



Pediatric Bipolar Disorder in an Era of “Mindless Psychiatry”

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Objective: Pediatric bipolar disorder (PBD) reflects shifts in conceptualizing bipolar disorder among children and adolescents since the mid-1990s. Since then, PBD diagnoses, predominantly in the United States, have increased dramatically, and the diagnosis has attracted significant controversy. During the same period, psychiatric theory and practice has become increasingly biological. The aim of this paper is to examine the rise of PBD in terms of wider systemic influences. Method: In the context of literature referring to paradigm shifts in psychiatry, we reviewed the psychiatric literature, media cases, and information made available by investigative committees and journalists. Results: Social historians and prominent psychiatrists describe a paradigm shift in psychiatry over recent decades: from an era of “brainless psychiatry,” when an emphasis on psychodynamic and family factors predominated to the exclusion of biological factors, to a current era of “mindless psychiatry” that emphasizes neurobiological explanations for emotional and behavioral problems with limited regard for contextual meaning. Associated with this has been a tendency within psychiatry and society to neglect trauma and attachment insecurity as etiological factors; the “atheoretical” (but by default biomedical) premise of the Diagnostic and Statistical Manual of Mental Disorders (3rd and 4th eds.); the influence of the pharmaceutical industry in research, continuing medical education, and direct-to-consumer advertising; and inequality in the U.S. health

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system that favors “diagnostic upcoding.” Harm from overmedicating children is now a cause of public concern. Conclusion: It can be argued that PBD as a widespread diagnosis, particularly in the United States, reflects multiple factors associated with a paradigm shift within psychiatry rather than recognition of a previously overlooked common disorder.

KEYWORDS affective disorders, attachment, behavioral disorders, behavioral medicine, emotion regulation, childhood trauma, professional attitudes, diagnostic validity, pediatric illness, DSM validity

BACKGROUND

It has long been accepted that bipolar disorder has its peak onset in late adolescence to young adulthood. It is also true that early episodes of hypomania can be difficult to diagnose. However, Biederman and colleagues (Wozniak et al., 1995) proposed that *most* cases of bipolar disorder have a preschool age onset and that irritability, not elevated mood, is the core feature. Such children were described as presenting “as irritable, with ‘affective storms’ or prolonged and aggressive temper outbursts” and with “chronic and continuous rather than episodic and acute” clinical course (Biederman et al., 1996, p. 998). In the same year, Geller and colleagues (1995), in another departure from traditional concepts of manic depressive illness, proposed that most cases of bipolar disorder in children still exhibited elevated mood but also featured ultradian mood cycles—several cycles of mania and depression per day. Geller and Luby (1997), in a review article in the *Journal of the American Academy of Child and Adolescent Psychiatry (JAACAP)*, stated,

Pre-pubertal onset manic depressive disorder . . . may present . . . with continuous, mixed manic, rapid-cycling of multiple brief episodes. . . . Thus, children may be having a laughing fit and happily doing arts and craft when, without any environmental prompt, they suddenly become miserable and acutely suicidal . . . parents describe their children rapidly cycle sometimes numerous times a day. (p. 1172)

Over the next decade these pediatric bipolar disorder (PBD) constructs gained acceptance in the United States. Another review article in *JAACAP* (Pavuluri, Birmaher, & Naylor, 2005) noted that the National Institute for Mental Health roundtable on pre-pubertal PBD, convened in April 2000, had termed the chronic irritable mood group *broad phenotype PBD* and the elevated mood group *narrow phenotype PBD*. When *JAACAP* published

“Treatment Guidelines for Children and Adolescents with Bipolar Disorder” (Kowatch et al., 2005), a commentary (McClellan, 2005) raised doubts about the diagnostic validity of PBD, but skeptical articles in the literature were few. Biederman (2006), although acknowledging the debate over the validity of PBD, asserted that the literature supported the diagnosis and that “up to 20% of psychiatrically referred children satisfy criteria for bipolar spectrum disorders” (p. 901).

However, follow-up studies have shown that non-episodic irritable broad phenotype PBD does not progress to adult bipolar disorder, and thus it has been relabeled *severe mood dysregulation* (Stringaris et al., 2010). This may have tempered the spread of PBD diagnoses; nonetheless, publications like the recent book *Is Your Child Bipolar?* (Wozniak & McDonnell, 2008), reviewed by Levin (2010), still propound the broad as well as narrow versions of PBD.

PBD was popularized to the public in the bestselling book *The Bipolar Child: The Definitive and Reassuring Guide to Childhood’s Most Misunderstood Disorder* (Papolos & Papolos, 2000) and as the cover story of *Time* magazine (Kluger & Song, 2002). Both the book and the article suggested that bipolar disorder could begin in utero. Advocacy groups like the Child and Adolescent Bipolar Foundation (www.bpchildren.com) and the Juvenile Bipolar Research Foundation (www.jbrf.org) provided parent education and an online diagnostic questionnaire.

Upon this background, diagnoses of bipolar disorder in children and youth increased 4,000% from 1994–1995 to 2002–2003 (Moreno et al., 2007), and by 2004 PBD had become the most common diagnosis in U.S. pre-pubertal psychiatric inpatient units (Blader & Carlson, 2007).

However, after 15 years PBD remains a contentious diagnosis. Its validity is questioned both academically (Frances, 2010; Parens & Johnson, 2010) and increasingly in the public media through stories of heavily medicated children and conflicts of interest involving researchers and the pharmaceutical industry.

Psychiatry is as much social science as a biomedical discipline, and its tenets are subject to influence by the prevailing paradigm. We believe the phenomenon of PBD as a new, commonly used diagnostic entity confined mainly to the United States is best comprehended from a broad systemic perspective. Such a perspective needs to explore beyond the PBD academic literature with its focus on symptom cluster analyses, neuroimaging, and medication responses to consider overarching paradigmatic shifts in psychiatry, particularly shifts in nosology and research methodology, individual and societal repression of trauma, the vagaries of managed care in the U.S. health system, and the influence of the pharmaceutical industry.

This article therefore takes a narrative approach. We acknowledge our skepticism, which is based on our clinical experience, reading of the literature and wider media, and communication with colleagues. Differences

in practice and training between the United States and other countries are factored in, with a focus on differences where we work—Australia (Peter Ignatius Parry) and the United States (Edmund C. Levin).

MEDIA CASES

The media have reported several cases of the overmedication of very young children featuring the PBD diagnosis. The story of Rebecca Riley, diagnosed at age 2 and deceased from a medication overdose at age 4, is widely known (CBS *60 Minutes*, 2007). Although Rebecca died after her parents allegedly gave extra clonidine plus a cough medicine, the autopsy report indicated that her regime of clonidine, quetiapine, and divalproex had caused “damage to her heart and lungs from prolonged abuse of these prescription drugs, rather than one incident” (Wen, 2007).

Another case involved Destiny Hager, diagnosed with PBD at age 3 and prescribed two antipsychotics concurrently: quetiapine, 600 mg/day; and ziprasidone, unspecified dose. He died of fecal impaction (Carpenter, 2009).

A 2008 cover story of *Newsweek* was of “Max,” a 10-year-old diagnosed and medicated around his second birthday. He was treated with 38 psychiatric drugs over the next 8 years (Carmichael, 2008). The *New York Times* recently highlighted the case of Kyle Warren, misdiagnosed with autism and PBD and treated with polypharmacy that commenced with an antipsychotic at 18 months of age. He experienced significant weight gain and loss of motivation (Wilson, 2010).

PARADIGM SHIFT FROM “BRAINLESS PSYCHIATRY” TO “MINDLESS PSYCHIATRY”

These cases signal a profound shift in the conceptualization and management of childhood emotional and behavioral problems. Such changes in practice imply a shift in the paradigm under which psychiatry is practiced. Kuhn (1962) proposed that science always proceeds in a social and historical context. The prevailing paradigm governs what is considered for study and treatment and what is not. Under the influence of a paradigm, even research of high intellect, internal consistency, and technical quality can lead to false conclusions.

Eisenberg (1986), head of the American Psychiatric Association’s section on child and adolescent psychiatry, coined the terms *brainless psychiatry* and *mindless psychiatry*. These describe the poles of the pendulum swing from the pre-*DSM-III* (*Diagnostic and Statistical Manual of Mental Disorders*, 3rd ed.) excesses of speculative psychoanalysis, overly zealous family therapy, and the anti-psychiatry movement to the excessive biological reductionism of the past two decades.

The *DSM-III*, published in 1980, was a key turning point, and the paradigm shift was under way by January 1990 when President George H. Bush declared the “decade of the brain.” Since then there have been significant advances in neuroimaging, neurochemistry, and genomics. However, *Homo sapiens* evolved as a social species, and the biopsychosocial model remains a more philosophically robust basis for the health sciences (Borrell-Carrió, Suchman, & Epstein, 2004).

Beginning two decades ago, there have been warning voices about biomedical reductionism. Silove (1990), in the *Australian and New Zealand Journal of Psychiatry*, quoted Eisenberg with his reference to mindless psychiatry and stated,

Australian psychiatry should consider the recent ideological shift in the USA to an extreme biological model of mental disorders . . . the field is at risk of being overwhelmed by a reductionist ‘biologism’ which assumes an organic causation for all abnormal human behaviour. (p. 461)

In 1989, Lipowski stated, “After a period marked by one-sided emphasis on psychodynamic and social issues, or what could be called ‘brainless’ psychiatry . . . we are witnessing an opposite trend towards extreme biologism or ‘mindless’ psychiatry” (p. 249). Tasman (1999) noted that economic forces have diminished psychodynamic training in the United States to the extent that “many fear we are in danger of training a generation of psychiatrists and physicians who lack . . . a framework for understanding mental functioning from a psychodynamic perspective” (p. 189). Boyce (2006), in an address to the Royal Australian and New Zealand College of Psychiatrists, blamed the “dumbing down” of psychiatry on “increased service demand, the deification of *DSM*, the influence of the pharmaceutical industry, a misunderstanding of evidence-based medicine (EBM), managerialism and the influence of consumerism” (p. 4). Commenting further on this paradigm shift, Scull (2010) noted, “A simplistic biological reductionism (has) increasingly ruled the psychiatric roost. Patients and their families learned to attribute mental illness to faulty brain biochemistry. . . . It was biobabble as deeply misleading and unscientific as the psychobabble it replaced” (p. 1247).

It appears to us that the common application of the PBD diagnosis reflects research and clinical practice that, consistent with the prevailing paradigm, underutilizes psychodynamics, family dynamics, attachment, trauma, and context. Frances (2010), the former *DSM-IV* task force chair, has gone so far as to critique PBD as a “fad diagnosis” of “epidemic” proportions.

Nonetheless, anecdotally it has been difficult for critics of PBD to publish in the psychiatric literature. In an era in which quantitative research is held in higher regard than qualitative research, it may be that contrary views about PBD are seen as opinion based and lacking data, reflecting a “catch 22”: Those who dispute the construct validity of PBD are unlikely to have generated data on something they don’t see.

One published exception in *JAACAP* was a commentary by McClellan (2005) to the Treatment Guidelines. McClellan bluntly stated, “Labelling tantrums as a major mental illness lacks face validity and undermines credibility in our profession” (p. 238). He also stressed the traditional basics of child psychiatry: “The developmental and family systemic context of children’s moods and behavior reflect complex problems interwoven with temperament, attachment, parent-child relationships, cognition and other moderating/mediating factors including trauma” (p. 237). He implied that this sophisticated biopsychosocial paradigm is lacking in the PBD literature.

NOT EVERYTHING THAT COUNTS CAN BE COUNTED

One aspect of this paradigm shift has been an emphasis on structured interviews and rating scales, which are necessary in research. However, this comes at the expense of introspection and reflection about the presenting phenomenology of patients in their life narrative and context. Carlson (1998), despite being among the first to raise the issue of pre-pubertal mania, critiqued the checklist approach to diagnosis in PBD research. Carlson and Meyer (2006) noted, “The diagnosis of bipolar disorder is often made by mindlessly applying criteria . . . without understanding developmental history and context” (p. 963) and went on to propose “that bipolar research could benefit from a developmental psychopathology approach” (p. 963).

It can be argued that the extensive PBD research literature reflects a current biomedical reductionist and taxonomic approach to the phenomenology of children’s and teenagers’ behavior. But even in physics the quantitative approach is not everything. Einstein, whose ideas came more from intuition than calculation, hung a plaque in his office at Princeton University that stated “Not everything that counts can be counted, and not everything that can be counted, counts” (“Albert Einstein,” 2008).

Biederman et al. (1995) have used subscales of the Child Behavior Checklist (CBCL) to define broad phenotype PBD or juvenile bipolar disorder (JBD)—hence “CBCL-JBD.” However, a 10-year follow-up of pre-pubertal children diagnosed by the CBCL-JBD was found to lack predictive validity into adolescence for bipolar disorder (Halperin, Rucklidge, Powers, Miller, & Newcorn, 2011). A diagnostic checklist from “The Bipolar Child” and accessible online at www.jbrf.org also was found to lack predictive capacity for bipolar disorder in a study that used it retrospectively (Rucklidge, 2008).

NEGLECT OF TRAUMA AND ATTACHMENT FACTORS

Blader and Carlson (2007) found that a disproportionate number of Afro-American children received the PBD diagnosis. J. Harris (2005), a child psychiatrist working on a preteen inpatient unit in Boston, noted that

many children diagnosed with PBD were in foster care and had attachment trauma histories.

Edmund C. Levin, dealing with children in a residential program on polypharmacy cocktails typical for treating PBD, found over a 2-year period that milligrams of psychotropic medications could be reduced by 80% while aggressive incident reports fell by 100%. The reductions became possible by tapering medications while addressing trauma, attachment, milieu, and other factors. Most of the children at admission had a diagnosis of mood disorder not otherwise specified with comorbid attention-deficit/hyperactivity disorder. None warranted those diagnoses at discharge. Developmental trauma disorder (DTD; van der Kolk & Courtois, 2005) was felt to better describe their presentations (Levin, 2009).

We are not advocating brainless psychiatry. Developmental trauma can predispose or precipitate those constitutionally vulnerable to major psychiatric disorders like schizophrenia and bipolar disorder into manifesting the illnesses, but the effects of trauma can also present as affective instability and other ego defenses that may superficially resemble psychotic or severe mood disorders. Dissociation as a defense against trauma can particularly lead to symptoms easily confused with hypomanic and psychotic states (Silberg & Dallam, 2009).

Biomedical research is leading to significant advances in understanding brain development in the context of a child's attachment relationships and the effects of attachment disruption and trauma (Schoore, 2002). Attachment theory is a bedrock concept of child psychiatry and the wider field of developmental psychology. However, a search of the PBD literature for reference to attachment theory finds almost no mention of it (Parry, 2010). There also is little mention of trauma and abuse. The Washington University in St. Louis group, who proposed what has since been termed narrow phenotype PBD, found no cases of posttraumatic stress disorder (PTSD) and only mentioned sexual abuse as a differential diagnostic consideration to "manic hypersexuality." Only 1% of their PBD cohort had a history of sexual abuse. This very low rate is at odds with the literature on child sexual abuse and is also low compared to a study (Rucklidge, 2006) of narrow phenotype PBD that used the same diagnostic methodology. This study found that more than 50% had a history of trauma and 21% met criteria for lifetime PTSD (10% trauma exposure, 0% PTSD among controls). The Harvard/Massachusetts General Hospital group, who proposed what has since been termed broad phenotype PBD, referenced Wozniak et al. (1999) to hypothesize that PTSD occurs secondary to PBD (i.e., a child who develops PBD early in childhood may create stressful situations by misbehaving). That may then lead to the child's being traumatized.

Herman (1992) posited that society is biased against the acknowledgment of trauma:

All the perpetrator asks is that the bystander do nothing. He appeals to the universal desire to see, hear, and speak no evil. The victim, on

the contrary, asks the bystander to share the burden of pain. The victim demands action, engagement, and remembering. (p. 7)

Thus, nuclear families and sole parents, struggling in a modern world of complex stressors that offers minimal extended family, tribe, or village-like support, are likely to be attracted to simple biomedical explanations for disturbed childhood emotions and behaviors—particularly as such diagnoses imply no blame or need for difficult changes to the modern family. There is also the allure of a quick biomedical fix for both families and health providers, particularly pediatricians and psychiatrists, for whom writing a prescription may bestow a sense of action and assistance.

Although we find little coverage of these issues in the PBD research literature, academics have debated in the public media. Pavuluri (Carey, 2007b) enunciated the benefits of the diagnosis: “These are kids that have rage, anger, bubbling emotions that are just intolerable for them, and it is good that this is finally being recognized as part of a single disorder” (i.e., PBD). However, van der Kolk, a psychiatrist prominent in PTSD research, said, “The (PBD) diagnosis is made with no understanding of the context of their life.” Carlson has added, “Bipolar is being over diagnosed in children, and the major downside is that people then think they have a solution and are not amenable to listening to alternatives (which may not include drugs)” (Carey, 2007a). Williams (2008) critiqued PBD from a systemic perspective and described a 10-year-old boy erroneously diagnosed with PBD who was concurrently on eight psychotropics.

“DIAGNOSTIC UPCODING” IN THE U.S. HEALTH SYSTEM

Thus far, PBD has been a diagnosis mainly confined to the United States. Illustrating this are differences at various child and adolescent psychiatry conferences. In 2009 at the American Academy of Child and Adolescent Psychiatry (AACAP) conference in Hawaii there were at least 40 presentations on PBD and a further half dozen in a session chaired by Carlson about severe mood dysregulation as an alternative description for broad phenotype PBD. In contrast, there were *zero* presentations on PBD at both the 2009 Australian and New Zealand Child and Adolescent Psychiatry (CAP) conference in New Zealand and the larger European Society of CAP conference in Hungary. Furthermore, the British National Institute for Health and Clinical Excellence (2006) guidelines on bipolar disorder specifically recommend against using the PBD diagnosis in clinical practice. A German survey of child psychiatrists (Meyer, Koßmann-Böhm, & Schlotke, 2004) found that only 8% had ever seen a pre-pubertal bipolar disorder case.

Why is this so? One reason may be that the United States is one of the few nations to allow direct-to-consumer advertising. Psychotropics and

bipolar disorder have featured prominently in such advertising (Healy, 2006). Although the global media and Internet allow practitioners and parents to hear of PBD, still the diagnosis has not erupted as in the United States. Aspects of the U.S. health system appear to induce diagnostic upcoding pressures that drive a higher rate of bipolar disorder diagnoses. Diagnosis upcoding occurs wherever medical practitioners are under pressure to give a diagnostic label in order to provide treatment and be reimbursed.

Parry, Furber, and Allison (2009) surveyed Australian and New Zealand child psychiatrists about PBD. The survey noted that 90% thought PBD was “over-diagnosed” in the United States, 6% were “unsure,” and only 3.5% thought it was “under-diagnosed” or “appropriately diagnosed” by American colleagues. In discussion, U.S. colleagues noted how health insurers may demand a diagnosis like bipolar before providing reimbursement. Blader and Carlson (2007) postulated diagnosis upcoding as a reason for the increase in PBD. In light of such pressures, Eist (1999), former president of the American Psychiatric Association, called the U.S. managed care health system “corpicare,” as the system primarily serves the profit interests of private insurers. In particular, corpicare has tended to disadvantage the provision of psychotherapies more so than pharmacotherapy.

In Australia, diagnosis upcoding has emerged with Asperger’s disorder with children inappropriately labeled because the diagnosis confers educational and family financial welfare assistance (Basu, 2010). But because it is based on clinical need, Australia’s universal single payer health system does not require diagnoses for reimbursement for therapy and thus does not encourage a PBD epidemic.

INFLUENCE OF THE PHARMACEUTICAL INDUSTRY

Carlson alluded to causes other than upcoding for the PBD epidemic (Carey, 2007b): “We are just inundated with stuff from drug companies, publications, throwaways, that tell us six ways from Sunday that, Oh my God, we’re missing bipolar.” Scull (2010) noted that the rise of “biobabble” makes priceless “marketing copy” and that “drug money has come to dominate psychiatry. It underwrites psychiatric journals and psychiatric conferences (where the omnipresence of pharmaceutical loot startles the naive outsider)” (p. 1247).

Investigations by Senator Charles Grassley, Chair of the Senate Finance Committee, question the relationships between the pharmaceutical industry and some academic psychiatry departments (Grassley, 2008). Internal industry documents indicate that companies seek a wider bipolar diagnosis to boost sales of antipsychotics. Analysis of these documents (Spielmans & Parry, 2010) leads to the view that much psychiatric literature and continuing medical education would be better described as promoting “marketing-based medicine” rather than “evidence-based medicine.” This problem

has been described by former chief-editors of the *New England Journal of Medicine* in “Industry-Sponsored Clinical Research: A Broken System” (Angell, 2008) and of the *British Medical Journal* in “Medical Journals Are an Extension of the Marketing Arm of Pharmaceutical Companies” (Smith, 2005).

Some pharmaceutical company documents (Spielmanns & Parry, 2010) detail how, with the expiration of patents for many antidepressants in the past decade, new markets have been required to meet commercial needs. With most so-called second-generation antipsychotics (SGAs) still on patent, there has been interest in a wider bipolar diagnosis and a rebranding of SGAs as “mood stabilizers.” Researchers with theories that converged with industry goals were more likely to get financial support. There is nothing intrinsically wrong with this if evidence-based medicine is truly adhered to. But such influence can promote positions that benefit industry financially.

The Grassley Committee, the *New York Times*, and the *Wall Street Journal* in their investigations focused upon some academic departments of child psychiatry. Documents of interest included the 2002 Annual Report “The Johnson and Johnson (J&J) Center for Pediatric Psychopathology at the Massachusetts General Hospital” (G. Harris & Carey, 2008), which stated,

An essential feature of the Center is its ability to conduct research satisfying three criteria: a) it will lead to findings that improve the psychiatric care of children; b) it will meet high levels of scientific quality and c) it will move forward the commercial goals of J&J.

No one would fault the first two criteria; however, the third criterion is scientifically and ethically problematic. Janssen, a subsidiary of J&J, manufactures the SGA Risperdal. The report outlined the aims of the research:

Because parents, patients and clinicians are exposed to a media that frequently questions the validity of childhood disorders, genetic and brain imaging studies are needed to show the validity of these disorders as brain disorders that respond to medication. . . . Without such data, many clinicians question the wisdom of aggressively treating children with medications, especially those like neuroleptics.

Mental health professionals should be familiar with systemic thinking that includes the biopsychosocial model. But it is not just the biopsychosocial factors acting upon the child and his or her family that need to be considered; indeed, the societal pressures that act upon psychiatry and mental health services also need to be considered. The pharmaceutical industry spends vast sums of money on marketing, research, and continuing medical education, and furthermore economic pressures place pharmaceutical

companies in fierce competition. In this context, the words of the chief executive officer of Eli Lilly, the manufacturer of Zyprexa, as written in an internal e-mail, reveal pressures to find markets in the pediatric age group: “The fact we are now talking to child psychs and peds and others about Strattera means that we must seize the opportunity to expand our work with Zyprexa in this same child-adolescent population” (Berenson, 2008).

There has been growing awareness within the medical profession that liaisons with the pharmaceutical industry can be fraught with ethical dilemmas. As an editorial in the *American Journal of Psychiatry* with 26 signatories put it, “The interacting system of industry-supported clinical trials, advisory boards, and speakers’ bureaus not always, but nonetheless too often, has resulted in conflicts of interest that have demeaned both psychiatry and the pharmaceutical industry” (Freedman et al., 2009, p. 275). Healy and LeNoury (2007) considered that as industry and others gain from the diagnosis, PBD can even be likened to a case of Munchausen’s by proxy.

THE *DSM-III* AND *-IV* HAVE UNDERSTATED ATTACHMENT, TRAUMA, AND CONTEXT

Wittgenstein proposed that language and concepts affect perception (i.e., what is in our vocabulary we see; what is not can easily remain invisible). In psychiatric nosology, Scull (2010) pointed to the *DSM-III*, saying the “revolution” came in the form of an “anti-intellectual system published in book form: a checklist approach to psychiatric diagnosis and treatment . . . with scant regard for whether the new labels . . . cut nature at the joints” (p. 1247). Lane (2007) interviewed several on the *DSM-III* task force to conclude that a political agenda to depose psychoanalysis from its perch atop psychiatry’s power structure drove the “atheoretical model” of the *DSM-III*. Despite significant advances in the attachment theory and traumatology research literature, both the *DSM-III* and *DSM-IV* have generally not incorporated this work. Silberg and Dallam (2009), focusing on dissociation in children and its association with disorganized attachment, relational stress, and trauma, noted that “children with dissociative disorders are frequently misdiagnosed because of their comorbid symptomatology,” and one factor is because “child-specific categories of dissociation do not exist in *DSM-IV*” (p. 70). The problem for psychiatric nosology is that diagnoses, including PBD within the bipolar disorder not otherwise specified rubric, lack relational context and suffer from reification and oversimplification (Dignam, Parry, & Berk, 2010; Parry, 2009).

Neuroimaging of children with disorganized attachment and trauma histories has revealed impaired right prefrontal cortex control over a hyperactive right amygdala. This can be explained in terms of the function of these structures in attachment relationships and for survival in the face of threat

(Schoore, 2002). Neuroimaging of children diagnosed with PBD (DelBello, 2009; Pavuluri, 2009; Pavuluri, Passarotti, Harral, & Sweeney, 2009) found essentially the same findings but made no reference to attachment and trauma factors. As it specifically deals with attachment issues, DTD can be proposed as a more accurate descriptor for many children diagnosed with PBD (Levin, 2009). However, DTD is not officially within the *DSM-IV*. Thus, in the PBD neuroimaging research attention-deficit/hyperactivity disorder and PBD receive consideration, but DTD and attachment and contextual factors do not appear to.

IATROGENIC DISASTER?

Hyman, former director of the National Institute of Mental Health, has said, “The (PBD) diagnosis has spread too broadly, so that powerful drugs are prescribed too widely . . . we are going to have hell to pay in terms of side effects” (Groopman, 2007, p. 31). Elias (2006) reported, “Between 2000 and 2004 there were at least 45 deaths of kids where the ‘primary suspect’ was an atypical (antipsychotic) and more than 1,300 reports of other serious side effects.” G. Harris, Carey, and Roberts (2007) reported, “In 2006 alone the [Food and Drug Administration] received reports of at least 29 children dying and at least 165 more reports of other serious side effects in children where an antipsychotic was listed as the ‘primary suspect.’” Harris (2008) also reported that from “1993 through the first three months of 2008, 1,207 children given Risperdal suffered serious problems, including 31 who died.” This investigative journalism used similar research methodology (personal communication, G. Harris with P. I. Parry, 2008) as academic research by Moore, Cohen, and Furberg (2007; personal communication, Moore with P. I. Parry, 2008), which found that atypical antipsychotics figure highly as a “primary cause” of death in all age groups on the Food and Drug Administration database.

Metabolic adverse effects are a concern with SGAs. In addition, although SGAs are supposedly low in extrapyramidal side effects, 430 children in foster care in the state of Texas in 2004 “were prescribed antidyskinetics drugs to control side effects from antipsychotics” (Strayhorn, 2006, p. 77). The academic literature (Wonodi et al., 2007) adds concern with a finding of a 6% rate of tardive dyskinesia in a cohort of 5- to 18-year-olds on SGAs for over 6 months. Zito et al. (2008) have drawn further academic attention to the harms of polypharmacy for Texas foster children.

In addition to physical morbidity and mortality, there can be adverse effects on a young person’s self-concept and psychosocial development from an erroneous label of PBD (Purcell, 2007). It can also be argued that parent-child communication is constricted in meaning if reduced to, or overly focused upon, the vocabulary of mental symptoms and medication.

PENDULUM SWINGING BACK FROM PBD AND MINDLESS PSYCHIATRY

There are signs that psychiatry's paradigmatic pendulum may be swinging back from the mindless extremity of its arc. A 2-day workshop (Parens & Johnson, 2010) on controversies in PBD attended by some leading figures in child psychiatry concluded that "the bipolar label may fit poorly many of the children who have received it over the last decade" (p. 20) and highlighted the importance of a child's social "context." The workshop also pointed to problems of diagnostic upcoding: "It is a deeply regrettable feature of our current mental health and educational systems that some *DSM* diagnoses are better than others at getting children and families access to the care and services they so desperately need." The 2010 AACAP meeting included two symposia on PBD (AACAP, 2010a, 2010b), both questioning the diagnosis in many cases and highlighting research on contextual factors in affect regulation. Finally, one sign of change coming from the highest levels of the AACAP is that a September 2, 2010, *New York Times* article on Kyle Warren (Wilson, 2010) was e-mailed to all members of the AACAP by the president, Larry Greenhill. Professor Greenhill requested that AACAP members "please take a moment to read the article and watch the (associated) video." We would like to request the same of our readers.

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