapies for BPD, the National Health and Medical Research Council recommends therapists adapt the frequency of sessions to the person's needs and circumstances and generally consider providing at least one session per week.

(https://bpdfoundation.org.au/images/mh25_borderline_personality_guideline.pdf. Last accessed March 24, 2020.)

Strength of Recommendation/Level of Evidence: Consensus-based recommendation (Recommendation formulated by the guideline development committee/group, using a consensus-reaching process, in the absence of high-quality evidence.)

SPECIALIST PSYCHOSOCIAL THERAPIES

Psychosocial therapies for BPD delivered by mental health professionals with advanced training fall into the two broad categories: CBTs and psychodynamic psychotherapies.

Cognitive-Behavioral-Based Therapies

Dialectical Behavioral Therapy

Dialectical behavioral therapy is based on the theoretical principle that maladaptive behaviors, including self-injury, are attempts to manage intense overwhelming affect of biosocial origin. Dialectical behavioral therapy incorporates the two key elements of a behavioral, problem-solving approach blended with acceptance-based strategies, with an emphasis on dialectical processes. This therapy approach emphasizes balancing behavioral change, problem-solving, and emotional regulation with validation, mindfulness, and acceptance of patients. Therapists follow a detailed procedural manual [22; 87]. The term "dialectical" refers to the philosophical principle of opposite truths, such that constructs can be opposing yet true at the same time. A core dialectic in this therapy is

accepting patients where they are in the moment and working to help them change [225]. The five components of dialectical behavioral therapy are [87; 225]:

- Capability enhancement (skills training)
- Motivational enhancement (individual behavioral treatment plans)
- Generalization (access to therapist outside clinical setting, homework, inclusion of family in treatment)
- Structuring the environment (emphasis on reinforcing adaptive behaviors)
- Capability and motivational enhancement of therapists (therapist team consultation group)

Dialectical behavioral therapy involves weekly individual sessions, skills-training group sessions, phone consultation available at all times with a therapist, and team consultation meetings. Therapeutic targets are ranked in hierarchical order, with life-threatening behaviors addressed first, followed by therapy-interfering behaviors, and then behaviors that interfere with quality of life [225].

Specific strategies used by dialectical behavioral therapists include alternating between acceptance and change strategies, adding intuitive knowing to emotional experience and logical analysis, playing the role of devil's advocate, exploring novel and alternate points of view, turning problems into assets, and introducing and exploring a middle path [243].

EVIDENCE-BASED PRACTICE RECOMMENDATION

The Department of Veterans Affairs recommends that dialectical behavioral therapy may be considered for patients with BPD and recent self-directed violence. (https://www.healthquality.va.gov/guidelines/MH/srb. Last accessed March 24, 2020.)

Level of Evidence: I (At least one properly done randomized controlled trial)

Cognitive and Cognitive-Behavioral Therapy

CBT is a structured psychologic treatment that helps the patient identify maladaptive beliefs and thoughts, connect these beliefs to their feelings and behaviors, and replace maladaptive beliefs and thoughts with adaptive ones. CBT was originally developed for treating depression and anxiety disorders. In patients with depression or anxiety, CBT is generally focused on present situations. In patients with BPD, CBT is modified to address the contribution of previous experiences in the development of core beliefs (termed "schemas"), the structure of therapy and problems that can disrupt the therapeutic relationship, shifting problems and goals, losing focus on therapy objectives, losing structure, and homework non-compliance [43; 87].

CBT for BPD is usually delivered in weekly 30- to 90-minute sessions over 9 to 36 months. Patients are given homework between sessions, and some clinics or practices have therapists available by telephone outside of clinic visits [43].

Systems Training for Emotional Predictability and Problem Solving

Systems Training for Emotional Predictability and Problem Solving (STEPPS) is a manualized, CBT-based skills development package intended to supplement primary BPD therapy. The components of STEPPS include BPD psychoeducation, emotion management skills training, and behavior management skills training. It includes a two-hour session for family members and significant others, including members of the treatment team, to introduce them to the concepts and skills enabling them to provide support and reinforcement to the patient. STEPPS is delivered in 20 two-hour weekly group meetings led by two co-therapists, with each session addressing a specific emotion or behavior management skill. Throughout the 20 weeks, patients are instructed to monitor their thoughts, feelings, and behaviors to facilitate the recognition and monitoring of changes in the intensity and frequency of emotional episodes [43; 232; 245].

Cognitive Analytic Therapy

Cognitive analytic therapy is an integrative and relational approach combining CBT with attention to the therapeutic relationship as the vehicle of change. The aim of cognitive analytic therapy is to help the patient understand how problematic, harsh, and punitive relationship patterns with self and others have been learned and continue to be re-enacted. Cognitive analytic therapy uses the methods of narrative and diagrammatic reformulation. These are used to describe the recurrent patterns of relating with others by helping patients understand their experience of "switching" between different states of mind in response to unmanageable feelings or unmet needs. BPD is viewed as representing a form of severe and pervasive damage to self, largely due to complex developmental trauma and deprivation. This leads to a tendency to dissociate into different "self-states," with resultant highly distressing impairment of self-reflective capacity and sense of identity, impaired executive function, and disturbed interpersonal relations. Therapy aims to provide a reparative relational experience and to provide patients with the motivation, skills, and opportunities to learn new patterns of relating to oneself and others [43; 242].

Psychodynamic-Based Therapies

Dynamic Deconstructive Psychotherapy

Dynamic deconstructive psychotherapy is a 12- to 18-month, manual-driven treatment for adults with BPD that combines elements of translational neuroscience, object relations theory, and deconstruction philosophy to help patients heal from a negative self-image and maladaptive processing of emotionally charged experiences. Neuroscience research suggests the complex behavior problems of patients with BPD result from deactivation of brain regions responsible for verbalizing emotional experiences, attaining a sense of self, and differentiating self from other; instead, the activated brain regions contribute to hyperarousal and impulsivity [247].

Dynamic deconstructive psychotherapy helps patients connect with their experiences in order to develop authentic and fulfilling connections with others. During the weekly, one-hour sessions, patients discuss recent interpersonal experiences, label their emotions, and reflect on their experiences in increasingly complex and realistic ways to begin the process of self-acceptance. An important clinician skill is learning to recognize, understand, and use the intense emotional reactions elicited by patients with BPD to foster patient recovery, avoid burnout, and provide novel experiences in the client-therapist relationship that support individuation and challenge basic assumptions of themselves and others harbored by the patient [247].

Mentalization-Based Therapy

Mentalization-based therapy is a psychodynamic approach based on attachment and cognitive theory. Mentalization refers to the ability to accurately imagine the mental states of others, a normal developmental milestone attained by stable early attachment relationships. Patients with BPD are thought to have deficits in mentalization resulting from problematic early attachments. The core features of BPD are thought to reflect this failure to develop mentalizing ability and the resultant profound disorganization of self-structure [225; 248].

The objective of mentalization-based therapy is to increase patient curiosity about their feelings and thoughts and to develop skills in identifying feelings and thoughts in themselves and others in the context of attachment relationships. By developing mentalizing skills and the capacity to understand their mental states and those of others in attachment contexts, patients are able to address problems with affect, impulse control, and interpersonal functioning, and reduce triggers for self-harm and suicidal behavior. Mentalization-based therapy is delivered in weekly individual and group therapy sessions [88; 249].

Schema-Focused Psychotherapy

Schemas are pervasive patterns of thinking, feeling, and behaving, and schema-focused psychotherapy is based on the theory that patients with BPD acquire four dysfunctional schemas in early life that maintain their psychopathology and impairment: the detached protector, the punitive parent, the abused/abandoned child, and the angry/impulsive child. These schemas are maintained by inflexible processes that prevent new learning, termed schema maintenance, schema avoidance, and schema compensation. Schema-focused psychotherapy strives to facilitate affective engagement and re-learning, and this may involve elements of re-parenting. Approaches include a range of behavioral, cognitive, and experiential methods that focus on the therapeutic relationship, daily life outside of therapy, and past experiences (including trauma, if relevant). Patients explore the role these core beliefs played in helping them adapt to early environmental adversity and to question whether they remain appropriate for helping adaptation to their current circumstances. Recovery in schema-focused psychotherapy occurs when dysfunctional schemas no longer control the patient's life. Schema-focused psychotherapy is usually provided in biweekly 50-minute sessions [43; 88; 248].

Transference-Focused Psychotherapy

Transference-focused psychotherapy is based on Kernberg's conceptualization of the core problem of BPD—excessive early aggression led the young child to split positive and negative images of him/herself and his or her mother [250]. The pre-borderline child is unable to merge positive and negative images and corresponding affects to attain a more realistic and ambivalent view of self and others. The primary goal of transference-focused psychotherapy is to reduce symptomatology and self-destructive behavior by modifying representations of self and others as enacted in the transference relationship. Clarifications, confrontations, and transference interpretations are the primary techniques of this twice-weekly psychotherapy [88; 248].

Psychodynamic Interpersonal Therapy

Interpersonal therapy is a structured, time-limited supportive therapy first developed for patients with major depression. With this approach, the clinician addresses interpersonal sensitivity, role transitions, interpersonal disputes, or losses and links these to changes in mood [43]. Psychodynamic interpersonal therapy is a manualized therapy for BPD in which the clinician establishes a therapeutic atmosphere that facilitates "connectedness" between the patient and therapist to develop a shared language for feelings. This serves to amplify expression of the patient's personal and inner world during conversation, increasing the opportunity for clinicians to identify traumatic memories that surface and help patients with their integration into the system of self. These disjunctions appear as negative affect, linear thinking, orientation toward events and the outer world, changes in the self-state, and the development of transference [43].

Psychodynamic/Psychoanalytic Psychotherapy

Psychodynamic/psychoanalytic psychotherapy emphasizes the role of unconscious conflict within the context of internal representations of self and others. Relationship problems are replicated in the therapeutic relationship through transference and are interpreted by the clinician. Psychodynamic/psychoanalytic psychotherapy diverges from the traditional approach of psychoanalysis that encouraged therapist neutrality to form a "blank screen" onto which the patient projected inner conflicts and wishes. Instead, with psychodynamic/psychoanalytic psychotherapy, the therapist provides greater structure and is more active with patients [43].

Comparative Efficacy of Specialist Psychosocial Therapies

Several therapies developed for patients with BPD have been evaluated in clinical trials, and the efficacy of these approaches was compared in a review of literature published before 2011 [251]. This analysis found that dialectical behavioral therapy was superior to treatment as usual on outcomes of anger, parasuicidality, and overall mental health, and comparable on treatment retention [251; 252].

Compared with the outcomes of patients randomized to control therapy, patients receiving several therapies developed for BPD showed significantly greater improvements in core BPD pathology and associated psychopathology. These included dialectical behavioral therapy; dialectical behavioral therapy adapted for patients with BPD plus PTSD; mentalization-based treatment in a partial hospitalization or outpatient setting; transference-focused psychotherapy; and interpersonal psychotherapy modified for BPD. Standard interpersonal psychotherapy was effective in reducing associated depression only. CBT and deconstructive dynamic psychotherapy did not show significant benefit on BPD core pathology or associated psychopathology outcomes. Dialectical behavioral therapy was more effective in improving core and associated psychopathology than patient-centered therapy, and schema-focused therapy was more effective than transference-focused psychotherapy on measures of BPD severity and treatment retention [251; 252]. A 2014 study suggests dialectical behavioral therapy uniquely benefits patients with BPD by improving expressions of anger and experiential avoidance and that improvement in overall negative emotion comes from non-specific factors common to specialist and generalist therapies [253].

While dialectical behavioral therapy, mentalization-based therapy, transference-focused psychotherapy, and interpersonal psychotherapy modified for BPD have all shown substantial benefit, no single therapy approach has emerged with greatest efficacy, which suggests that clinicians should offer the therapy modality that best matches their training, theoretical orientation, and preferences [42; 252]. This point is underscored by repeated findings that treatment outcomes in patients with BPD is particularly influenced by the individual therapist [42].

Importantly, the highly structured comparator treatments used in many of the randomized controlled trials have also been found effective. This evidence of comparable benefit from tailored specialized therapies and structured control group therapies is thought to reflect core elements that are common to both groups of psychologic treatment [254].

GENERALIST PSYCHOSOCIAL THERAPIES

While many of the specialist therapies for patients with BPD have clear benefit, these therapy modalities are intensive, lengthy in duration, require special practitioner training, and are expensive to provide. The level of skill and training necessary to ensure their effective delivery exceeds those of most private practitioners, mental health clinics, and even major medical centers to provide. These practical barriers have prompted the search for less intensive and more cost-effective forms of psychosocial treatment for BPD [248].

This need initiated the development of several generalist approaches designed for delivery by generalist mental health clinicians and implementation in generalist (rather than specialist) clinical settings. These treatment approaches are based on skills and knowledge that, for the most part, are already present in skilled general mental health clinicians. This allows providers to make relatively modest adaptations to therapy approaches they already use instead of needing to learn new techniques. As such, they require only modest time involvement in training and supervision to be effective [255].

Four generalist treatment modalities have been developed for patients with BPD. They are structured clinical management, good clinical care, supportive psychotherapy, and general psychiatric management. These approaches are highly feasible and have been empirically validated in real-world clinical settings [255].

SHARED FEATURES OF EFFECTIVE SPECIALIST AND GENERALIST BPD THERAPIES	
Feature	**Description**
A primary clinician	Designate one clinician to discuss diagnosis, assess progress, monitor safety, and oversee communications. Approach is active and at times directive.
Structured therapy model	Manualized therapy assists clinician in structuring, and structure is maintained by active clinician involvement.
Mutually agreed treatment plan structure	Clearly define therapy goals, clinician role, personal limits, and crisis management parameters.
Connect feelings and behaviors to events	Explore with patient problematic behaviors in the context of precipitating events, thoughts, and feelings.
Support	Validate patient distress and transmit hope and confidence of patient ability to change.
Actively involve the patient	Involvement helps the patient know that progress hinges on his/her active efforts to take control over their feelings, behaviors, and future.
Interactive clinician	Approach is active and interrupts silence and digression. Focus is on the here-and-now and responds to safety issues with concern but resists cautiously. This helps patient explore thoughts, feelings, and behaviors.
Clinician self-monitoring of countertransference, consultation with colleagues	Awareness of idealization or devaluation as the patient's interpersonal style, that reaction with urge to rescue or punish the patient is natural and predictable (countertransference) but disruptive to treatment, and that consultation can help manage these emotional reactions.
Source: [18; 255]	*Table 4*

Shared Features of Effective Specialist and Generalist BPD Therapies

The findings of comparable positive patient outcomes between specialized therapies and generalist therapies in clinical trials are believed to reflect common shared features (***Table 4***), which makes intuitive sense as both groups of therapy were tailored for the clinical needs of patients with BPD. While these common factors are elements of all evidence-based treatments effective for patients with BPD, generalist treatments are not meant to replace specialist treatments. At this point, the respective roles of specialist and generalist approaches are not clear, but further research may find the majority of BPD can be successfully treated with generalist treatment, with a subgroup needing specialist treatment [255]. Implicit with generalist treatment is recognition that patients not showing improvement require referral to mental health providers with specialized training.

Generalist Therapy Approaches

Structured Clinical Management

Structured clinical management is based on a counseling model that resembles supportive therapy, with the addition of case management, advocacy support, problem solving, a crisis plan, medication review, and assertive follow-up if the patient begins missing appointments. Medication is used as an adjunct, when clinically indicated. Structured clinical management is provided by non-specialist clinicians, usually as weekly individual and group therapy sessions [255].

A comparison of mentalization-based therapy with structured clinical management found substantial improvements with both treatments across a range of clinical outcome measures. Mentalization-based therapy achieved steeper and somewhat larger effect sizes after 18 months, but structured clinical management was equally effective over the initial 6 months and patients receiving structured clinical management showed faster reduction in self-harming behaviors [249].

Good Clinical Care

Good clinical care is a CBT-informed approach that incorporates a problem-solving paradigm as the core treatment intervention and stresses the importance of effective organizational structure. Psychologists trained in CBT provide therapy and case management. Patients are discussed in a weekly team meeting, with each team including a psychiatrist. A therapy session is typically offered once per week, which is flexible if patients need an additional contact, up to a maximum of 24 therapy sessions over six months. Case management is also flexibly provided, with clinical trials averaging around three management sessions for every therapy session. This underscores the point that effective intervention involves more than formal psychotherapy [187; 255]. A study that randomized adolescents with BPD or BPD traits to cognitive analytic therapy or good clinical care found that subjects in both groups showed significant improvements across a range of clinical outcome measures, with little difference in benefit between therapies [187; 256].

Supportive Psychotherapy

Supportive psychotherapy places an emphasis on establishing and maintaining a comfortable, relaxed therapy relationship, with minimal use of interpretation. The focus of supportive psychotherapy is to offer emotional support and advice on current problems encountered by the patient. Transference is followed and managed by the therapist, while interpretation is intentionally avoided. The mechanism of change with supportive psychotherapy is thought to involve the client identifying with the consistent attitudes of benevolence, interest, kindness, and nonjudgmental acceptance by the therapist. Sessions are provided weekly and supplemented with additional sessions as needed [255; 257].

The outcomes of patients with BPD receiving dialectical behavioral therapy, transference-focused psychotherapy, or supportive psychotherapy were compared after one year of treatment. Patients in all three groups demonstrated significant improvements across a range of clinical measures, with outcomes from all three treatments reported as generally equivalent [87; 177]. Patients assigned to supportive psychotherapy received one scheduled session per week plus additional sessions if needed, while patients in the two specialist treatments received two scheduled sessions per week, a clear cost-savings advantage [89; 255].

General Psychiatric Management

General psychiatric management is a psychodynamically informed approach that includes case management and symptom-targeted adjunctive medication. The psychodynamic approach stresses that disturbed early attachments are a primary deficit in BPD [222]. General psychiatric management was developed to provide professionals involved in the care of patients with BPD with the basic knowledge necessary to manage this patient group without the need for intensive advanced training. General psychiatric management includes four basic elements [144; 171]:

- Psychoeducation
- Persistent focus on patient's life outside of therapy, to connect long-term goal attainment with the need for learning emotional and self-harming control
- Therapist acknowledgement and use of their dual role as a professional and a person:
  - Professional role: Sharing knowledge, providing concerned but unemotional responses to patient bursts of emotion, striving to understand patient's recurring concerns of therapist motives, feelings, and trustworthiness
  - Person role: Therapist explanation of what they meant, disclosure of feelings such as confusion or apprehension, stating to the patient the wish to help
- A highly interactive and directly engaging provider approach

SEQUENCE OF EXPECTED CHANGE WITH GENERAL PSYCHIATRIC MANAGEMENT OF BPD			
Target Area	**Expected Changes**	**Time**	**Relevant Interventions**
Distress and dysphoria	Reduce anxiety and depression	1 to 6 weeks	Support, situational changes Increase self-awareness
Behavior	Reduce self-harm, rages, and promiscuity	2 to 6 months	Increase awareness of self and interpersonal triggers Increase problem-solving strategies
Interpersonal	Reduce devaluation Increase assertiveness and positive dependency	6 to 12 months	Increase mentalization Increase stability of attachment
Social function	Improvements in school, work, and domestic responsibilities	6 to 18 months	Reduce fear, failure, and abandonment Coaching
Source: [214; 222]			*Table 5*

- General psychiatric management is generally delivered over four phases of therapy:
  - Phase 1, Building a Contractual Alliance: Patient engagement and agreeing on goals and roles (1 to 3 months)
  - Phase 2, Building a Relational Alliance: Liking and trusting therapist intentions (1 to 12 months)
  - Phase 3, Positive Dependency: Explicit encouragement, with the patient attaining a level of comfort with connection to the therapist (6 to 18 months to 2 to 5 years)
  - Phase 4, Becoming Non-Borderline (Recovery): Patient applies the skills learned in therapy to all other endeavors (2 to 10 years)

During the initial phases, the targets of therapeutic focus and improvement or resolution of symptoms follow a sequential pattern (***Table 5***).

One study randomized subjects with severe BPD to one year of dialectical behavioral therapy or general psychiatric management [84]. The study found that general psychiatric management and dialectical behavioral therapy both led to significant and comparable improvements across a broad of clinical outcome measures, which persisted at two-year follow-up. With general psychiatric management, 66% of providers were psychiatrists [255].

PSYCHOSOCIAL INTERVENTIONS FOR PRIMARY CARE PROVIDERS

Treating primary care patients with BPD requires careful management to ensure quality, patient-centered medical care. Primary care providers are often left feeling frustrated, angry, or helpless when caring for patients with provocative, demanding, dependent, aggressive, angry, and manipulative behaviors. Several approaches have been tailored for primary care providers and designed for use during a 15-minute clinical visit; these modalities can help providers feel more effective and confident with patients with BPD. Primary care approaches are user-friendly, non-confrontational, practical for single-visit or longitudinal delivery, effective, and can help the clinician avoid being drawn into the patient's pathologic personality traits, which often leads to conflict [244; 246].

Because BPD is a chronic condition that, in most cases, requires long-term contact and management involving primary care providers (as with many other chronic diseases), it is important for these providers to understand the characteristics of the disorder and its implications on the provider-patient relationship. As effective treatments for BPD continue to emerge, primary care providers can direct treatment and improve long-term patient care [244].

MOTIVATIONAL INTERVIEWING IN A HYPOTHETICAL HELP-REJECTING PATIENT WITH BPD	
Goal	**Example Script**
Ask for permission to discuss the problem: Increase patient awareness of a problem he/she is avoiding or denying.	I'm concerned about our working relationship because it seems you often dismiss my medical advice, but continue to ask for recommendations. Would it be okay for us to discuss this now?
Elicit talk about change: Generate thoughts of the drawbacks in the status quo, the benefits of change, specific change possibilities, and taking the first step toward change.	What do you think will happen if the pattern of dismissing medical advice does not change? What could work for you if you decided to change? What might be some good things about changing? What would you be willing to try as a first step?
Importance check: Instruct the patient to rate his/her readiness and motivation to embrace behavior change. Reinforce talk about change.	On a scale of 1 to 10, with 1 being the lowest and 10 being the highest, how important is it for you to change the pattern we have discussed and try a new approach?
Ability check: Assess patient confidence in his/her ability to change and to overcome barriers to change.	On a scale of 1 to 10, how confident are you that you will succeed in making a behavior change? What do you see as barriers to becoming more self-confident and to independently make informed choices? How might you overcome these obstacles?
Statement to terminate the motivational interview: Summarize the main discussion points, patient commitment to change, and the follow-up plan. Re-state what the patient has agreed to and what the patient has not agreed to.	If I may summarize our discussion, the problem in our working relationship appears to be the pattern of dismissing medical advice. You are motivated to make changes with my encouragement, and you are specifically going to work on <behavior>. Let's re-visit this in a couple of weeks to check on your progress, to talk about how you are coping with barriers to change, and to modify the solution a bit, if needed.
Source: [230; 244]	*Table 6*

Two approaches for the primary care setting are motivational interviewing and problem solving. These are not treatment approaches per se, but instead are concrete, problem-focused tools that allow the clinician to improve the outcomes of patients with BPD while navigating some of the problematic behaviors and attitudes driven by the personality traits of BPD. Both were developed to address the very legitimate provider concerns over emotional endurance and job satisfaction when managing patients with BPD. When treating patients with BPD, clinicians should also consider a collaboratively developed crisis and safety plan and should use an overall approach of active listening, mindfulness, and strengthening patients' connections to their most important values [242; 244; 246].

Motivational Interviewing

One of the biggest challenges in the successful treatment of patients with BPD and other personality disorders is the patient's tendency for irregular treatment attendance, disengagement and premature discontinuation of treatment, and resistance to help and intervention. Motivational interviewing has shown promise in assisting the provider to motivate patients with BPD to engage in therapy and effect positive change (***Table 6***) [230; 244].

Problem Solving

Patients with BPD typically possess poor judgment, poor problem solving, and an inability to tolerate distress. They can react to a crisis with behaviors strongly motivated to reduce the overwhelming distress they experience—typically impulsive, manipulative, or self-harming behaviors. This behavioral response creates a subsequent crisis,

PROBLEM-SOLVING IN A HYPOTHETICAL CLINGY AND DEPENDENT PATIENT WITH BPD	
Goal	**Example Script**
Problem identification: Identify a specific problem interfering with good medical care.	What is the problem here? What needs to be fixed?
Consider multiple potential solutions: Collaboratively consider and brainstorm alternative solutions to the agreed-on problem.	What might you do differently so that less care or support from others will not prevent you from following medical advice? What are the possible consequences of each option we identified in our brainstorming conversation?
Seek patient commitment: Obtain patient commitment to trying a new, preferred solution and to set a starting time.	Which solutions are you willing to try? Please state exactly what you are going to do and when. When are you willing to start?
Summary statement: Summarize main points of the discussion, schedule follow-up to assess outcomes so patient is not discouraged if the first solution does not work, address new barriers that may arise, encourage patient to apply the solution consistently, and model positive self-reinforcement for small initial success.	If I may summarize our discussion, the problem seems to be a lack of self-confidence and, perhaps, a fear of disapproval when you need to make decisions about your medical care. These lead to being too dependent on others for making choices. You are willing to try a new solution or plan with my encouragement, and you are specifically going to do <new behavior> starting <day>. Does this sum up the plan fairly? Let's discuss this again in a couple of weeks to check your progress and coping with barriers to change and to modify the solution, if necessary.
Source: [244]	*Table 7*

prompting a maladaptive response, and a vicious cycle is created. Patients may also respond to stress or a crisis with regressive behavior or by shutting down [224].

The problem-solving technique can be used to intervene in patients with BPD to assist them in identifying a more adaptive solution to the current problem and empowering them to change the maladaptive behavior (***Table 7***). Problem solving is performed by implementing the following steps [224]:

1. Define the problem.
2. Define the goal of problem solving.
3. Brainstorm solutions.
4. Choose a solution.
5. Troubleshoot the solution.
6. Implement the solution.
7. Evaluate the effectiveness of the solution.

PHARMACOTHERAPY

Several systematic reviews and meta-analyses of pharmacotherapy for BPD have been published since 2010. In these reviews, studies are selected using highly stringent criteria, requiring a randomized, double-blind design using a placebo or comparison treatment control group. Controlling for placebo effect is important in evaluating symptom reduction in subjects with BPD, as they have shown a propensity for high placebo response rates. On the other hand, many studies are eliminated in such reviews, which in many cases leaves a single evaluable study for a given drug and tentative inference from the results [42; 201].

EVIDENCE-BASED PRACTICE RECOMMENDATION

According to the National Health and Medical Research Council, pharmacotherapy should not be used as primary therapy for BPD, because available medications have only modest and inconsistent effects and do not change the nature and course of the disorder.

(https://bpdfoundation.org.au/images/mh25_borderline_personality_guideline.pdf. Last accessed March 24, 2020.)

Strength of Recommendation/Level of Evidence: Evidence-based recommendation B (Body of evidence can be trusted to guide practice in most situations.)

Medications may effectively reduce a single or narrow range of targeted symptoms in BPD but have not yet shown convincing efficacy in addressing the core features (i.e., frantic efforts to avoid abandonment, emptiness, identity disturbance, and dissociation). Medication is considered adjunctive to psychotherapy, and prescribing psychotropic medication can help build a positive alliance with the patient with BPD. The selection and prescribing of medication for BPD is more complicated than in patients with other psychiatric conditions [18; 199]. For example, patients with BPD are often highly perceptive to physiologic stimuli and medication side effects. Several strategies can be used to help optimize pharmacotherapy response and minimize interference from BPD pathology [18; 42; 214]:

- Emphasize the need for collaboration.
- Set realistic expectations that medications are unlikely to produce BPD symptom remission and that therapeutic effects may be difficult to assess.
- Involve the patient to help identify therapeutic targets, improve compliance, ensure safety, weigh possible benefits against drawbacks from side effects.
- Do not prescribe prophylactically, only with patient request or when severely distressed. When requested but patient is not severely distressed, pharmacotherapy may still be considered to help establish an alliance. In these cases, be cautious and prescribe selective serotonin reuptake inhibitors, because despite modest benefits, they carry a low lethality risk in overdose.
- If a patient is severely distressed but declines medication, encourage but do not push.
- Use judgment in assessing medication benefit, because patients with BPD may value or devalue medications as a proxy for their perception of the prescriber relationship. Patients' decisions regarding medication may be based on fear of being controlled, not feeling cared for, or expectation of being cured.
- Establish a policy that if medication response is absent, initiation of an alternate medication is contingent on full taper of the first medication (or cross-taper in severely distressed patients).
- Stress the necessity for responsible usage to evaluate effectiveness.
- When pharmacotherapy is used to help manage a BPD crisis, the medication should be withdrawn after the crisis has been resolved. The treatment course,dose, planned duration, and review intervals should be documented and communicated to other prescribers involved in the patient's care.

Several randomized controlled trials of BPD pharmacotherapy have been conducted, usually with small samples, variable outcome measures, and limited duration [214]. No medication has been found uniformly or dramatically effective, and no drug has received U.S. Food and Drug Administration approval as effective in the treatment of BPD. Pharmaceutical industry-sponsored research has been limited due to concerns over violent or suicidal acts and associated liability risk.

Polypharmacy is associated with multiple side effects and has not shown improved efficacy over monotherapy. In fact, the number of prescribed medications is inversely related to improvement. Minimal attention has been given to medication effects on interpersonal relationships.

In most trials, antipsychotic medications were typically dosed at about one-third to one-half of the dose utilized for primary psychotic disorders. Mood stabilizer dosing was similar to the dose used for the treatment of bipolar disorder, and antidepressant dosing tended to be higher than the dose used for the treatment of MDD [232].

Among the more rigorously designed clinical trials of drug therapy for BPD, no drug agent has been found effective in improving the core features of abandonment fears and hypersensitivity, chronic feelings of emptiness, identity disturbance, or dissociation. This may be due to outcome assessments lacking the ability to detect change in these symptoms or because these core BPD symptoms are not treatable with currently available drug therapies [196].

ANTIDEPRESSANTS

Aside from the finding of amitriptyline efficacy in reducing depression associated with BPD, tricyclic antidepressants have been found ineffective across a range of outcome measures. This may be due to the prominent anticholinergic side effects further compromising the already tenuous behavioral control over impulsivity, aggression, and suicidality [197].

Monoamine oxidase inhibitors have been preferentially used in patients with atypical depression, characterized by rejection sensitivity and affective reactivity [193]. Their efficacy in this patient subgroup and overlapping interpersonal features with BPD prompted clinical trials in patients with BPD. Phenelzine led to pronounced improvements in aggression, hostility, and anxiety, but clinical use is limited by side effects that can be difficult to tolerate. The serious side effect of hypertensive crisis can be avoided only through rigid adherence to a restrictive diet [190; 201; 236].

Overall, antidepressants have not shown significant therapeutic benefit and lack strong recommendations in treating BPD. The generally modest effect sizes may reflect the inability of current antidepressants to selectively target receptors or mesocorticolimbic brain regions associated with amygdala hyper-reactivity [155; 156; 157].

ANTIPSYCHOTICS

Trials of first-generation or "typical" antipsychotics found significant improvements in anger with haloperidol and suicidality with flupentixol, but overall, typical antipsychotics were not found beneficial for psychosis, irritability, or affective symptoms [201].

Second-generation atypical antipsychotics are more frequently prescribed for BPD, because they possess greater tolerability and show a broader therapeutic range due to their serotonergic and noradrenergic activity. Broadly, atypical antipsychotics are efficacious in reducing impulsive aggression, mood instability, anxiety, anger, impulsivity, and cognitive symptoms. Olanzapine and aripiprazole have accounted for a sizeable proportion of positive clinical trial results and have shown significantly improved affective instability, impulsivity, psychosis, and interpersonal dysfunction [197; 201; 251].

Aripiprazole has the added benefit of a long half-life and favorable metabolic profile, making administration easier and possibly increasing adherence and therapeutic benefit. As a partial agonist at D2 and 5-HT1A receptors and an antagonist at 5-HT2A receptors, aripiprazole may possess greater efficacy in reducing the impulsivity and aggression associated with BPD [141; 142]. Despite a similar mechanistic profile, ziprasidone has not shown benefit in BPD [139].

Dose ranges are usually lower than for primary psychotic disorders. Metabolic side effects, such as weight gain and type 2 diabetes, are more common with atypical than with typical antipsychotics. Because eating disorders and obesity frequently co-occur with BPD, careful consideration is required of this side effect profile when treating patients with BPD [131].

MOOD STABILIZERS

The mood stabilizers carbamazepine, valproate, lamotrigine, and topiramate have received the most evaluation for treating BPD. A class-wide benefit of moderate-to-large effect size has been demonstrated with anticonvulsant medications, as shown by improvements in impulsive aggression, affective instability, and overall functioning. While anticonvulsants possess the common pharmacologic effect of stabilizing excitatory neurotransmission, their effects on glutamate and gamma-aminobutyric acid (GABA) signaling and broader mechanism of action are heterogeneous [201]. A greater improvement of overall functioning has been shown with these agents compared with atypical antipsychotics [132]. Topiramate may benefit anger and interpersonal functioning in particular, but adverse cognitive effects may impede full engagement and benefit from psychotherapy for some patients with BPD. Topiramate use is associated with weight loss, which may become problematic when comorbid eating disorders are present. Lamotrigine is found to improve impulsivity, affective symptoms, and aggression, but mitigation of potential side effects of life-threatening skin rash and toxicity requires prolonged dose titration. Valproate is particularly effective in patients with BPD and prominent impulsive aggression, to a greater extent than in patients with prominent affective instability [132; 133; 134].

STUDY OUTCOMES USING A SYMPTOM-TARGETED APPROACH

Areas of research interest have involved a targeted approach to address highly problematic symptoms of BPD. Some studies have used drug agents alone, and others have investigated drug agents that interact synergistically with psychotherapies to produce added benefit. Specifically, the mechanism of action of certain drug therapies are theorized to preferentially target and reduce BPD symptoms that inhibit learning and benefit from psychotherapy. Specific symptom domains and associated characteristics of these targeted approaches include cognitive-perceptual disturbances, impulsivity, and affective dysregulation. This area of research is likely to produce important findings that may greatly benefit the comprehensive treatment of BPD [199; 232].

In one randomized trial, 24 female patients with BPD and high levels of irritability and anger received six months of dialectical behavioral therapy alone or plus olanzapine. Both groups showed significant improvements in irritability, depression, aggression, and self-injury, but the olanzapine group showed more rapid decreases in irritability and aggression [135]. A similar double-blind, placebo-controlled study randomized 60 patients with BPD to four months of dialectical behavioral therapy plus either olanzapine or placebo. The olanzapine group showed a lower drop-out rate and greater overall symptom improvement [129].

OTHER PHARMACOLOGIC THERAPIES

Omega-3 Fatty Acids

Omega-3 fatty acids have received increasing recognition for their benefit in mood stabilization. One study of omega-3 ethyl-eicosapentaenoic acid supplementation in patients with moderate-to-severe BPD found efficacy in decreasing aggression and affective symptoms [53].

Clonidine

A trial of patients with comorbid PTSD and BPD found some benefit with clonidine, an α-adrenergic agonist, but this effect was limited to alleviation of PTSD symptoms [125].

Naltrexone

Preliminary trials of opioid antagonists have shown limited and inconsistent benefit in patients with BPD. A placebo-controlled trial of naltrexone failed to demonstrate significant improvement in dissociative symptoms [118; 119].

MEDICATIONS TO AVOID IN PATIENTS WITH BPD

Use of benzodiazepines has not received empirical support and is generally contraindicated for patients with BPD due to the risks of behavioral disinhibition, addiction, and overdose. Benzodiazepines also impose the risks of inhibiting learn-

ing and interfering with skills acquirement [236]. Tricyclic antidepressants also pose a high risk of toxicity in overdose, including death due to fatal arrhythmia, and are generally avoided [104].

ADDRESSING THE FAMILY

There is broad awareness that families of patients with BPD should, in most cases, be involved in the therapeutic process. One reason is that destructive family dynamics can greatly contribute to treatment drop-out by patients with BPD. In addition, families typically experience significant distress from living with and trying to cope with the problems of the patient with BPD. Regardless of the role family played in life adversity of the patient with BPD, they can become entangled in dysfunctional relationships with the patient that impede treatment. Family intervention can include providing psychoeducation concerning BPD and its origin, course, and treatment; teaching family members problem-solving skills to address difficult patient dynamics and provide the patient with validation; and transmit other communication skills to address the emotional reactivity of the relative with BPD [97; 98; 102].

THE ROLE OF THE FAMILY

Psychiatry and psychotherapy have traditionally focused on the individual patient, with limited or no contact with the family; this is partially the result of ethics and patient confidentiality laws. However, the field is increasingly recognizing the importance of family involvement in cases of BPD. There may be relatively high-functioning patients with BPD whose therapy is best conducted in the traditional, "individual therapy-only" approach. However, many patients with BPD are adolescents or young adults who are involved with and dependent on their families, and even patients in their 30s or 40s may remain dependent on their parents or a partner/spouse. In both of these groups, the ongoing contact with and dependency on families dictate the need to involve their families [171].

HIERARCHY OF FAMILY INTERVENTIONS

Several interventions are available for family members of patients with BPD, with varying levels of intensity [214]. The first level is basic psychoeducation. This should be offered to all parents, spouses, and involved others and has the lowest intensity. The next level is counseling, which involves meeting with a therapist who assists family members with advice and problem solving. Families usually welcome these sessions. Support groups are offered in the community where available and include Family Connections, sponsored by the National Education Alliance for Borderline Personality Disorder (NEA-BPD), and various support groups through the National Alliance on Mental Illness. Attendance and involvement can be ongoing and long-term. Conjoint therapy sessions with the patient and parents can be useful for planning, problem solving issues related to budget, sleep hygiene, treatment adherence, emergencies, and provider vacations. This intervention can be very helpful in sustaining the holding environment and decreasing patient splitting. It is usually led by a family counselor, primary care clinician, or both.

The most intensive option is family therapy. This intervention may be destructive unless patient and parents/family can discuss conflicts without interruption, angry outbursts, or storming out of the session. Parent blaming can be useful only when parents are willing to accept, with regrets, whatever allegations by the patient are true.

GENERAL APPROACH FOR WORKING WITH FAMILIES OF PATIENTS WITH BPD

Family members can benefit when the family is viewed as the secondary client. As every patient is unique, the therapist should adapt the nature of family involvement to specific patient background and needs, while applying the following general principles [171].

EVIDENCE-BASED PRACTICE RECOMMENDATION

The National Health and Medical Research Council asserts that health professionals should advise families, partners, and carers of patients with BPD about helpful ways of interacting with the patient, including:

- Showing empathy and a non-judgmental attitude
- Encouraging the person to be independent by allowing and supporting him/her to make his/her own decisions, but intervening for safety when necessary
- Listening to the person with BPD when he/she expresses problems and worries

(https://bpdfoundation.org.au/images/mh25_borderline_personality_guideline.pdf. Last accessed March 24, 2020.)

Strength of Recommendation/Level of Evidence: Consensus-based recommendation (Recommendation formulated by the guideline development committee/group, using a consensus-reaching process, in the absence of high-quality evidence.)

Include Families in Initial Patient Evaluation

Involving the family allows access to valuable input regarding patient history and clinical presentation. It also provides the opportunity for family education on the nature of BPD and a realistic understanding of treatment options and long-term course [171].

Psychoeducation

Therapist discussion of BPD and referral to informational resources are critical during the evaluation phase. The therapist should know that a better-informed family usually translates into greater help and added treatment benefit for the patient [171]. Families often blame themselves for their relative's BPD, and psychoeducation helps the family understand the complex nature of BPD and avoid assigning blame, regardless of patient perception or accusation.

Psychoeducation includes describing the disorder, expected symptoms of the illness and how best to respond, and information on the biology and psychology of the illness and how these interact. Positive but realistic expectations for treatment should be conveyed. Specialized treatments now show a track record of long-term improvement, but given the 10% fatality rate from suicide, the family should have a guarded optimism and patience with their relative while he or she participates in the treatment process. This realistic appraisal can help families better prepare for the process ahead.

Discussing the role of the family in the patient's life is essential because, despite their best intentions, some families sabotage progress (e.g., by providing financial support without guidelines). The provider can help the family understand to what extent support is realistic and how to balance patient support with patient empowerment for eventual autonomy.

A Communication Plan

Parameters for patient communication should be established during initial treatment. While patient communication to the therapist is confidential (except with potentially life-threatening content), the family can access the therapist to communicate concerns the patient may not disclose [171]. The nature of family communication should be tailored to patient need, such as the option for family to call the therapist with concerns or structuring the communication to include a monthly family meeting. In most cases, it is prudent to ask the patient to sign a consent form for release of information between therapist and family.

As the communication system is discussed and developed, encourage the patient and family to communicate as openly as possible with each other. When the therapist becomes the conduit of information between patient and family, this is a misuse of therapy.

The Family as Secondary Client

Addressing family as the secondary client affirms the importance of assessing family needs as an aspect of care that engages the entire family system. The clinician can inform and support the family in gaining coping strategies for their distress and confusion resulting from chronic exposure to their relative's BPD symptoms [171]. For patients with severe BPD symptoms, the negative emotional

impact on family members is best addressed by referral to family therapy. In family therapy, all parties participate to resolve communication problems and other family system stressors.

Family members can also be referred to additional support, such as that provided by the Treatment and Research Advancements for Borderline Personality Disorder (TARA) and the NEA-BPD Family Connections System models. The TARA model uses family psychoeducation (including an in-depth understanding of BPD) and elements of treatment approaches that can be applied to family communication. The NEA-BPD model uses the principles of dialectical behavioral therapy to help families better understand BPD and incorporate dialectical behavioral concepts into their communication with the patient.

A good general resource for families is the BPD Resource Center website (https://www.nyp.org/bpdresourcecenter). This website includes access to trained specialists who can address questions, give information, and provide linkage to additional resources. It also allows communication with others who have also experienced the stress of living with a family member with BPD.

Setting Expectations

All parties should understand the course of BPD treatment can be intense and may occasionally be very difficult. Periods of greatest difficulty may be triggered by external events and for reasons that are unclear at the moment. It is essential for the provider, patient, and family to continue working together during the most difficult periods with honest communication [171].

Providers working with the BPD population should have the ability to "weather the storm" of intense displays of patient affect without over-reacting or retreating. By "containing" intense patient affects, the provider shows the patient and family that these discharges of emotion can be experienced, reflected on, and mastered. If the patient's condition worsens during treatment, the provider should obtain outside consultation with an expert to determine if another approach could be more helpful. In most cases, the best provider strategy during symptom exacerbation is to continue working with the patient and family using the same therapy modality.

Family Assistance to Monitor Medications

Medication is often part of the broader treatment plan. In some cases, the patient with BPD requires a medication choice that carries greater toxicity or lethality risk with overdose. Family members should be fully informed about medications, including the specific target symptoms, anticipated symptom changes from the medication, potential side effects, and actions to take in case of emergency [171].

ANGER TOWARD FAMILY

One challenge for clinicians is determining how to respond to the frequent anger and alienation patients with BPD feel toward their families. Family members also often feel anger and helplessness toward the patient with BPD—similar to what clinicians may experience when working with these patients. Failure to recognize this can aggravate the alienation of family members at a time when emotional and financial support is needed. Clinical experience has found that family involvement improves understanding of BPD and patient support, facilitates patient-family communication, and decreases the emotional and financial burden imposed by the relative with BPD [49; 91; 98].

TREATMENT OF COMORBIDITIES

The literature provides limited guidance on managing most of the common psychiatric comorbidities found with BPD. The first step is the careful diagnosis and differential diagnosis of BPD and comorbid disorders. The clinical course of comorbidities are connected to the course of BPD [41].

In patients with BPD and current co-occurring psychiatric conditions, the question arises over which disorder should be first addressed. ***Table 8*** shows which comorbidities, such as active substance use disorder, require initial management, and which comorbidities, such as MDD, are unlikely to

PSYCHIATRIC COMORBIDITY IN BPD: DETERMINING PRIMARINESS				
Comorbid Condition	**Prevalence in BPD**	**BPD Prevalence in Other Disorder**	**BPD Primary[a]**	**Rationale for Treatment Sequence**
Major depressive disorder	50%	15%	Yes	Should remit with BPD remission
Panic disorder	Unknown	Unknown	Yes	Will remit if BPD does, can precipitate BPD relapse
Substance use disorder	35%	10%	No	Three to six months of sobriety makes BPD treatment feasible
Antisocial personality disorder	25%	25%	Unknown	Determine if treatment is for secondary gain
Narcissistic personality disorder	15%	25%	Yes	Will improve if BPD does
Post-Traumatic Stress Disorder				
Overall	30%	8%	—	—
Complex, early-onset	—	—	No	Too vigilant to attach/be challenged
Adult-onset	—	—	Yes	BPD predisposes to onset, and PTSD should remit if BPD does
Bipolar Disorder				
Overall	15%	15%	—	—
Manic	—	—	No	Unable to use BPD therapy
Not manic	—	—	Yes	Recurrence lower if BPD remits
Eating Disorders				
Overall	20%	20%	—	—
Anorexia	—	—	No	Unable to use BPD treatment
Bulimia	—	—	Unknown	Determine if physical health is stable
[a]If BPD is primary, BPD should be the initial focus of intervention. If BPD is not primary, the comorbidity should be addressed first.				
Source: [214]				*Table 8*

respond to therapy if BPD is neglected. This latter group tends to improve in tandem with improvement in BPD.

MAJOR DEPRESSIVE DISORDER

Although MDD is virtually ubiquitous as a comorbidity in BPD and despite some overlap between MDD and BPD symptoms (such as chronic dysphoria in BPD and sadness and worthlessness in MDD), patients meeting full criteria may not benefit from antidepressants. The reasons for reduced antidepressant response remain unclear. Clinical trials have found that in patients with BPD and a co-occurring major depressive episode, improvement in BPD symptoms resulted in later improvements in major depressive symptoms, but the reverse was not found. In addition, patients with BPD receiving psychotherapy have shown reductions in self-reported depressive symptoms. Thus, specific treatment that targets BPD may be effective treatment for both disorders [41; 202; 251].

Although some pharmacotherapy trials have found reductions in comorbid depressive symptoms, these results are difficult to interpret, as most of the studies excluded patients with comorbid MDD. Furthermore, while the interventions led to reductions in subsyndromal depression symptoms, remission rates in patients with comorbid BPD and MDD were not evaluated. Studies combining medication and specialized psychotherapy have shown

mixed results, generally supporting the conclusion that treatment of BPD leads to improvement in depressive symptoms. Psychotherapy is generally more important than pharmacotherapy in positive treatment outcomes of BPD and comorbid MDD [41; 251].

BIPOLAR DISORDER

For the subset of patients in whom BPD and comorbid bipolar disorder are accurately diagnosed, mood stabilizers are required for the treatment of bipolar disorder and specialized psychotherapy is required for treating BPD [11].

ANXIETY DISORDERS

When comorbid with BPD, the course of anxiety disorders is similar to that of MDD comorbidity. Positive outcomes have been found with psychotherapies, with both conditions benefiting primarily from specialized psychotherapy designed for BPD [41].

POST-TRAUMATIC STRESS DISORDER

Comorbid PTSD is a more complex problem. It is important that BPD is not treated as a variant of PTSD. Evidence-based psychotherapies for BPD, such as dialectical behavioral therapy, tend to focus on the present and short-term future, and these alone are not helpful for PTSD. In some cases, time-limited, evidence-based CBT for PTSD has been useful [41].

SUBSTANCE USE DISORDER

An ongoing substance use disorder is highly important in the course of BPD and can be very problematic. There is some evidence that active substance use disorder is associated with more severe BPD symptoms and a worse intermediate-term prognosis, but the adverse effects on prognosis may attenuate over time. Clinical trials of patients with BPD and comorbid substance use disorder suggest that successful psychotherapy can reduce BPD and substance use disorder symptoms. Added benefits have been found using a dialectical behavioral therapy-based smartphone application in reducing substance use urges [92; 93].

EVIDENCE-BASED PRACTICE RECOMMENDATION

If a patient's substance use is severe, life-threatening, or interfering with BPD therapy, the National Health and Medical Research Council recommends that health professionals actively work to engage the patient in effective BPD treatment, but give priority in the first instance to the stabilization of the substance use disorder to allow effective BPD treatment.

(https://bpdfoundation.org.au/images/mh25_borderline_personality_guideline.pdf. Last accessed March 24, 2020.)

Strength of Recommendation/Level of Evidence: Consensus-based recommendation (Recommendation formulated by the guideline development committee/group, using a consensus-reaching process, in the absence of high-quality evidence.)

EATING DISORDERS

When eating disorders are comorbid with BPD, rates of the eating disorder tend to decline over time, although change to another eating disorder may also occur. This suggests that eating disorder symptoms reflect the core impulsivity of BPD. Patients who exhibit serious weight loss from anorexia nervosa require treatment for the eating disorder before starting BPD therapy. Binging and purging behaviors can be addressed by the same treatment approaches used to reduce self-harming behaviors in specialized psychotherapies for BPD [41; 94].

PATIENT PROGNOSIS

As individuals with BPD age, their symptoms and/or the severity of the illness often diminish. Following hospitalization and involvement in therapy, about 40% to 50% of patients with BPD remit within two years, and this rate rises to 85% by 10 years. Unlike most other major psychiatric disorders, relapse is uncommon in those who achieve remission (defined as no longer meeting DSM criteria for BPD diagnosis) [3].

However, the course of BPD rarely, if ever, shows a simple linear improvement. The frequent alternation between progress and setback is emotionally draining for everyone involved. Long-term studies of the course of BPD found the first five years of treatment are typically the most crisis-ridden. A series of intense, unstable relationships that end angrily, with subsequent self-destructive or suicidal behaviors, are characteristic. Although such a pattern may persist for years, decreasing frequency and seriousness of self-destructive behaviors, decreased suicidal ideation and acts, and declining frequency and duration of hospitalization are early indicators of improvement. Following hospitalization, roughly 60% of patients with BPD are readmitted in the first 6 months; this declines to 35% 18 to 24 months after initial hospitalization. In aggregate, utilization of psychiatric care gradually diminishes over time to involve briefer, less intensive interventions [3].

Interpersonal and social functioning are much slower to improve, and improvements are usually smaller in magnitude than with other BPD symptoms. Approximately 25% of patients with BPD eventually achieve stable relationships or successful vocational adjustment. Many more show limited vocational success and become more avoidant of close relationships. While many patients attain symptom stabilization and improved life satisfaction, impairment of social role functioning is highly persistent and often disappointing [3].

CONCLUSION

Primary care clinicians are the providers from whom patients with BPD are most likely to seek medical or psychiatric care, and the longitudinal nature of BPD requires that patients have continuity of care in the primary care setting. It is imperative that all healthcare professionals receive the latest research-informed education concerning the nature of BPD, effective psychosocial and drug therapies, and interaction strategies to avoid being drawn into the patient's pathologic personality traits [244].

RESOURCES

The Linehan Institute Behavioral Tech
1107 NE 45th Street, Suite 114
Seattle, Washington 98105
(206) 675-8588
https://behavioraltech.org

Borderline Personality Disorder Resource Center
New York Presbyterian Hospital
(888) 694-2273
https://www.nyp.org/bpdresourcecenter

National Education Alliance for Borderline Personality Disorder (NEA-BPD)
https://www.borderlinepersonalitydisorder.com

New England Personality Disorder Association (NEPDA), Inc.
115 Mill Street
Belmont, Massachusetts 02478
http://www.nepda.org

Personality Disorders Awareness Network
1072 West Peachtree Street NW #79468
Atlanta, Georgia 30357
(937) 732-9273
http://www.pdan.org

Treatment and Research Advancements for Borderline Personality Disorder (TARA)
23 Greene Street
New York, NY 10013
(212) 966-6514
(888) 4-TARABPD
http://www.tara4bpd.org

Works Cited

1. Grant BF, Chou SP, Goldstein RB, et al. Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *J Clin Psychiatry*. 2008;69(4):533-545.
2. Trull TJ, Jahng S, Tomko RL, Wood PK, Sher KJ. Revised NESARC personality disorder diagnoses: gender, prevalence, and comorbidity with substance dependence disorders. *J Pers Disord*. 2010;24(4):412-426.
3. Gunderson JG. A BPD Brief: An Introduction to Borderline Personality Disorder: Diagnosis, Origins, Course, and Treatment. Available at https://www.borderlinepersonalitydisorder.com/wp-content/uploads/2011/07/A_BPD_Brief_REV2011.pdf. Last accessed February 3, 2020.
4. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. Washington, DC: American Psychiatric Publishing; 2013.
5. Howe E. Five ethical and clinical challenges psychiatrists may face when treating patients with borderline personality disorder who are or may become suicidal. *Innov Clin Neurosci*. 2013;10(1):14-19.
6. Gunderson JG. Borderline personality disorder: ontogeny of a diagnosis. *Am J Psychiatry*. 2009;166(5):530-539.
7. Gunderson JG, Kolb JE. Discriminating features of borderline patients. *Am J Psychiatry*. 1978;135(7):792-796.
8. Substance Abuse and Mental Health Services Administration. Report to Congress on Borderline Personality Disorder. Available at http://www.ncdsv.org/images/SAMHSA_Report-to-Congress-on-Borderline-Personality-Disorder_5-2011.pdf. Last accessed February 14, 2017.
9. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 3rd ed. Washington, DC: American Psychiatric Publishing; 1980.
10. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed., Text Revision. Washington, DC: American Psychiatric Association; 2000.
11. Paris J, Gunderson J, Weinberg I. The interface between borderline personality disorder and bipolar spectrum disorders. *Compr Psychiatry*. 2007;48(2):145-154.
12. New AS, Triebwasser J, Charney DS. The case for shifting borderline personality disorder to Axis I. *Biol Psychiatry*. 2008;64(8): 653-659.
13. Samuel DB, Widiger TA, Pilkonis PA, Miller JD, Lynam DR, Ball SA. Conceptual changes to the definition of borderline personality disorder proposed for DSM-5. *J Abnorm Psychol*. 2012;121(2):467-476.
14. Oldham JM. The alternative DSM-5 model for personality disorders. *World Psychiatry*. 2015;14(2):234-236.
15. Biskin RS, Paris J. Diagnosing borderline personality disorder. *CMAJ*. 2012;184(16):1789-1794.
16. Lenzenweger MF, Loranger AW, Korfine L, Neff C. Detecting personality disorders in a nonclinical population: application of a two-stage procedure for case identification. *Arch Gen Psychiatry*. 1997;54(4):345-351.
17. Torgersen S, Kringlen E, Cramer V. The prevalence of personality disorders in a community sample. *Arch Gen Psychiatry*. 2001;58(6):590-596.
18. Gunderson JG, Weinberg I, Choi-Kain L. Borderline personality disorder. *FOCUS*. 2013;11(2):129-145.
19. Saha S, Chant D, Welham J, McGrath J. A systematic review of the prevalence of schizophrenia. *PLoS Med*. 2005;2(5):e141.
20. Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry*. 2005;62(6):593-602.
21. Ellison WD, Rosenstein LK, Morgan TA, Zimmerman M. Community and clinical epidemiology of borderline personality disorder. *Psychiatr Clin North Am*. 2018;41(4):561-573.
22. Laporte L, Paris J, Zelkowitz P. Estimating the prevalence of borderline personality disorder in mothers involved in youth protection services. *Personal Ment Health*. 2018;12(1):49-58.
23. Hayashi N, Igarashi M, Imai A, et al. Psychiatric disorders and clinical correlates of suicidal patients admitted to a psychiatric hospital in Tokyo. *BMC Psychiatry*. 2010;10:109.
24. Gross R, Olfson M, Gameroff M, et al. Borderline personality disorder in primary care. *Arch Intern Med*. 2002;162(1):53-60.
25. Reid WH. Borderline personality disorder and related traits in forensic psychiatry. *J Psychiatr Pract*. 2009;15(3):216-220.
26. Black DW, Gunter T, Allen J, et al. Borderline personality disorder in male and female offenders newly committed to prison. *Compr Psychiatry*. 2007;48(5):400-405.
27. Torgersen S. Epidemiology. In: Wideger T (ed). *The Oxford Handbook of Personality Disorders*. New York, NY: Oxford University Press; 2012: 186-205.
28. Lenzenweger MF, Lane MC, Loranger AW, Kessler RC. DSM-IV personality disorders in the national comorbidity survey replication. *Biol Psychiatry*. 2007;62(6):553-564.

29. Grilo CM, Pagano ME, Skodol AE, et al. Natural course of bulimia nervosa and of eating disorder not otherwise specified: 5-year prospective study of remissions, relapses, and the effects of personality disorder psychopathology. *J Clin Psychiatry*. 2007;68(5):738-746.
30. Zanarini MC, Frankenburg FR, Ridolfi ME, Jager-Hyman S, Hennen J, Gunderson JG. Reported childhood onset of self-mutilation among borderline patients. *J Pers Disord*. 2006;20(1):9-15.
31. Loranger AW, Sartorius N, Andreoli A, et al. The International Personality Disorder Examination: The World Health Organization/Alcohol, Drug Abuse, and Mental Health Administration international pilot study of personality disorders. *Arch Gen Psychiatry*. 1994;51(3):215-224.
32. Stepp SD, DJ Whalen, PA Pilkonis, AE Hipwell, MD Levine. Children of mothers with borderline personality disorder: identifying parenting behaviors as potential targets for intervention. *Personal Disord*. 2011;3(1):76-91.
33. Carlson EA, Egeland B, Sroufe LA. A prospective investigation of the development of borderline personality symptoms. *Dev Psychopathol*. 2009;21(4):1311-1334.
34. Chanen AM, Kaess M. Developmental pathways to borderline personality disorder. *Curr Psychiatry Rep*. 2012;14(1):45-53.
35. Cohen P. Child development and personality disorder. *Psychiatr Clin North Am*. 2008;31(3):477-493.
36. Fruzzetti AE, Shenk C, Hoffman PD. Family interaction and the development of borderline personality disorder: a transactional model. *Dev Psychopathol*. 2005;17(4):1007-1030.
37. McLean LM. The relationship between early childhood sexual abuse and the adult diagnoses of borderline personality disorder and complex posttraumatic stress disorder: diagnostic implications. *Dissertation Abstracts International*. 2001;62(04B):2069.
38. McLean LM, Gallop R. Implications of childhood sexual abuse for adult borderline personality disorder and complex post-traumatic stress disorder. *Am J Psychiatry*. 2003;160(2):369-371.
39. Stepp SD, Lazarus SA, Byrd AL. Systematic review of risk factors prospectively associated with borderline personality disorder: taking stock and moving forward. *Personal Disord*. 2016;7(4):316-323.
40. O'Connor E, Bureau JF, McCartney K, Lyons-Ruth K. Risks and outcomes associated with disorganized/controlling patterns of attachment at age three in the NICHD study of early child care and youth development. *Infant Ment Health J*. 2011;32(4): 450-472.
41. Biskin RS, Paris J. Management of borderline personality disorder. *CMAJ*. 2012;184(17):1897-1902.
42. National Health and Medical Research Council. Clinical Practice Guideline for the Management of Borderline Personality Disorder. Available at https://www.nhmrc.gov.au/about-us/publications/clinical-practice-guideline-borderline-personality-disorder. Last accessed February 3, 2020.
43. National Institute for Health and Clinical Excellence. Borderline Personality Disorder: Treatment and Management. Available at https://www.nice.org.uk/guidance/cg78. Last accessed February 3, 2020.
44. Soeteman DI, Hakkaart-van Roijen L, Verheul R, Busschbach JJ. The economic burden of personality disorders in mental health care. *J Clin Psychiatry*. 2008;69(2):259-265.
45. Sansone RA, Sansone LA. Emotional hyper-reactivity in borderline personality disorder. *Psychiatry (Edgemont)*. 2010;7(9):16-20.
46. Sansone RA, Lam C, Wiederman MW. Road rage: relationships with borderline personality and driving citations. *Int J Psychiatry Med*. 2010;40(1):21-29.
47. Skoglund C, Tiger A, Rück C, et al. Familial risk and heritability of diagnosed borderline personality disorder: a register study of the Swedish population. *Mol Psychiatry*. 2019; [Epub ahead of print].
48. Committee on Child Maltreatment Research, Policy, and Practice for the Next Decade: Phase II; Board on Children, Youth, and Families; Committee on Law and Justice; Institute of Medicine; National Research Council; Petersen AC, Joseph J, Feit M (eds). *New Directions in Child Abuse and Neglect Research*. Washington, DC: Academies Press; 2014.
49. Gunderson JG. Borderline personality disorder. *N Engl J Med*. 2011;364:2037-2042.
50. Bender DS, Dolan RT, Skodol AE, et al. Treatment utilization by patients with personality disorders. *Am J Psychiatry*. 2001;158(2):295-302.
51. Sansone RA, Sellbom M, Songer DA. Borderline personality disorder and mental health care utilization: the role of self-harm. *Person Disord*. 2018;9(2):188-191.
52. Pascual JC, Córcoles D, Castaño J, et al. Hospitalization and pharmacotherapy for borderline personality disorder in a psychiatric emergency service. *Psychiatr Serv*. 2007;58(9):1199-1204.
53. Zanarini MC, Frankenburg FR, Parachini EA. A preliminary, randomized trial of fluoxetine, olanzapine, and the olanzapine-fluoxetine combination in women with borderline personality disorder. *J Clin Psychiatry*. 2004;65(7):903-907.
54. Zanarini MC, Frankenburg FR, Hennen J, Silk KR. Mental health service utilization by borderline personality disorder patients and Axis II comparison subjects followed prospectively for 6 years. *J Clin Psychiatry*. 2004;65(1):28-36.
55. Goodman M, Tomas IA, Temes CM, et al. Suicide attempts and self-injurious behaviours in adolescent and adult patients with borderline personality disorder. *Personal Ment Health*. 2017;11(3):157-163.

56. Black DW, Blum N, Letuchy E, Carney Doebbeling C, Forman-Hoffman VL, Doebbeling BN. Borderline personality disorder and traits in veterans: psychiatric comorbidity, healthcare utilization, and quality of life along a continuum of severity. *CNS Spectr*. 2006;11(9):680-689.
57. Bell RQ, Chapman M. Child effects in studies using experimental or brief longitudinal approaches to socialization. *Dev Psychol*. 1986;22:595-603.
58. Ge X, Conger RD, Cadoret RJ, et al. The developmental interface between nature and nurture: a mutual influence model of child antisocial behavior and parent behaviors. *Dev Psychol*. 1996;32(4):574-589.
59. Kendler KS. Parenting: a genetic-epidemiologic perspective. *Am J Psychiatry*. 1996;153(1):11-20.
60. Gunderson JG, Lyons-Ruth K. BPD's interpersonal hypersensitivity phenotype: a gene-environment-developmental model. *J Pers Disord*. 2008;22(1):22-41.
61. Prada P, Perroud N, Rüfenacht E, Nicastro R. Strategies to deal with suicide and non-suicidal self-injury in borderline personality disorder, the case of DBT. *Front Psychol*. 2018;9:2595.
62. Morgan TA, Zimmerman M. Is borderline personality disorder underdiagnosed and bipolar disorder overdiagnosed? In: Choi-Kain LW, Gunderson JG (eds.) *Borderline Personality and Mood Disorders: Comorbidity and Controversy*. Washington, DC; Springer Science+Business Media: 2015.
63. Carlson EA. A prospective longitudinal study of disorganized/disoriented attachment. *Child Dev*. 1998;69(4):1107-1128.
64. Bozzatello P, Bellino S, Bosia M, Rocca P. Early detection and outcome in borderline personality disorder. *Front Psychiatry*. 2019;10(710):1-16.
65. van IJzendoorn MH, Schuengel C, Bakermans-Kranenburg MJ. Disorganized attachment in early childhood: meta-analysis of precursors, concomitants, and sequelae. *Dev Psychopathol*. 1999;11(2):225-249.
66. Holmes J. Disorganized attachment and borderline personality disorder: a clinical perspective. *Attach Hum Dev*. 2004;6(2):181-190.
67. Allen DN, Becker ML. Diagnostic and symptom interviews for adults. In: Goldstein G, Allen DN, DeLuca J (eds). *Handbook of Psychological Assessment*. 4th ed. Cambridge, MA: Elsevier; 2019: 355-393.
68. Modell AH. Primitive object relationships and the predisposition to schizophrenia. *Int J Psychoanal*. 1963;44(3):282-292.
69. Masterson JF. Treatment of the adolescent with borderline syndrome: a problem in separation-individuation. *Bull Menninger Clin*. 1971;35(1):5-18.
70. Bradley SJ. The relationship of early maternal separation to borderline personality in children and adolescents: a pilot study. *Am J Psychiatry*. 1979;136(4A):424-426.
71. Gunderson JG, Kerr J, Englund DW. The families of borderlines: a comparative study. *Arch Gen Psychiatry*. 1980;37(1):27-33.
72. Zweig-Frank H, Paris J. Parents' emotional neglect and overprotection according to the recollections of patients with borderline personality disorder. *Am J Psychiatry*. 1991;148(5):648-651.
73. Reich DB, Zanarini MC. Developmental aspects of borderline personality disorder. *Harv Rev Psychiatry*. 2001;9(6):294-301.
74. O'Connor TG, Craft CM. A twin study of attachment in preschool children. *Child Dev*. 2001;72(5):1501-1511.
75. American Psychiatric Association. The Structured Clinical Interview for DSM-5. Available at https://www.appi.org/products/structured-clinical-interview-for-dsm-5-scid-5. Last accessed February 11, 2020.
76. Bokhorst CL, Bakermans-Kranenburg MJ, Fearon RM, van IJzendoorn MH, Fonagy P, Schuengel C. The importance of shared environment in mother-infant attachment security: a behavioral genetic study. *Child Dev*. 2003;74(6):1769-1782.
77. Loranger AW. IPDE-ICD-10 International Personality Disorder Examination. Available at https://www.annarbor.co.uk/index.php?main_page=index&cPath=416_248_207. Last accessed February 11, 2020.
78. Madigan S, Bakermans-Kranenburg MJ, Van Ijzendoorn MH, Moran G, Pederson DR, Benoit D. Unresolved states of mind, anomalous parental behavior, and disorganized attachment: a review and meta-analysis of a transmission gap. *Attach Hum Dev*. 2006;8(2):89-111.
79. Lyons-Ruth K, Yellin C, Melnick S, Atwood G. Expanding the concept of unresolved mental states: Hostile/helpless states of mind on the Adult Attachment Interview are associated with disrupted mother-infant communication and infant disorganization. *Dev Psychopathol*. 2005;17(1):1-23.
80. Hobson RP, Patrick M, Crandell L, García-Pérez R, Lee A. Personal relatedness and attachment in infants of mothers with borderline personality disorder. *Dev Psychopathol*. 2005;17(2):329-347.
81. White CN, Gunderson JG, Zanarini MC, Hudson JI. Family studies of borderline personality disorder: a review. *Harv Rev Psychiatry*. 2003;11(1):8-19.
82. Silverman JM, Pinkham L, Horvath TB, et al. Affective and impulsive personality disorder traits in the relatives of patients with borderline personality disorder. *Am J Psychiatry*. 1991;148(10):1378-1385.
83. Zanarini MC, Frankenburg FR, Yong L, et al. Borderline psychopathology in the first-degree relatives of borderline and Axis II comparison probands. *J Pers Dis*. 2004;18(5):439-447.
84. McMain SF, Links PS, Gnam WH, et al. A randomized trial of dialectical behavior therapy versus general psychiatric management for borderline personality disorder. *Am J Psychiatry*. 2009;166(12):1365-1374.

85. Witt SH, Streit F, Jungkunz M, et al. Genome-wide association study of borderline personality disorder reveals genetic overlap with bipolar disorder, major depression and schizophrenia. *Transl Psychiatry*. 2017;7(6):e1155.
86. Paris J. Suicidality in borderline personality disorder. *Medicina (Kaunas)*. 2019;55(6):223.
87. Storebø OJ, Stoffers-Winterling JM, Völlm BA, et al. Psychological therapies for people with borderline personality disorder. *Cochrane Database Syst Rev*. 2018;2.
88. Temes CM, Zanarini MC. Recent developments in psychosocial interventions for borderline personality disorder. *F1000Res*. 2019;8.
89. Meuldijk D, McCarthy A, Bourke ME, Grenyer BFS. The value of psychological treatment for borderline personality disorder: systematic review and cost offset analysis of economic evaluations. *PLoS One*. 2017;12(3):e0171592.
90. Lyons-Ruth K, Melnick S, Patrick M, Hobson RP. A controlled study of hostile-helpless states of mind among borderline and dysthymic women. *Attach Hum Dev*. 2007;9(1):1-16.
91. Gunderson JG, Lyoo IK. Family problems and relationships for adults with borderline personality disorder. *Harv Rev Psychiatry*. 1997;4(5):272-278.
92. Linehan MM, Dimeff LA, Reynolds SK, et al. Dialectical behavior therapy versus comprehensive validation therapy plus 12-step for the treatment of opioid dependent women meeting criteria for borderline personality disorder. *Drug Alcohol Depend*. 2002;67(1):13-26.
93. Rizvi SL, Dimeff LA, Skutch J, Carroll D, Linehan MM. A pilot study of the DBT coach: an interactive mobile phone application for individuals with borderline personality disorder and substance use disorder. *Behav Ther*. 2011;42(4):589-600.
94. Kröger C, Schweiger U, Sipos V, et al. Dialectical behaviour therapy and an added cognitive behavioural treatment module for eating disorders in women with borderline personality disorder and anorexia nervosa or bulimia nervosa who failed to respond to previous treatments: an open trial with a 15-month follow-up. *J Behav Ther Exp Psychiatry*. 2010;41(4):381-388.
95. Distel MA, Willemsen G, Ligthart L, et al. Genetic covariance structure of the four main features of borderline personality disorder. *J Pers Disord*. 2010;24(4):427-444.
96. Hankin BL, Barrocas AL, Jenness J, et al. Association between 5-HTTLPR and borderline personality disorder traits among youth. *Front Psychiatry*. 2011;1(2):6.
97. Goodman M, Patil U, Triebwasser J, Hoffman P, Weinstein ZA, New A. Parental burden associated with borderline personality disorder in female offspring. *J Pers Disord*. 2011;25(1):59-74.
98. National Education Alliance for Borderline Personality Disorder. Family Connections Program. Available at https://www.borderlinepersonalitydisorder.org/family-connections. Last accessed February 13, 2020.
99. Distel MA, Middeldorp CM, Trull TJ, et al. Life events and borderline personality features: the influence of gene-environment interaction and gene-environment correlation. *Psychol Med*. 2011;41(4):849-860.
100. Verschoor E, Markus CR. Affective and neuroendocrine stress reactivity to an academic examination: influence of the *5-HTTLPR* genotype and trait neuroticism. *Biol Psychol*. 2011;87(3):439-449.
101. Orr SP, Lasko NB, Macklin ML, et al. Predicting post-trauma stress symptoms from pre-trauma psychophysiologic reactivity, personality traits and measures of psychopathology. *Biol Mood Anxiety Disord*. 2012;2:8.
102. Gunderson JG, Frank AF, Ronningstam EF, Wachter S, Lynch VJ, Wolf PJ. Early discontinuance of borderline patients from psychotherapy. *J Nerv Ment Dis*. 1989;177(1):38-42.
103. Donegan NH, Sanislow CA, Blumberg HP, et al. Amygdala hyperreactivity in borderline personality disorder: implications for emotional dysregulation. *Biol Psychiatry*. 2003;54(11):1284-1293.
104. Soloff PH, George A, Nathan RS, Schulz PM, Perel JM. Behavioral dyscontrol in borderline patients treated with amitriptyline. *Psychopharmacol Bull*. 1987;23(1):177-181.
105. Ruocco AC, Medaglia JD, Ayaz H, Chute DL. Abnormal prefrontal cortical response during affective processing in borderline personality disorder. *Psychiatry Res*. 2010;182(2):117-122.
106. Silbersweig D, Clarkin JF, Goldstein M, et al. Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. *Am J Psychiatry*. 2007;164(12):1832-1841.
107. Ruocco AC, Amirthavasagam S, Choi-Kain LW, McMain SF. Neural correlates of negative emotionality in borderline personality disorder: an activation-likelihood-estimation meta-analysis. *Biol Psychiatry*. 2013;73(2):153-160.
108. Mayberg HS, Lozano AM, Voon V, et al. Deep brain stimulation for treatment-resistant depression. *Neuron*. 2005;45(4):651-660.
109. King-Casas B, Sharp C, Lomax-Bream L, Lohrenz T, Fonagy P, Montague PR. The rupture and repair of cooperation in borderline personality disorder. *Science*. 2008;321(5890):806-810.
110. LeGris J, van Reekum R. The neuropsychological correlates of borderline personality disorder and suicidal behaviour. *Can J Psychiatry*. 2006;51(3):131-142.
111. Fertuck EA, Lenzenweger MF, Clarkin JF, Hoermann S, Stanley B. Executive neurocognition, memory systems, and borderline personality disorder. *Clin Psychol Rev*. 2006;26(3):346-375.

112. Zimmerman DJ, Choi-Kain LW. The hypothalamic-pituitary-adrenal axis in borderline personality disorder: a review. *Harv Rev Psychiatry*. 2009;17(3):167-183.

113. Stanley B, Siever LJ. The interpersonal dimension of borderline personality disorder: toward a neuropeptide model. *Am J Psychiatry*. 2010;167(1):24-39.

114. Domes G, Heinrichs M, Michel A, Berger C, Herpertz SC. Oxytocin improves "mind-reading" in humans. *Biol Psychiatry*. 2007;61(6):731-733.

115. Bartz J, Simeon D, Hamilton H, et al. Oxytocin can hinder trust and cooperation in borderline personality disorder. *Soc Cogn Affect Neurosci*. 2011;6(5):556-563.

116. Stanley B, Sher L, Wilson S, Ekman R, Huang Y-Y, Mann JJ. Nonsuicidal self-injurious behavior, endogenous opioids and monoamine neurotransmitters. *J Affect Disord*. 2010;124(1-2):134-140.

117. Pike JL, Smith TL, Hauger RL, et al. Chronic life stress alters sympathetic, neuroendocrine, and immune responsivity to an acute psychological stressor in humans. *Psychosom Med*. 1997;59(4):447-457.

118. Philipsen A, Schmahl C, Lieb K. Naloxone in the treatment of acute dissociative states in female patients with borderline personality disorder. *Pharmacopsychiatry*. 2004;37(5):196-199.

119. Schmahl C, Kleindienst N, Limberger M, et al. Evaluation of naltrexone for dissociative symptoms in borderline personality disorder. *Int Clin Psychopharmacol*. 2012;27(1):61-68.

120. Joyce PR, McKenzie JM, Mulder RT, et al. Genetic, developmental and personality correlates of self-mutilation in depressed patients. *Aust N Z J Psychiatry*. 2006;40(3):225-229.

121. Davidson RJ, Irwin W. The functional neuroanatomy of emotion and affective style. *Trends Cogn Sci*. 1999;3(1):11-21.

122. Fuster JM. Frontal lobe and cognitive development. *J Neurocytol*. 2002;31(3-5):373-385.

123. Hughes AE, Crowell SE, Uyeji L, Coan JA. A developmental neuroscience of borderline pathology: emotion dysregulation and social baseline theory. *J Abnorm Child Psychol*. 2012;40(1):21-33.

124. Coan JA. Toward a neuroscience of attachment. In: Cassidy J, Shaver PR (eds). *Handbook of Attachment: Theory, Research, and Clinical Implications*. 3rd ed. New York, NY: The Guilford Press; 2016: 242-273.

125. Ziegenhorn AA, Roepke S, Schommer NC, et al. Clonidine improves hyperarousal in borderline personality disorder with or without comorbid post-traumatic stress disorder: a randomized, double-blind, placebo-controlled trial. *J Clin Psychopharmacol*. 2009;29(2):170-173.

126. Coan JA. Adult attachment and the brain. *Journal of Social and Personal Relationships*. 2010;27(2):210-217.

127. Kemeny ME. The psychobiology of stress. *Current Directions in Psychological Science*. 2003;12(4):124-129.

128. Rolls ET. Emotion elicited by primary reinforcers and following stimulus-reinforcement association learning. In: Coan JA, Allen JJB (eds). *The Handbook of Emotion Elicitation and Assessment*. New York, NY: Oxford University Press; 2007: 137-157.

129. Soler J, Pascual JC, Campins J, et al. Double-blind, placebo-controlled study of dialectical behavior therapy plus olanzapine for borderline personality disorder. *Am J Psychiatry*. 2005;162(6):1221-1224.

130. Way BM, Taylor SE, Eisenberger NI. Variation in the mu-opioid receptor gene (*OPRM 1*) is associated with dispositional and neural sensitivity to social rejection. *Proc Natl Acad Sci U S A*. 2009;106(35):15079-15084.

131. Frankenburg FR, Zanarini MC. Obesity and obesity-related illnesses in borderline patients. *J Personal Disord*. 2006;20(1):71-80.

132. Ingenhoven T, Lafay P, Rinne T, Passchier J, Duivenvoorden H. Effectiveness of pharmacotherapy for severe personality disorders: meta-analyses of randomized controlled trials. *J Clin Psychiatry*. 2010;71(1):14-25.

133. Mercer D, Douglass AB, Links PS. Meta-analyses of mood stabilizers, antidepressants and antipsychotics in the treatment of borderline personality disorder: effectiveness for depression and anger symptoms. *J Personal Disord*. 2009;23(2):156-174.

134. Hollander E, Swann AC, Coccaro EF, Jiang P, Smith TB. Impact of trait impulsivity and state aggression on divalproex vs. placebo response in borderline personality disorder. *Am J Psychiatry*. 2005;162:621-624.

135. Linehan MM, McDavid JD, Brown MZ, Sayrs JH, Gallop RJ. Olanzapine plus dialectical behavior therapy for women with high irritability who meet criteria for borderline personality disorder: a double-blind, placebo-controlled pilot study. *J Clin Psychiatry*. 2008;69(6):999-1005.

136. Carpenter RW, Trull TJ. Components of emotion dysregulation in borderline personality disorder: a review. *Curr Psychiatry Rep*. 2013;15(1):335.

137. Crowell SE, Beauchaine TP, Linehan MM. A biosocial developmental model of borderline personality: elaborating and extending Linehan's theory. *Psychol Bull*. 2009;135(3):495-510.

138. Domes G, Schulze L, Herpertz SC. Emotion recognition in borderline personality disorder-a review of the literature. *J Pers Disord*. 2009;23(1):6-19.

139. Pascual JC, Soler J, Puigdemont D, et al. Ziprasidone in the treatment of borderline personality disorder: a double-blind, placebo-controlled, randomized study. *J Clin Psychiatry*. 2008;69(4):603-608.

140. Kuo JR, Linehan MM. Disentangling emotion processes in borderline personality disorder: physiological and self-reported assessment of biological vulnerability, baseline intensity, and reactivity to emotionally evocative stimuli. *J Abnorm Psychol*. 2009;118(3):531-544.

141. Zanarini MC, Schulz SC, Detke HC, et al. A dose comparison of olanzapine for the treatment of borderline personality disorder: a 12-week randomized, double-blind, placebo-controlled study. *J Clin Psychiatry*. 2011;72(10):1353-1362.

142. Nickel MK, Loew TH, Pedrosa Gil F. Aripiprazole in treatment of borderline patients, part II: an 18-month follow-up. *Psychopharmacology (Berl)*. 2007;191(4):1023-1026.

143. Trull T, Solhan M, Tragesser SL, et al. Affective instability: measuring a core feature of borderline personality disorder with ecological momentary assessment. *J Abnorm Psychol*. 2008;117(3):647-661.

144. Hopwood CJ, Swenson C, Bateman A, Yeomans FE, Gunderson JG. Approaches to psychotherapy for borderline personality: demonstrations by four master clinicians. *Personal Disord*. 2014;5(1):108-116.

145. Suvak MK, Litz BT, Sloan DM, Zanarini MC, Barrett LF, Hofmann SG. Emotional granularity and borderline personality disorder. *J Abnorm Psychol*. 2011;120(2):414-426.

146. Coifman KG, Berenson KR, Rafaeli E, Downey G. From negative to positive and back again: polarized affective and relational experience in borderline personality disorder. *J Abnorm Psychol*. 2012;121(3):668-679.

147. Bornovalova MA, Matusiewicz A, Rojas E. Distress tolerance moderates the relationship between negative affect intensity with borderline personality disorder levels. *Compr Psychiatry*. 2011;52(6):744-753.

148. Selby EA, Anestis MD, Bender TW, Joiner TE. An exploration of the emotional cascade model in borderline personality disorder. *J Abnorm Psychol*. 2009;118(2):375-387.

149. Baer RA, Sauer SE. Relationships between depressive rumination, anger rumination, and borderline personality features. *Pers Disord Theory Res Treat*. 2011;2(2):142-150.

150. Rosenthal ZM, Cheavens JS, Lejuez CW, Lynch TR. Thought suppression mediates the relationship between negative affect and borderline personality disorder symptoms. *Behav Res Ther*. 2005;43(9):1173-1185.

151. Chapman AL, Dixon-Gordon KL, Walters KN. Experiential avoidance and emotion regulation in borderline personality disorder. *J Ration Emot Cogn Behav Ther*. 2011;29(1):35-52.

152. Whiteside SP, Lynam DR. The Five Factor Model and impulsivity: using a structural model of personality to understand impulsivity. *Personal Individ Differ*. 2001;30(4):669-689.

153. Tragesser SL, Robinson RJ. The role of affective instability and UPPS impulsivity in borderline personality disorder features. *J Pers Disord*. 2009;23(4):370-383.

154. Yen S, Zlotnick C, Costello E. Affect regulation in women with borderline personality disorder traits. *J Nerv Ment Dis*. 2002;190(10):693-696.

155. Higgins GA, Enderlin M, Haman M, Fletcher PJ. The 5-HT2A receptor antagonist M100,907 attenuates motor and "impulsive-type" behaviours produced by NMDA receptor antagonism. *Psychopharmacology (Berl)*. 2003;170(3):309-319.

156. Winstanley CA, Theobald DE, Dalley JW, Glennon JC, Robbins TW. 5HT2A and 5-HT2C receptor antagonists have opposing effects on a measure of impulsivity: interactions with global 5-HT depletion. *Psychopharmacology (Berl)*. 2004;176(3-4):376-385.

157. Homberg JR. Serotonin and decision-making processes. *Neurosci Biobehav Rev*. 2012;36(1):218-236.

158. Ludäscher P, Bohus M, Lieb K, Philipsen A, Jochims A, Schmahl C. Elevated pain thresholds correlate with dissociation and aversive arousal in patients with borderline personality disorder. *Psychiatry Res*. 2007;149(1-3):291-296.

159. Niedtfeld I, Kirsch P, Schulze L, Herpertz SC, Bohus M, Schmahl C. Functional connectivity of pain-mediated affect regulation in borderline personality disorder. *PLoS One*. 2012;7(3):e33293.

160. Belsky DW, Caspi A, Arseneault L, et al. Etiological features of borderline personality related characteristics in a birth cohort of 12-year-old children. *Dev Psychopathol*. 2012;24(1):251-265.

161. Johnson AB, Gentile JP, Correll TL. Accurately diagnosing and treating borderline personality disorder: a psychotherapeutic case. *Psychiatry (Edgemont)*. 2010;7(4):21-30.

162. American Psychiatric Association. *Practice Guideline for the Treatment of Patients with Borderline Personality Disorder*. Washington, DC: American Psychiatric Publishing; 2010.

163. Zanarini MC, Frankenburg FR, Reich DB, Fitzmaurice G. Attainment and stability of sustained symptomatic remission and recovery among patients with borderline personality disorder and axis II comparison subjects: a 16-year prospective follow-up study. *Am J Psychiatry*. 2012;169(5):476-483.

164. Gunderson JG, Stout RL, McGlashan TH, et al. Ten-year course of borderline personality disorder: psychopathology and function from the Collaborative Longitudinal Personality Disorders study. *Arch Gen Psychiatry*. 2011;68(8):827-837.

165. Zanarini MC, Frankenburg FR, Hennen J, Reich DB, Silk KR. The McLean Study of Adult Development (MSAD): overview and implications of the first six years of prospective follow-up. *J Pers Disord*. 2005;19(5):505-523.

166. Gunderson JG, Bender D, Sanislow C, et al. Plausibility and possible determinants of sudden "remissions" in borderline patients. *Psychiatry*. 2003;66(2):111-119.

167. Yen S, Pagano ME, Shea MT, et al. Recent life events preceding suicide attempts in a personality disorder sample: findings from the collaborative longitudinal personality disorders study. *J Consult Clin Psychol*. 2005;73(1):99-105.
168. Gunderson JG, Daversa MT, Grilo CM, et al. Predictors of two-year outcome for patients with borderline personality disorder. *Am J Psychiatry*. 2006;163(5):822-826.
169. Zanarini MC, Frankenburg FR, Reich DB, Fitzmaurice G. The 10-year course of psychosocial functioning among patients with borderline personality disorder and axis II comparison subjects. *Acta Psychiatr Scand*. 2010;122(2):103-109.
170. Berenson KR, Downey G, Rafaeli E, Coifman KG, Paquin NL. The rejection-rage contingency in borderline personality disorder. *J Abnorm Psychol*. 2011;120(3):681-690.
171. Yeomans F. A BPD Teaching Supplement for the Clinical Community. Available at https://www.bpdaustralia.org/wp-content/uploads/BPD-Excellent-TeachingGuide.pdf. Last accessed February 6, 2020.
172. Buteau E, Dawkins K, Hoffman P. In their own words: Improving services and hopefulness for families dealing with BPD. *Social Work in Mental Health*. 2008;6(1-2):203-214.
173. Horn N, Johnstone L, Brooke S. Some service user perspectives on the diagnosis of borderline personality disorder. *J Ment Health*. 2007;16(2):255-269.
174. Raven C. Borderline personality disorder: still a diagnosis of exclusion? *Ment Health Today*. 2009;6:26-31.
175. Paris J. Memories of abuse in borderline patients: true or false? *Harv Rev Psychiatry*.1995;3(1):10-17.
176. Mead S. Trauma-Informed Peer Support. Available at https://healingattention.org/wp-content/uploads/Trauma-informed-Peer-Support.pdf. Last accessed February 6, 2020.
177. Clarkin JF, Levy KN, Lenzenweger MF, Kernberg OF. Evaluating three treatments for borderline personality disorder: a multiwave study. *Am J Psychiatry*. 2007;164(6):922-928.
178. Meyerson DA, Triebwasser J, Passarelli V, Siever LJ. Is Borderline Personality Disorder Underdiagnosed? Paper presented at: American Psychiatric Association Conference; May 2009; San Francisco, CA.
179. Stone M. *The Fate of Borderline Patients*. New York, NY: The Guilford Press; 1990.
180. Paris J. Why psychiatrists are reluctant to diagnose borderline personality disorder. *Psychiatry*. 2007;4(1):35-39.
181. Kernberg OF, Yeomans FE. Borderline personality disorder, bipolar disorder, depression, attention deficit/hyperactivity disorder, and narcissistic personality disorder: practical differential diagnosis. *Bull Menninger Clin*. 2013;77(1):1-22.
182. Paris J. The nature of borderline personality disorder: multiple dimensions, multiple symptoms, but one category. *J Personal Disord*. 2007;21(5):457-473.
183. Bateman A, Fonagy P. 8-year follow-up of patients treated for borderline personality disorder: mentalization-based treatment versus treatment as usual. *Am J Psychiatry*. 2008;165(5):631-638.
184. Linehan MM, Comtois KA, Murray AM, et al. Two-year randomized controlled trial and follow-up of dialectical behavior therapy vs therapy by experts for suicidal behaviors and borderline personality disorder. *Arch Gen Psychiatry*. 2006;63(7):757-766.
185. Young JE, Klosko J, Weishaar ME. *Schema Therapy: A Practitioner's Guide*. New York, NY: The Guildford Press; 2003.
186. Clarkin JF, Yeomans FE, Kernberg OF. *Psychotherapy of Borderline Personality: Focusing on Object Relations*. Washington, DC: American Psychiatric Press; 2006.
187. Chanen AM, Jovev M, McCutcheon LK, Mcgorry PD. Borderline personality disorder in young people and the prospects for prevention and early intervention. *Curr Psychiatry Rev*. 2008;4(1):48-57.
188. Wonderlich SA, Rosenfeldt S, Crosby RD, et al. The effects of childhood trauma on daily mood lability and comorbid psychopathology in bulimia nervosa. *J Trauma Stress*. 2007;20(1):77-87.
189. Miller AL, Muehlenkamp JJ, Jacobson CM. Fact or fiction: diagnosing borderline personality disorder in adolescents. *Clin Psychol Rev*. 2008;28(6):969-981.
190. Soloff PH, Cornelius J, George A, Nathan S, Perel JM, Ulrich RF. Efficacy of phenelzine and haloperidol in borderline personality disorder. *Arch Gen Psychiatry*. 1993;50(5):377-385.
191. Winograd G, Cohen P, Chen H. Adolescent borderline symptoms in the community: prognosis for functioning over 20 years. *J Child Psychol Psychiatry*. 2008;49(9):933-941.
192. Pack S, Wakeham S, Beeby R, Fawkes L, Yeandle J, Gordon C. Management of borderline personality disorder. *Nurs Times*. 2013;109(15):21-23.
193. Staebler K, Helbing E, Rosenbach C, Renneberg B. Rejection sensitivity and borderline personality disorder. *Clin Psychol Psychother*. 2011;18(4):275-283.
194. Hines DA. Borderline personality traits and intimate partner aggression: an international multisite, cross-gender analysis. *Psychology of Women Quarterly*. 2008;32(3):290-302.
195. Dutton DG. Male abusiveness in intimate relationships. *Clin Psychol Rev*. 1995;15(6):567-581.
196. Lieb K, Völlm B, Rücker G, Timmer A, Stoffers JM. Pharmacotherapy for borderline personality disorder: Cochrane systematic review of randomised trials. *Br J Psychiatry*. 2010;196:4-12.

197. Ripoll LH, Triebwasser J, Siever LJ. Evidence-based pharmacotherapy for personality disorders. *Int J Neuropsychopharmacol*. 2011;14(9):1257-1288.
198. Zimmerman M, Ruggero CJ, Chelminski I, Young D. Psychiatric diagnoses in patients previously overdiagnosed with bipolar disorder. *J Clin Psychiatry*. 2010;71(1):26-31.
199. Nelson K, Schulz SC. Pharmacologic treatment of borderline personality disorder. *Curr Psychiatry*. 2011;10(8):30-40.
200. McGrath PJ, Miller JM. Pharmacologic management for treatment-resistant unipolar depression. In: Tasman A, Kay J, Lieberman J, First MB, Riba M (eds). *Psychiatry*. 4th ed. Chichester: Wiley-Blackwell; 2015.
201. Ripoll LH. Psychopharmacologic treatment of borderline personality disorder. *Dialogues Clin Neurosci*. 2013;15(2):213-224.
202. Gunderson JG, Morey LC, Stout RL, et al. Major depressive disorder and borderline personality disorder revisited: longitudinal interactions. *J Clin Psychiatry*. 2004;65(8):1049-1056.
203. Fiedorowicz JG, Black DW. Borderline, bipolar or both? Frame your diagnosis on the patient history. *Curr Psychiatr*. 2010;9(1):21-30.
204. Stone MH. Relationship of borderline personality disorder and bipolar disorder. *Am J Psychiatry*. 2006;163(7):1126-1128.
205. Herman J. *Trauma and Recovery: The Aftermath of Violence—From Domestic Abuse to Political Terror*. New York, NY: Basic Books; 2015.
206. Auchincloss EL, Samberg E (eds). *Psychoanalytic Terms and Concepts*. New Haven, CT: American Psychoanalytic Association; 2012.
207. Koenigsberg HW, Kernberg OF, Stone MH, Appelbaum AH, Yeomans FE, Diamond D. *Borderline Patients: Extending the Limits of Treatability*. New York, NY: Basic Books; 2000.
208. Sansone RA, Sansone LA. Gender patterns in borderline personality disorder. *Innov Clin Neurosci*. 2011;8(5):16-20.
209. Swan SC, Snow DL. A typology of women's use of violence in intimate relationships. *Violence Against Women*. 2002;8(3):286-319.
210. Kernberg O. Borderline personality organization. *J Am Psychoanal Assoc*. 1967;15(3):641-685.
211. Kernberg O. The treatment of patients with borderline personality organization. *Int J Psychoanal*. 1968;49(4):600-619.
212. Kernberg OF. Structural change and its impediments. In: Hartocollis P (ed). *Borderline Personality Disorders*. 3rd ed. New York, NY: International Universities Press; 1977: 275-306.
213. Maltsberger JT, Buie DH. Countertransference hate in the treatment of suicidal patients. *Arch Gen Psychiatry*. 1974;30(5):625-633.
214. Gunderson JG. 2014 Lecture for the National Education Alliance for Borderline Personality Disorder. Available at https://www.borderlinepersonalitydisorder.org. Last accessed February 11, 2020.
215. Robins E, Guze SB. Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *Am J Psychiatry*. 1970;126(7):983-987.
216. Stiver IP. The meaning of care: reframing treatment models. In: Jordon JV, Kaplan AG, Miller JB, Stiver IP, Surrey JL (eds). *Women's Growth in Connection: Writings from the Stone Center*. New York, NY: Guilford Press; 1991.
217. Henry KA, Cohen CI. The role of labeling processes in diagnosing borderline personality disorder. *Am J Psychiatry*. 1983;140(11):1527-1529.
218. Herman JL, Perry JC, van der Kolk BA. Childhood trauma in borderline personality disorder. *Am J Psychiatry*. 1989;146(4):490-495.
219. Gunderson JG. *Borderline Personality Disorder*. Washington, DC: American Psychiatric Press; 1984.
220. Siever LJ, Davis KL. A psychobiological perspective on the personality disorders. *Am J Psychiatry*. 1991;148(12):1647-1658.
221. Linehan MM. *Cognitive-Behavioral Treatment of Borderline Personality Disorder*. New York, NY: The Guilford Press; 1993.
222. Gunderson JG, Links PS. *Borderline Personality Disorder: A Clinical Guide*. 2nd ed. Washington, DC: American Psychiatric Press, Inc.; 2009.
223. Oldham JM, Gabbard GO, Goin MK, et al. Practice Guideline for the Treatment of Borderline Personality Disorder. Available at https://psychiatryonline.org/pb/assets/raw/sitewide/practice_guidelines/guidelines/bpd.pdf. Last accessed February 11, 2020.
224. Manning S. Dealing with Unrelenting Crisis and Inhibited Grieving in Your Loved One with Borderline Personality Disorder. Available at http://docplayer.net/21816334-Dealing-with-unrelenting-crisis-and-inhibited-grieving-in-your-loved-one-with-borderline-personality-disorder.html. Last accessed February 12, 2020.
225. Larrivée M-P. Borderline personality disorder in adolescents: the *He-who-must-not-be-named* of psychiatry. *Dialogues Clin Neurosci*. 2013;15(2):171-179.
226. Soloff PH, Chiappetta L. Prospective predictors of suicidal behavior in borderline personality disorder at 6-year follow-up. *Am J Psychiatry*. 2012;169(5):484-490.
227. Soloff PH, Chiappetta L. Subtyping borderline personality disorder by suicidal behavior. *J Pers Disord*. 2012;26(3):468-480.
228. Wedig MM, Silverman MH, Frankenburg FR, Reich DB, Fitzmaurice G, Zanarini MC. Predictors of suicide attempts in patients with borderline personality disorder over 16 years of prospective follow-up. *Psychol Med*. 2012;42(11):2395-2404.
229. Paris J, Zweig-Frank H. A 27-year follow-up of patients with borderline personality disorder. *Compr Psychiatry*. 2001;42(6):482-487.

230. McMurran M, Cox WM, Coupe S, Whitham D, Hedges L. The addition of a goal-based motivational interview to standardized treatment as usual to reduce dropouts in a service for patients with personality disorder: a feasibility study. *Trials*. 2010;11:98.
231. Sansone RA, McLean JS, Wiederman MW. The relationship between medically self-sabotaging behaviors and borderline personality disorder among psychiatric inpatients. *Prim Care Companion J Clin Psychiatry*. 2008;10(6):448-452.
232. Nelson KJ, Schulz SC. Treatment advances in borderline personality disorder. *Psychiatric Annals*. 2012;42(2):59-64.
233. Gregory RJ, Jindal S. Factitious disorder on an inpatient psychiatry ward. *Am J Orthopsychiatry*. 2006;76(1):31-36.
234. Paris JL. *Treatment of Borderline Personality Disorder: A Guide to Evidence-Based Practice*. New York, NY: The Guilford Press; 2010.
235. Gregory RJ. Managing suicide risk in borderline personality disorder. *Psychiatric Times*. 2012;5.
236. Cowdry RW, Gardner DL. Pharmacotherapy of borderline personality disorder: alprazolam, carbamazepine, trifluoperazine, and tranylcypromine. *Arch Gen Psychiatry*. 1988;45(2):111-119.
237. Skodol AE, Grilo CM, Keyes KM, et al. Relationship of personality disorders to the course of major depressive disorder in a nationally representative sample. *Am J Psychiatry*. 2011;168(3):257-264.
238. Yen S, Shea MT, Sanislow CA, et al. Borderline personality disorder criteria associated with prospectively observed suicidal behavior. *Am J Psychiatry*. 2004;161(7):1296-1298.
239. Gregory RJ. Borderline attributions. *Am J Psychother*. 2007;61(2):131-147.
240. Cooper J, Kapur N, Webb R, et al. Suicide after deliberate self-harm: a 4-year cohort study. *Am J Psychiatry*. 2005;162(2):297-303.
241. de Groot ER, Verheul R, Trijsburg RW. An integrative perspective on psychotherapeutic treatments for borderline personality disorder. *J Pers Disord*. 2008;22(4):332-352.
242. Searight HR. Efficient counseling techniques for the primary care physician. *Prim Care*. 2007;34(3):551-570.
243. Behavioral Tech. What is Dialectical Behavior Therapy (DBT)? Available at https://behavioraltech.org/resources/faqs/dialectical-behavior-therapy-dbt. Last accessed February 12, 2020
244. Angstman KB, Rasmussen NH. Personality disorders: review and clinical application in daily practice. *Am Fam Physician*. 2011;84(11):1253-1260.
245. National Registry of Evidence-Based Programs and Practices. Systems Training for Emotional Predictability and Problem Solving (STEPPS). Available at http://legacy.nreppadmin.net/ViewIntervention.aspx?id=243. Last accessed February 12, 2020.
246. Britton PC. Motivational interviewing to address suicidal ideation. In: Arkowitz H, Miller WR, Rollnick S (eds). *Motivational Interviewing in the Treatment of Psychological Problems*. 2nd ed. New York, NY: Guilford Press; 2017: 193-218.
247. National Registry of Evidence-Based Programs and Practices. Dynamic Deconstructive Psychotherapy. Available at http://legacy.nreppadmin.net/ViewIntervention.aspx?id=240. Last accessed February 12, 2020.
248. Zanarini MC. Psychotherapy of borderline personality disorder. *Acta Psychiatr Scand*. 2009;120(5):373-377.
249. Bateman A, Fonagy P. Randomized controlled trial of outpatient mentalization-based treatment versus structured clinical management for borderline personality disorder. *Am J Psychiatry*. 2009;166(12):1355-1364.
250. Kernberg OF, Selzer MA, Koeningsberg HW, Carr AC, Applebaum AH. *Psychodynamic Psychotherapy of Borderline Patients*. New York, NY: Basic Books; 1989.
251. Stoffers JM, Völlm BA, Rücker G, Timmer A, Huband N, Lieb K. Psychological therapies for people with borderline personality disorder. *Cochrane Database Syst Rev*. 2012;(8):CD005652.
252. Neville C. Psychological therapies for borderline personality disorder. *Nurs Times*. 2014;110(4):25.
253. Neacsiu AD, Lungu A, Harned MS, Rizvi SL, Linehan MM. Impact of dialectical behavior therapy versus community treatment by experts on emotional experience, expression, and acceptance in borderline personality disorder. *Behav Res Ther*. 2014;53:47-54.
254. Biskin RS. Treatment of borderline personality disorder in youth. *J Can Acad Child Adolesc Psychiatry*. 2013;22(3):230-234.
255. Bateman AW, Krawitz R. *Borderline Personality Disorder: An Evidence-Based Guide for Generalist Mental Health Professionals*. Oxford: Oxford University Press; 2013.
256. Chanen AM, Jackson HJ, McCutcheon LK, et al. Early intervention for adolescents with borderline personality disorder: quasi-experimental comparison with treatment as usual. *Aust N Z J Psychiatry*. 2009;43(5):397-408.
257. Appelbaum AH. Supportive psychotherapy. *FOCUS*. 2005;3(3):438-460.

Evidence-Based Practice Recommendations Citations

National Health and Medical Research Council. *Clinical Practice Guideline for the Management of Borderline Personality Disorder*. Melbourne: National Health and Medical Research Council; 2012. Available at https://bpdfoundation.org.au/images/mh25_borderline_personality_guideline.pdf. Last accessed March 24, 2020.

Assessment and Management of Risk for Suicide Working Group. *VA/DoD Clinical Practice Guideline for Assessment and Management of Patients at Risk for Suicide*. Washington, DC: Department of Veterans Affairs, Department of Defense; 2019. Available at https://www.healthquality.va.gov/guidelines/MH/srb. Last accessed March 24, 2020.