Bipolar Disorder in CHILDREN

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ABSTRACT
This article presents an overview of bipolar disorder (BPD) in children, a condition that only recently has been recognized as a legitimate diagnosis. Bipolar disorder in children is underrecognized for many reasons including lack of awareness, diagnostic confusion, and the different clinical picture in children. Available data strongly suggest that prepubertal childhood BPD is a
non-episodic, chronic, rapid cycling, mixed manic state. It may be comorbid with attention-deficit/hyperactivity disorder (ADHD) and conduct disorder (CD) or it may demonstrate features of ADHD and CD, further complicating recognition and subsequent treatment. Treatment issues are discussed, and some reasons for the urgency of early recognition and treatment are explained.
Just as the existence of childhood depression was denied for many years, a similar phenomenon has occurred in the debate about the existence of childhood bipolar disorder (BPD). Evidence for the existence of this serious affective disorder continues to accumulate, and it is only in the past decade that the psychiatric community has begun to accept that BPD can occur in children. However, because it appears in a very different way than in adults, controversy persists.

It is a controversy rooted in the way knowledge has developed and discovery has occurred in the field of psychiatry. To date, no articles have appeared in the nursing literature discussing the phenomenon of BPD in children. Therefore, the purposes of this article are to provide some of the background to the controversy, elaborate on the difficulties of diagnosis, present the current state of what is known about this condition, and alert clinicians to the importance of early identification and appropriate intervention.

**BACKGROUND**

The 1930s in the United States were dominated by the psychoanalytic school of thought. Psychodynamic theory viewed depression in adults as resulting from an intrapsychic conflict between the ego and the persecutory superego. Traditional psychodynamic theory viewed depression in children as an impossibility because of the lack of a sufficiently developed superego to direct aggression inward against the self. This view was accepted widely despite numerous reported clinical descriptions of children who seemed to be suffering from severe major depression. Only in 1975 was childhood depression officially accepted as a legitimate phenomenon at the National Institute of Mental Health Conference on Depression in Childhood (Goodwin & Jamison, 1990).

Similarly, clinicians and scholars have debated about whether children could experience mania, and as a result, this disorder may have been overlooked for many years as a legitimate differential when evaluating children for the presence of mental illness. Denial persisted for many years despite such historical luminaries as Kraepelin (1921) reporting that 4% of people with manic-depressive illness first exhibited symptoms prior to the onset of puberty and that the onset of first episodes of mania increased significantly after puberty.

This situation persisted until large-scale studies of adults with BPD indicated that approximately 20% of them showed symptoms prior to age 19 (Carlson, Davenport, & Jamison, 1977; Winokur, Clayton, & Reich, 1969). Later studies confirming these early results gave further legitimacy to the idea that BPD was possible in childhood and that it had been unrecognized, underrecognized, and misdiagnosed for many years (Carlson, Fennig, & Bromet, 1994; Joyce, 1984). Despite general agreement that childhood BPD is a more common disorder than previously thought, textbooks in psychiatry and nursing still pay scant attention to BPD in children.

**The Diagnostic Conundrum**

Even with heightened clinician awareness of BPD in children, the diagnosis is not made easily. Certainly the fact that children are always developing is one reason for the difficulties encountered in the diagnosis of all mental illnesses in children (Bowers, 1998; Mohr, 1999).

However, a large part of the problem is related to the inadequacy of the present system of classifying mental illnesses using the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (DSM-IV) (American Psychiatric Association [APA], 1994). Part of the problem is in the diagnostic categories themselves, which are downward extensions of adult categories and perhaps do not even represent a true picture of certain disorders as they are manifested in children (Mohr & Regan-Kubinski, 1999). Much of the extrapolation from the adult literature is due to the limitations and paucity of research conducted specifically on children (American Academy of Child and Adolescent Psychiatry [AACAP], 1997).

Another factor that adds to diagnostic confusion is comorbidity. Childhood disorders, under the DSM-IV classification system, exhibit an enormous amount of comorbidity. Comorbidity refers to the manifestation of two or more disorders whose co-occurrence is greater than what would be expected by chance alone. Comorbidity of disorders has been reported to be as high as 50% in some community samples and even higher in some clinical samples (Caron & Rutter, 1991). Some of the more common child and adolescent disorders said to exhibit comorbidity are (Mash & Dozois, 1996):

- Conduct disorder (CD) and attention-deficit/hyperactivity disorder (ADHD).
- Childhood depression and anxiety.
- Autistic disorder and mental retardation.
- Tourette's syndrome and ADHD.
Childhood BPD seems to be comorbid with ADHD (Bowers, 1998; Milberger, Biederman, Faraon, Murphy, & Tsuang, 1995; Paplos & Paplos, 1999). Debate continues regarding the definition and nature of comorbidity, and it is beyond the scope of this article to elaborate on this debate. However, scholars seem to agree that comorbidity may result from ambiguity in prevailing definitions of dysfunction and that perhaps some disorders do indeed cluster together (Kazdin & Kagan, 1994). Regardless of the reasons for the comorbidity phenomenon, there is general agreement that clinicians cannot rely exclusively on DSM-IV criteria to capture nuances in individual situations (Mohr & Regan-Kubinski, 1999).

**Epidemiology**

In contradistinction to the number of epidemiological studies conducted on depression, unfortunately only one study has been conducted to determine the incidence or prevalence of BPD in juvenile samples (Lewinsohn, Klein, & Seeley, 1995). Studying a community sample of adolescents ages 14 to 18 (N = 1,709), Lewinsohn et al. (1995) found a lifetime prevalence of BPD (primarily bipolar II disorder and cyclothymia) of approximately 1%. In retrospective studies that examined the onset of BPD in adult patients, 5% of the sample reported onset to have occurred between ages 5 and 9, and 7.5% reported onset between ages 10 and 14 (Loranger & Leivene, 1978).

Bipolar disorder seems to be less prevalent than depression in children. However, prevalence rates reported within restricted samples, such as inpatient units, challenge the previously accepted wisdom that mania is rare or nonexistent in children. Weller, Weller, Tucker, and Fristad (1986) estimated the rate of mania at 22% among severely disturbed youngsters. Estimates of prevalence from inpatient samples are complicated by underdiagnosis of BPD (Geller, 1996; Isaac, 1995). Therefore, although scant, the data are beginning to suggest that BPD in children may not be an uncommon phenomenon.

**CLINICAL PICTURE OF CHILDHOOD BPD**

As mentioned above, young children and adolescents demonstrate a different clinical picture than adults, partly due to differing developmental levels. This section provides a general overview of how BPD is manifested in the juvenile population. Additional detail is presented in the Table that contains a proposed new definition of juvenile-onset BPD (Paplos & Paplos, 1999).

The course of BPD in children has not been well studied, but authors report that it may emerge as a depression in children with no previous disturbance (Geller & Luby, 1997). On rare occasions a manic episode may precede a depressive episode. Children younger than age 9 exhibit more irritability and emotional lability, whereas children older than age 9 exhibit more classic euphoria, elation, paranoia, and grandiosity. Clinicians may find it difficult to elicit classic, discrete episodes of cycling because the symptoms appear to be highly chronic.

Parents report that their children with BPD manifest hypomanic symptoms as early as in utero. Mothers report infants as being overly alert, irritable, colicky, and requiring very little
Because children with bipolar disorder are impulsive, overly active, and have poor attention spans, as well as low frustration tolerance, they may exhibit poor school performance.

Children with BPD often exhibit marked disruptive behaviors, extreme moodiness, and difficulty falling asleep at night. As preschoolers, they may exhibit hyperactivity, excessive cheerfulness, inappropriate silliness, giddiness, and elation. As they age, they may exhibit hostility and anger that can culminate in intense explosive rages that may take several hours to deescalate (i.e., affective storms). Parents report that children may make homicidal threats and even attack the parents. These rages may or may not be in response to any identifiable stimulus. Because children with BPD are impulsive, overly active, and have poor attention spans, as well as low frustration tolerance, they may exhibit poor school performance (Bowers, 1998; Carlson & Weintraub, 1993; Lewinsohn et al., 1995).

Clinicians may be able to detect signs such as:
- Hypersexuality.
- Pressured, rapid speech.
- Flight of ideas.
- Delusional thinking.
- Grandiosity of thought.

At times clinicians also may be able to detect auditory or visual hallucinations. In taking histories, clinicians may receive reports from parents describing severe, aggressive behaviors directed at them, siblings, peers, or pets (Bowers, 1998; Woźniak et al., 1995).

Adolescents may manifest a complicated clinical picture characterized by psychotic symptoms that include mood-incongruent hallucinations, delusions of persecution or paranoia, and other features of idiosyncratic thinking. Their mood may be exceedingly labile, with rapid shifting or mixed manic and depressive features, and their behavior may be markedly deteriorated from baseline functioning (Akiskal et al., 1985; Ballenger, Reus, & Post, 1982; Carlson & Kashani, 1988).

**Clinical Course**

As described above, parents are the first to recognize that "something is not right" with their children. When children become sufficiently disruptive in their behavior and affective dysregulation, they first come to the attention of health care providers. However, there may be a 5-year to 10-year lag between onset of symptoms and display of the disorder serious enough to be recognized and require treatment (AACAP, 1997). Very early onset BPD has not been studied sufficiently to justify any conclusions about its course in these children. However, Papulos and Papulos (1999) have posited a chronological developmental progression of symptoms that can be traced to infancy.

The early course of BPD in adolescents is often chronic and refractory to treatment (Carlson, 1990). However, the long-term prognosis appears similar to that of adults (McClellan, Werry, & Ham, 1993; Werry, McClellan, & Chard, 1991). Twenty-five percent to 50% of patients with BPD attempt suicide, and as many as 40% of manic episodes may be characterized by overt acts of physical violence.

**Other Disorders in the BPD Spectrum**

According to the DSM-IV, cyclothymia is a condition in the BPD spectrum, but the fluctuations of affective states, both depressed and elevated, are considerably less intense than those that appear in BPD proper (APA, 1994). In addition to

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cyclothymia, the DSM-IV describes bipolar II disorder, which stipulates a history of at least one hypomanic episode and one major depressive episode in the absence of a full mania.

Comorbidity With Other Childhood Disorders
As mentioned above, BPD and ADHD share many similar features including distractibility, inattention, impulsivity, and hyperactivity (Milberger et al., 1995). However, the distinguishing features of BPD include psychosis, aggression, excitability, affective lability, depression, and inappropriate affect. Therefore, Biederman, Klein, Pine, and Klein (1998) suggested that the features that cause diagnostic ambiguity are primarily nonmood symptoms.

Conduct disorder also seems to overlap on some criteria such as impulsivity, substance abuse, aggressiveness, and problems with the law (Kovacs & Pollock, 1995). However, in BPD, when these behaviors occur, they occur in conjunction with elevated or irritable mood. Again, mood is the distinguishing feature of BPD.

TREATMENT
Children suspected of having a mood disorder should be evaluated by a child psychiatrist. Mood disorders in children are understudied, and especially in the case of BPD, it is important that the children and their families receive the care of someone who is familiar with the current research and practice parameters, and who has experience working with children in the context of a family system. The family as partner in the care of their child is key to success in any interventions involving children, but even more so when the illness is this chronic and serious (Anderson, in press).

Central to treatment planning and care of children in all health care settings is an understanding of where they are in their trajectory of development. In addition to developmental stage, health care providers should understand the dynamics of the contexts within which children have achieved stage-salient competencies (Mohr, 1999).

These systems continue to influence the children in a complex series of transactions throughout their lives. This implies that both developmental and contextual approaches to mental health problems should be the foundation of any planning and that this planning must include different disciplines working with the family in a coordinated effort to meet the child’s needs (Anderson, in press). Many such individualized, multisystemic programming initiatives exist, and they soon may be the standard of care with children who have serious emotional disturbances and their families (Anderson, in press).

Generally, treatment consists of a combination of interventions, employing family education, family therapy, independent education plans (IEP) with the assistance of school authorities, and referral to appropriate support groups (e.g., National Alliance for the Mentally Ill, Federation of Families for Children’s Mental Health, Parents of Bipolar Children). Individual psychotherapy may help children express their feelings and develop ways of coping with their illness. In addition, some medications have been found to be useful in the treatment of early onset BPD. The three most common mood stabilizers are:

- Lithium.
- Valproate (Depakote).
- Carbamazepine (Tegretol).
All three appear to have antimanic properties, and they have similar prophylactic antimanic and antidepressant effects (Bowers, 1998). A brief description of these three medications follows. Nurses who care for children with BPD are urged to become familiar in depth with each of these medications, their dosing kinetics, side effects, and drug interactions.

Lithium
Lithium has been the medication of choice for the treatment of BPD for many years. Lithium may be most effective in patients whose first episode is representative of mania, with response rates in adults being 80%, compared with a 20% response rate in patients whose first episode was a depression (Bowers, 1998; Hyman, 2000).

Despite voluminous data on its efficacy in the treatment of adult patients, little data exist that address the efficacy of lithium for early onset BPD. Some studies, including individual case reports, have found lithium to be effective in children (Sachs, Pritzl, Kahn, Carpenter, & Docherty, 2000), although the therapeutic response is not as great as for adults. The reason may be that children, particularly adolescents, seem to have mixed manic-depressive syndromes and are more likely to have psychotic features, both of which are more refractory to treatment (AACAP, 1997).

Valproate and Carbamazepine
Anticonvulsant mood stabilizers, such as carbamazepine and valproate, also have been found to be useful in the acute treatment of BPD. Clinicians
are using these medications more frequently because of their more acceptable side effect profiles. Carbamazepine has been reported to be effective in adolescents who are not responsive to lithium therapy (Hsu, 1986). Moreover, both carbamazepine and valproate seem to be more effective than lithium in treating patients with rapidly cycling, dysphoric, or mixed mania, as well as patients with comorbid substance abuse (Bowers, 1998).

Preliminary data from a multicenter study showed that 62% of 40 outpatients between ages 7 and 19 improved on valproate (Strober, Morrell, Lampert, & Burroughs, 1990). In another study, 42 children ages 8 to 18 were randomized to lithium, valproate, or carbamazepine. In the lithium and carbamazepine groups, the response rate was 38%, while in the valproate group, the response rate was 53% (Cepelak, Zanic-Grubisic, Mandusic, Repik, & Lenicek, 1998).

New Anticonvulsants
Some of the newer anticonvulsants such as lamotrigine (Lamictal) and gabapentin (Neurontin) also show promise in treating patients with BPD, but investigators urge caution in prescribing them because their antiepileptic efficacy does not equate per se to antimanic efficacy (Calabrese et al., 1999). Moreover, studies suggest that gabapentin is no more effective than placebo in acutely manic patients (Bowden, 2000) and that lamotrigine is associated with a 1% chance of children developing Stevens-Johnson syndrome (Papolos & Papolos, 1999).

Antipsychotic Medications
Some patients with BPD may require the addition of an antipsychotic medication during the acute phases of their illness because more than half of patients with BPD have psychotic features (Akisakal, 2000). Although no studies have examined the efficacy of neuroleptic medications for the treatment of early onset BPD, they are commonly used in clinical practice (AACAP, 1996). The newer neuroleptic medications such as risperidone (Risperdal) or olanzapine (Zyprexa) can help restore sleep patterns and reduce severely psychotic signs and symptoms, while at the same time lessening the chance of acute and delayed extrapyramidal adverse effects. In empirical studies, olanzapine has been shown to be superior to placebo for the treatment of mania, resulting in United States Food and Drug Administration’s approval of olanzapine for the treatment of mania (Bowden, 2000).

Benzodiazepines
Benzodiazepines have been found to be useful for treating manic states. When used in conjunction with antimanic agents and in place of neuroleptic medications, they can be helpful for psychomotor agitation, irritability, and insomnia in acute mania (Vieselman, Ylayyan, Weller, & Weller, 1993). Clonazepam (Klonopin) and lorazepam (Alznapam) are used most often in adults, but no literature exists on their use in children and adolescents with acute mania (Werry & Aman, 1993).

IMPORTANCE OF EARLY RECOGNITION AND INTERVENTION
The literature suggests that patients who have prior episodes of the disorder may have a poorer response to lithium and that a patient’s past history in terms of episodes and mood instability makes a difference in treatment response (Swann, Bowden, Calabrese, Dilsaver, & Morris, 1999). The implications of this are clear—there is a need for prevention and early detection. Scientists believe variable responses to medications may result from a form of kindling and sensitization, and that this kindling can happen spontaneously as a natural progression of the illness and its cycles. There is also compelling evidence that it may be induced by medications such as tricyclic antidepressants and stimulants (Post & Weiss, 1998). The following sections provide a brief description of this process.

Neuroplasticity, Sensitization, and the Kindling Effect
Neuroplasticity is a lifelong process that mediates the structural and functional reaction of the central nervous system to new experiences, attrition, and injuries. Manifestations of neuroplasticity include phenomena such as axonal sprouting, neurite extension, synaptogenesis, and neurogenesis (Mesulam, 2000). The process of neuroplasticity plays an important role in response to neuronal injury. Injury to neurons or neuronal death may lead to compensatory activity in which dendrites branch to accommodate synaptic inputs that have lost their original targets.

Kindling was the first neuroplasticity phenomenon suggested to be useful for studying memory processes and learning (Goddard & Douglas, 1975). Kindling is a form of sensitization, taking the name from the idea that a tiny spark will lead to a roaring fire. In similar fashion, a tiny electrical stimulus to neurons, repeated over and over,
A PROPOSED DEFINITION OF JUVENILE ONSET BIPOLAR DISORDER

1. Marked variations in mood and energy characterized by rapid, wide swings of emotion, levels of arousal, excitability, and motor activity.
   a. Hypomanic or manic episodes vary in duration and severity and may be associated with:
      - Elated (i.e., silly, giddy), euphoric, or irritable mood states.
      - Elevated sense of self-esteem.
      - Rapid or pressured speech.
      - Racing thoughts.
      - Flights of ideas.
      - Diminished need for sleep.
      - Increased energy and activity.
      - Decreased appetite, sometimes with weight loss.
   b. Depressive episodes vary in duration and severity and most commonly are associated with:
      - Melancholia or loss of interest in previously enjoyed activities.
      - Decreased sense of self-esteem.
      - Decreased energy.
      - Psychomotor retardation.
      - Sweet and carbohydrate cravings, sometimes with weight gain.

2. A developmental progression of symptoms and behavioral characteristics that evolve from infancy in chronological sequence, sharing features of attention, anxiety, and conduct disorders.

3. Frequent association with multiple comorbid DSM-IV diagnoses including enuresis, encopresis, night terrors, separation anxiety, panic and phobic disorders, attention-deficit/hyperactivity disorder, obsessive-compulsive disorder, oppositional defiant disorder, conduct disorder, major depression, bulimia, and post-traumatic stress disorder, as well as substance abuse and borderline personality disorder in adolescence.

4. A low threshold for arousal and a corresponding tendency toward a heightened startle response, high anxiety levels (e.g., morbid fears of separation and loss), panic or irritable mood states, and sleep/wake cycle dysregulation.
   a. Tendency to become overaroused, anxious, and fearful when exposed to novel, unwanted, or unexpected sensory stimulation.
   b. Tendency to react with excessive anxiety and fearfulness to specific social stressors, particularly loss and separation, or the anticipation of loss and separation, and transitions from one context to another.
   c. Sleep/wake cycle disturbances, night terrors, frightening nightmares, and other parasomnias, with variability in duration of sleep depending on phase of sleep.

5. A low threshold for frustration tolerance in situations that require sustained attention and effort, as well as postponement of immediate gratification. Disturbances in regulation of aggressive and sexual impulses.
   a. Episodes of anger dyscontrol or temper tantrums, usually of more than 30 minutes in duration, spontaneously or situationally precipitated. These may be accompanied by profanity and physical violence.
   b. Heightened sexual interest and curiosity, as well as precocious sexual behavior, often accompanied by inappropriate public displays.

6. Disturbances in the regulation of reward systems that predispose to poor regulation of self-esteem (e.g., grandiosity, worthlessness) and appetite, as well as substance abuse and addictive disorders.

7. Overreactive stress response, hyperarousal, and heightened sensitivity to real or imagined stressors, leading to avoidant or oppositional behavior.

8. Disturbances in central nervous system functions that predispose to cognitive and motor excesses, such as obsessive or ruminative thinking, repetitive and stereotypic movements, and impulsive or compulsive activities.

9. Disturbances in central nervous system functions that predispose to subserve various cognitive functions (e.g., attention, concentration, behavioral organization, strategic planning, accessibility of short-term memory). Associated features include distractibility, daydreaming, school performance deficits, difficulty in priority setting and organizational skills, and frequent unintentional loss of personal belongings.

10. Increased likelihood of occurrence of cycles in response to circannual variations in light and dark and ambient temperature, as well as in response to menstrual cycles in girls.

11. Tendency toward cycle induction when exposed to antidepressants, stimulants, and steroids.

12. Commonly associated with a bilineal family history of mood disorders or mood disorders and alcoholism.

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eventually will lead to full-blown activity.

During experiments by Goddard (1967) on electrical stimulation of the amygdaloid complex and the effect on learning, he noted that rats developed seizure activity after repeated stimulation. He hypothesized that their brains actually were changing in response to continual stimulation, and in his later work, he demonstrated that this same effect could occur as a result of chemical stimulation (Goddard, McIntyre, & Leech, 1969). He recognized that this change constituted a form of plasticity.

Understanding plasticity is important for at least two reasons. If a repeated application of electrical stimulation applied to the brain at low levels causes sufficient change such that full-blown seizures occur when the same electrical stimulus is applied later, it may help explain certain phenomena, such as the progression of BPD in the absence of intervention. Another reason for clinical awareness of sensitization and kindling is that studies of BPD suggest that the kindling phenomenon may be responsible for difficulties encountered by clinicians in patients' treatment.

Post and Weiss (1996, 1998) hypothesized that kindling was responsible for what clinicians saw as a loss of response to pharmacological treatments. Kindling from repeated stimulation due to unchecked episodes of affective illness constituted a sensitization of neuronal pathways in the central nervous system. After these pathways became sensitized, they would be more resistant to pharmacotherapeutics.

Post and Weiss (1996, 1998) extended their hypothesis to consider how similar principles in different neurochemical systems could account for the increase in frequency and duration of episodes of affective illnesses and their progression, cyclicity, and drug tolerance. The kindling model also may help explain difficulties in understanding chronic affective disorders regarding how variability in episodic recurrence and cycling patterns occurs within and among individuals (Post & Weiss, 1996).

**Clinical Implications of Kindling**

The phenomenon of kindling addresses the complexity of affective disorders and has critical implications for early recognition and intervention by clinicians. One of these implications is that if the condition remains unrecognized and untreated, future episodes of illness may occur independent of any stimulus, and they will occur with increasing frequency (Papolos & Papolos, 1999). Given the reality that there is a lag between the onset of symptoms and when they come to the attention of health care providers, the real danger of unrecognized cycling and subsequent unchecked neuronal sensitization emphasizes the urgency of early identification.

Another implication of the kindling effect has to do with conceptual confusion surrounding the phenomenon itself. As mentioned above, BPD appears to be comorbid with ADHD. Both stimulants and antidepressants are used in the treatment of ADHD, and both may be implicated in the precipitation of mania in genetically vulnerable children (Geller, Fox, & Fletcher, 1993; Papolos & Papolos, 1999). In one study, Altshuler et al. (1995) found that 35% of patients with BPD had a manic episode rated as likely to have
been antidepressant induced. Moreover, an acceleration of cycling was associated with antidepressant treatment in 26% of patients, and younger age at first treatment was a predictor of vulnerability to antidepressant induced cycle acceleration (Altshuler et al., 1995).

Given the current diagnostic confusion, it is imperative that clinicians take a detailed, thorough, longitudinal history before prescribing medications. A rush to judgment can have unintended consequences in that medications that are appropriate for the treatment of ADHD may worsen the course and prognosis of BPD.

CONCLUSION

In this article, a truncated overview of a disorder that has been and continues to be a complex issue in childhood psychopathology has been provided. Available data strongly suggest that prepubertal childhood BPD is a non-episodic, chronic, rapid cycling, mixed manic state—a rather different presentation than its adult counterpart. It may be comorbid with ADHD and CD, or it may demonstrate features of ADHD and CD, further complicating recognition and subsequent treatment. Likewise, the clinical picture in adolescents is different, with more adult-like symptoms present, but with cycles still difficult to elicit.

Despite the need for early detection, some studies indicate that often there are many years between the time patients experience symptoms of BPD and it being diagnosed. Childhood BPD has not yet been accepted as a diagnosis for inclusion in the DSM-IV, and some scholars continued to question the validity of the phenomenon well into the mid 1990s (Bowers, 1998).

Despite compelling early evidence for the existence of childhood BPD and the responsivity of manic children to mood stabilizers as late as 1996, scholars were having difficulty publishing their data in major journals (Biederman et al., 1998).

The ongoing debate about the manifestations childhood BPD and its complexities and permutations are daunting. This ongoing debate, the revolution in the understanding of molecular neurobiology, and the rapidity with which knowledge in the field changes presents a significant challenge to nurses. It underscores the crucial need for continuing self-education and suggests the moral implications inherent in maintaining a knowledge base that changes by the nanosecond.

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KEYPOINTS

1. Bipolar disorder (BPD) now is known to occur in children as young as toddlers. It is highly comorbid with conduct disorder, attention-deficit/hyperactivity disorder, Tourette's syndrome, depression, and anxiety disorders.

2. Childhood BPD generally emerges as a depression, but mania may precede depression in rare cases. The clinical picture of mania in children younger than age 9 is one of irritability and emotional lability. Children older than age 9 exhibit more classic euphoria, elation, paranoia, and grandiosity.

3. A hallmark of childhood BPD is intense rage. Children may exhibit seemingly unprovoked rage episodes that can last up to 2 to 3 hours.

4. Children with unrecognized BPD, who have been medicated inappropriately with stimulants or antidepressants, may experience treatment resistance.


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