CASE STUDY

47 BIPOLAR DISORDER

For the Patient Case for this case study, see the printed book.

DISEASE SUMMARY

Definition

Bipolar disorder (BD) is a common, serious, persistent, and highly complex brain and mood disorder characterized by recurring episodes of depression and mania with intervening periods of normal moods. The disorder was originally named for its characteristic profound changes in mood (i.e., opposite "poles of emotion"), cycling between euphoric happiness and extreme sadness. Everyone has occasional "highs" and "lows" in their moods. However, people with BD have extreme mood swings. They can go abruptly from feeling very sad, despairing, helpless, worthless, and hopeless to feeling as if they are "on top of the world," creative, and engaging. Depression or mania may be predominant at any given time, one mood may alternate with the other, or elements of both moods may be present simultaneously.

There are two primary types of BD, referred to as bipolar I (BPI) and bipolar II (BPII). A diagnosis of BPI requires at least one episode each of full-fledged mania and major depression. A diagnosis of BPII requires at least one episode each of partial mania or *hypomania* (i.e., elevated levels of energy and impulsiveness that are not as extreme as the symptoms of mania but last a minimum of 4 days) and major depression. A mild form of BD known as *cyclothymia* involves periods of hypomania and mild depression with less severe mood swings.

Bipolar disorder has been recognized at least since the time of Hippocrates, who described patients with the condition as "amic" and "melancholic." In 1899, Emil Kraepelin defined manic-depressive illness and noted that the disorder was not associated with deterioration or dementia, both of which he associated with another psychiatric disorder—schizophrenia. In the past, BD was commonly known as *manic-depressive illness* or simply as *manic depression*.

Prevalence

Comparable to an international lifelong prevalence rate of 0.3–1.5%, the lifelong prevalence rate of BD in the United States is 1.0–1.6%. Approximately 2.3 million people in the United States have been diagnosed with BD. An estimated 0.8% of people age 18 years and older in any given year are living with BPI. Approximately 0.5% of adults have BPII. The overall prevalence of BPI in adolescents is approximately 1%, whereas the prevalence in children is 0.2–0.4%.

No racial predilection exists for the disorder. BPI occurs equally in both genders. However, rapid-cycling BPI (i.e., four or more episodes each year) is more common in women than in men. The frequency of BPII is also higher in females.

Age of onset for both BPI and BPII varies greatly from childhood to age 50 years with a mean age of approximately 21 years. Most cases are diagnosed when individuals are age 15–30 years.

Significance

Bipolar disorder is often a serious, chronic, and recurring struggle for both patients and their families. Both phases of the disease—mania and depression—affect thinking, judgment, and social behavior and can be harmful to the patient. The disorder can lead to poor school performance, risky and reckless behaviors, marital problems, and damaged personal relationships and careers. Extreme mania can lead to aggressive behavior and patients may become homicidal by acting on delusions, while severe depression carries with it thoughts and acts of suicide. Between one fourth and one half of all individuals with BD attempt suicide, and 11% complete the suicidal act. The flares of BD can last for months, causing a significant disturbance in the lives of those affected, their friends, and their families. Often, the cycling between mania and depression accelerates with age.

Because of the extreme and risky behaviors associated with BD, it is important that the disorder be diagnosed promptly after onset. With proper and early diagnosis, the condition can be treated. However, there is a wide spectrum of symptoms and mood changes and the pattern of highs and lows varies for each person. As a result, BD is a complex disease to diagnose. Accurate diagnosis of BD when the initial symptom is depression can be especially difficult and long delayed. Furthermore, the ability of BD to mimic aspects of many other mental illnesses and a strong association with substance abuse can also make the initial diagnosis difficult. Approximately three of five patients with BD are also dependent on drugs and/or alcohol.

Bipolar disorder can also be very problematic to treat. At most, 60% of patients with BPI gain control of their symptoms with medication, while 40% suffer from a persistent disorder.

Bipolar disorder is equally difficult for families of those affected and is one of the most difficult mental illnesses for relatives to accept. When a person seems completely normal and sometimes very productive, then suddenly becomes unreasonable or irrational, it appears more like bad behavior than a disease and wreaks havoc within the family.

Finally, the disease has a significant economic impact on the nation. The cost of lost productivity alone in the United States has been estimated at approximately $15.5 billion annually.

Causes and Risk Factors

Decades of clinical and genetic research findings indicate that BD has a number of contributing factors, including genetic, biochemical, psychodynamic, and environmental elements. Furthermore, a combination of genetic vulnerability, neurodevelopmental events, and both physiologic and psychosocial stressors appear to be involved.

A current view of mood disorders in general is that these types of illnesses stem from a complex interplay between the inheritance of certain genes that increase susceptibility to a mood disorder and environmental influences that trigger the onset of symptoms. The development of a mood disorder like BD appears to depend on how individuals with a genetic risk respond to their environment.

Genetic and familial factors have an especially profound influence on the development of BD, as the disorder tends to run in families. A positive family history of depression appears to exist in 80–90% of all cases of BD. The risk for developing BD among offspring in which one parent has BD is estimated to be approximately 30–35%, compared with a risk of 1% in the general population. When both parents are bipolar, the risk increases to 70–75%. Twin studies have shown a 65% concordance rate (ranging from 33 to 90%) in monozygotic/identical twins (who share all the same genes), compared with a concordance rate of only 14% in dizygotic/non-identical twins. Even among adopted children with a biologic family history of mood disorders, the frequency of BD is higher than among adoptees with a negative family history. Genetic studies of patients with BD are ongoing and are expected to be facilitated by recent advances in information and technology developed, in part, from the Human Genome Project.

Although genetic influences are clearly important as a contributing factor of mood disorders, environmental factors—such as stressful events involving loss, humiliation, or threat—play a significant role in increasing risk. Pregnancy is also a type of external stress common to women with a history of BD and may serve to trigger the onset of the illness.

There is also a clear biochemical link to BD, as various chemical agents are known to cause depression (e.g., reserpine, methyldopa) or mania (e.g., amphetamines, cocaine, levodopa). Hormonal imbalances and disruptions in normal physiologic pathways among the hypothalamus, pituitary gland, and adrenal glands—which are involved in both homeostasis and the stress response—also may contribute to the clinical picture of BD.

Many practitioners see the major moods of BD as being linked through one common psychodynamic pathway. They see depression as a manifestation of a significant loss (e.g., death of a spouse) and mania as a coping mechanism for feelings of depression.

Finally, cognitive and neurodevelopmental factors also seem to contribute to BD. A recent case-cohort study of adolescents with mood disorders revealed that neurodevelopmental delays are over-represented among patients with BD. These delays occur in language, social, and motor development approximately 10–18 years before symptoms of mental illness develop. In addition, intelligence quotient (IQ) scores were significantly lower (mean of 88.8) in patients with early-onset BD.

Pathophysiology

The pathophysiology of BD has not been completely determined, and no objective biological markers that correspond definitively with the disease state have been identified. Most experts in the field agree, however, that there are multiple pathophysiologic mechanisms that cause BD.

The strong tendency for BD to run in families has encouraged a search for specific gene mutations. Findings from gene research suggest that BD does not result from a single abnormal gene. Rather, it appears likely that multiple mutations (i.e., *polygenic*) acting together with factors in an individual's environment cause the illness. Identifying these mutations, each of which probably contributes only a small fraction to genetic vulnerability, has been extremely difficult. Still, several promising molecular genetic studies suggest that genes on chromosomes 4, 12, 18, 21, and/or 22 may be involved.

Recent findings from gene expression studies of postmortem brain tissue samples from people who suffered from BD have yielded some insight into the pathophysiology of the disorder. In particular, the expression of oligodendrocyte-myelin–related genes appears to be decreased in BD. Oligodendrocytes synthesize myelin, which wraps around and insulates neuronal axons and facilitates conduction of nerve impulses in the brain. The loss of myelin is believed by some to interrupt communication between neurons, leading to the thought disturbances observed in patients with BD and related illnesses. Brain imaging studies of patients with BD also show abnormal myelination in several regions of the brain that have been linked to the disorder. In some cases of familial BD, brain imaging studies have also demonstrated a reduction in the volume of gray matter in the prefrontal cortex with associated decreased activity in this region. Clinical studies have suggested that this area of the brain is an important mediator of mood states.

Investigators have recently demonstrated that two chemically unrelated drugs used to effectively treat BD—lithium and valproate—both up-regulate (i.e., increase) the expression of the cytoprotective protein *Bcl-2* in the frontal cortex and hippocampus of rat brains. Neuroimaging studies of bipolar patients also provide evidence of atrophy (i.e., decrease in cell size) or cell death in these same regions of the brain. As a result, another potential mechanism in the pathophysiology of BD is injury to cells (e.g., from drugs of abuse or excessive glucocorticoid stimulation from stress) in the brain circuitry that regulate mood.

Bipolar disorder may also be, at least in part, the result of a chemical imbalance within the brain. Functions of the brain are regulated by chemicals known as *neurotransmitters*. An imbalance in neurotransmission involving one or more neurotransmitters may trigger the onset of depression or mania. The *biogenic amine hypothesis* suggests that subnormal amounts of serotonin, norepinephrine, or dopamine within the synaptic cleft are responsible for depression, while abnormally high amounts of these monoamines cause mania. This hypothesis, therefore, stems from an assumption that depression and mania are the result of

two different neurobiologic processes. It is noteworthy that the three major classes of antidepressant medications—monoamine oxidase inhibitors, tricyclic antidepressants, and selective serotonin reuptake inhibitors—share a common property, albeit through different pharmacologic mechanisms, that of increasing neurotransmitter concentrations within the synapse. It has become increasingly clear, however, that simple changes in the concentration of amines within neuronal synapses cannot totally explain the complexities of BD. Neuromodulatory systems in the brain interact with one another in a complex fashion; for example, neuronal pathways mediated by acetylcholine, glutamate, and γ-aminobutyric acid (GABA) may also play key roles because they influence the activity of various neurotransmitter-producing neurons.

Kindling refers to a phenomenon in which a stressor creates an electrophysiologic vulnerability to future stressful events by producing long-lasting changes in neuronal function. Kindling may provide the basis for rapid cycling in BD—the more frequently that a person has a shift in mood, cycling into either mania or depression, the easier it becomes to experience another episode. There is now evidence that many psychiatric disorders, not just BD, may be subject to this phenomenon.

Diagnosis: Clinical Manifestations and Laboratory Tests

The appropriate first step in evaluating a patient for BD is to ensure that a physical medical condition is not causing mood and thought disturbances. As a result, the evaluation of the patient is best started by obtaining a history of current and past medical and behavioral symptoms and treatments. The possibility that substance abuse or dependence, trauma to the brain, and/or a seizure disorder may be a contributing factor is explored. Central nervous system insults (e.g., deterioration of brain function due to renal failure) or chemically induced (e.g., corticosteroids) mood changes are also considered. Information about the family's psychiatric history is another essential part of the patient's history. Gathering additional information from family members and friends, teachers, caregivers, physicians, and mental healthcare workers is common practice when the patient is experiencing an altered mood or behavioral state. In addition, the patient's subjective experience is essential in the overall evaluation.

The hallmark of BD is at least one episode each of mild-to-severe depression and mild-to-severe mania. ***An episode of depression is present when five or more of the following symptoms last most of the day, nearly every day, for a minimum of 2 weeks.***

- persistent sad, anxious, or empty mood with occasional crying spells
- feelings of excessive worry, hopelessness, or pessimism
- feelings of guilt, worthlessness, or helplessness
- loss of interest or pleasure in activities once enjoyed, including sex
- very little energy or a feeling of fatigue or of being "slowed down"
- difficulty concentrating, remembering, or making decisions
- restlessness or irritability
- abnormal sleep pattern (e.g., sleeping too much during the day with insomnia at night)
- little regard for general appearance
- social withdrawal
- poor or voracious appetite
- chronic pain or other persistent symptoms that are not caused by physical illness or injury
- persistent thoughts about death or suicide

An episode of mania is usually present if elevated mood occurs with three or more of the following symptoms most of the day, nearly every day, for a minimum of 1 week. If extreme irritability, however, is one of the symptoms, a minimum total of five symptoms must be present.

- increased energy, activity, and restlessness (may manifest as pacing or tapping the feet)
- excessively "high" euphoric mood
- extremely irritable and argumentative with poor temper control
- racing thoughts, talking fast, and changing subjects rapidly

- distractibility with an inability to concentrate
- lack of sleep from euphoric state
- unrealistic beliefs in one's abilities and powers (e.g., inflated self-esteem, feeling indestructible)
- poor judgment and decision making
- lasting period of unusual behavior that is characterized by a lack of self-control and increased activities with high risk for painful consequences (e.g., spending sprees, sexual promiscuity, gambling, reckless driving, risky investments)
- abuse of drugs, especially cocaine, alcohol, and sleeping medications
- denial that anything is wrong with refusal to see a medical professional
- delusions (i.e., false beliefs) or hallucinations (i.e., seeing or hearing things that are not real)

Episodes of mania begin abruptly, are sometimes precipitated by stress, and may last for several months. Spring and summer tend to be peak periods of occurrence. Generally, manic episodes are of shorter duration than depressive episodes.

Patients with four or more discrete episodes of mania or depression in 1 year are known as "rapid cyclers" and have a high frequency of hypothyroidism. *Cyclothymia* refers to mild episodes of depression and mania that have a minimal duration of 2 years. Occasionally, symptoms escalate into a full-blown manic or depressive episode, in which case reclassification as BPI or BPII is warranted.

Any potentially treatable physical condition that can account for severe mood swings is excluded first. The physical examination focuses on a general neurologic examination and tests for cardiovascular and pulmonary function. Abnormal pulmonary function with hypoxemia (i.e., subnormal oxygen tension in the blood) or poor blood flow to the brain has been known to cause abnormal mood, behavior, or cognition. No laboratory test or imaging study is available that will confirm the diagnosis of BD. However, many tests are often conducted to rule out physical causes of mood swings. Disease Summary Table 47.1 shows medical tests that may reveal physical causes of depression and/or mania.

Disease Summary Table 47.1 Medical Tests That May Reveal Physical Causes of Depression and/or Mania

Medical Test	Potential Cause of Abnormal Moods
Complete blood count (CBC) with differential	Anemia → depression
Erythrocyte sedimentation rate (ESR)	Influenza, viral hepatitis, or infectious mononucleosis → depression Influenza, St. Louis encephalitis, Q fever → mania
Electrolyte panel	Hyponatremia → depression
Fasting serum glucose concentration	Diabetes mellitus
Serum calcium and parathyroid hormone concentrations	Hyperparathyroid disease → depression
Thyroid function studies	Hyperthyroidism → mania Hypothyroidism → depression
Serum cortisol concentration	Hypercortisolism (Cushing syndrome) → depression Hypocortisolism (Addison disease) → depression
Screening tests for drugs of abuse	Amphetamine- and cocaine-induced mania Barbiturate-induced depression
Urine copper concentration	Wilson disease
Antinuclear antibody (ANA) test	Systemic lupus erythematosus → depression or mania
Human immunodeficiency virus (HIV) test	HIV-related disease → depression
Venereal Disease Research Laboratories (VDRL) test	Neurosyphilis
Serum creatinine and blood urea nitrogen (BUN)	Kidney failure → depression
Electroencephalogram	Seizure disorder, brain tumor
Computed tomography (CT) or magnetic resonance imaging (MRI) of brain	Blood clot, bleeding, brain tumor
Spinal tap (lumbar puncture)	Central nervous system infection (i.e., meningitis, encephalitis)
Serum vitamin B12 concentration	Vitamin B12 deficiency
Serum niacin concentration	Niacin deficiency → depression

Source: Soreff S, McInnes LA. Bipolar affective disorder. eMedicine website. Available at: www.emedicine.com/med/topic229.htm. Date accessed: July 2007.

If these examinations do not reveal a physical condition that may be contributing to the patient's mental state, the diagnosis is most likely a psychiatric disorder and a mental health evaluation is usually performed. ***Mental health professionals rely almost exclusively on the patient's symptoms to diagnose mental disorders.***

The Mental Status Examination (MSE) is the essential component of a mental health evaluation. The MSE assesses general appearance and demeanor, speech, movement, and interpersonal relatedness of the patient with the examiner. Mood and cognitive abilities (e.g., orientation to circumstance; attentiveness; immediate-, short-, and long-term memory) are also assessed with the MSE. Some of the most important components of the MSE are those that address issues of safety; thus, suicidal and homicidal issues are explored. Screening tests for the more subtle forms of psychosis—such as paranoid or delusional states—are also conducted. Finally, insight into the patient's mental and physical status, current circumstances of medical or mental health care, and the patient's ability to use age-appropriate judgments are assessed and integrated into the evaluation of the patient's current overall mental state. Disease Summary Table 47.2 highlights major findings in patients with BD based on the MSE.

Disease Summary Table 47.2 Major Findings in Patients with Bipolar Disorder Based on the Mental Status Examination

Feature Assessed	Depressive Episode	Manic Episode
APPEARANCE	Minimal eye contact Clothes not clean, not ironed, ill-fitting Poor grooming, has not shaved or washed In women: fingernails show different layers of polish, inattentiveness to hair In men: dirty fingernails and hands	Hyperactive Talks fast Clothes are too bright, colorful, or garish and dress frequently attracts attention
MOVEMENT	Little movement Slow movement Speaks in low tones or monotone voice	Hyperactive and restless
AFFECT/MOOD	Sadness Hopelessness Helplessness Isolation Emptiness	Inappropriately joyous, elated, jubilant May demonstrate annoyance, irritability when mania has been present for some time
THOUGHT	Preoccupied with negative ideas Focus is on death, suicide	Excessively self-confident Highly optimistic thinking See themselves as being highly engaging and creative Highly distractible Rapid shifting of thoughts and ideas
PERCEPTIONS	May be accompanied by delusions and hallucinations	May be accompanied by delusions of power, prestige, position, self-worth
SUICIDE/SELF-DESTRUCTION	Incidence of suicide is high; the more specific the suicide plan, the higher the danger	Incidence of suicide is low
HOMICIDE/ VIOLENCE/ AGGRESSION	Homicide followed by suicide may occur	Openly combative and aggressive No patience or tolerance for others Highly demanding, violently assertive, highly irritable Homicidal element with delusions
JUDGMENT/ INSIGHT	Failure to act on important issues Difficulty planning for the future Little insight into own behavior	Poor decisions made at work or with relatives Poor financial investments Become over-involved with work Do not listen to suggestions or advice No insight into extreme nature of their demands, plans, behavior
COGNITION	Impairments in orientation and memory rare Difficulty concentrating when depression is extreme	Impairments in orientation and memory rare Information recall may be expanded and vivid Difficulty concentrating when mania is extreme

Source: Soreff S, McInnes LA. Bipolar affective disorder. eMedicine website. Available at: www.emedicine.com/med/topic229.htm. Date accessed: July 2007.

Disease Summary Question 1. Indicate with the letters **D** (for depression) or **M** (for mania) the appropriate phase of bipolar disorder for the patients whose words appear directly below.

_____ "I doubt completely my ability to do anything well."

_____ "It seems as though my mind has slowed down and burned out to the point I am totally useless."

_____ "Ideas come too fast and there are so many."

_____ "I am haunted with the total and desperate hopelessness of it all."

Appropriate Therapy

Treatment and management of BD are complicated. The primary goals of treatment are to control and minimize symptoms of BD, lengthen the periods of normal mood states, minimize the number of hospitalizations required, minimize medication-induced side effects to a tolerable level, and optimize quality of life for the patient.

Treatment of BD is, in part, directly related to the specific phase of the episode—either depression or mania—and the severity of the phase. A person who is extremely depressed and exhibits suicidal behavior requires inpatient treatment. In contrast, an individual with moderate depression who still can work is treated as an outpatient. Indications for hospitalization of a patient with BD include danger to self, danger to others, total inability to function, totally out of control, and medical conditions that warrant monitoring of medications.

Partial hospitalization or a daily treatment program is an option for patients with severe symptoms but who also have both a significant level of control and a stable living environment. A patient with severe depression and thoughts of suicide but no plan to act upon them, a high degree of motivation to get well, and the help of an involved and supportive family may benefit from this type of treatment. Partial hospitalization also offers a bridge to return to work.

Outpatient treatment has three major goals: (1) to identify areas of patient stress and design strategies to manage this stress; (2) to monitor the effects of and support the patient's need for long-term medication; and (3) to educate both the patient and family about BD. ***The most effective treatment for BD is a combination of medication and psychosocial intervention.*** Occasionally, electroconvulsive therapy (ECT) is employed.

Most people with BD take medication to regulate their moods, and the choice of medications is customized for each patient. When treated with mood-stabilizing agents, bipolar patients have fewer episodes of mania and depression and the severity of these episodes is decreased. ***Lithium has been widely used as a mood stabilizer and is generally considered the first-line agent for long-term prophylaxis of bipolar illness (both BPI and BPII) and for treatment of a manic episode.*** However, the drug may not take full effect for 2 weeks. Evidence suggests that lithium, unlike any other mood stabilizer, also has a specific antisuicidal effect.

As many as 30% of all bipolar patients do not respond to lithium; therefore, other medications are often tried to control symptoms. *Antiseizure medications* have also been effective in preventing mood swings associated with BD, especially in patients with rapid-cycling BD. The most widely used antiseizure agents have been carbamazepine, divalproex (i.e., valproic acid + valproate sodium), lamotrigine, and, more recently, oxcarbazepine and topiramate. Topiramate also has the unique feature of promoting weight loss as a side effect. *Antidepressants* are sometimes used to treat depression associated with BD but must be used with a mood-stabilizing agent to prevent onset of a manic episode. Paroxetine, fluoxetine, sertraline, and bupropion are commonly used antidepressants by patients with BD. In circumstances when patients have lost touch with reality, *antipsychotic agents* such as risperidone, olanzapine, or quetiapine are used. *Benzodiazepines* (e.g., clonazepam) generally are avoided because of their addictive potential but may be temporarily useful in restoring sleep or in regulating irritability or agitation.

As an addition to medication, psychosocial treatments are helpful in providing support, education, and guidance for bipolar patients and their families. Studies have shown that

psychosocial interventions can lead to increased mood stability, fewer hospitalizations, and improved overall function. Psychosocial interventions commonly used for BD include cognitive behavioral therapy, psychoeducation, family therapy, and interpersonal and social rhythm therapy. *Cognitive behavioral therapy* helps patients to change inappropriate or negative thought patterns and behaviors associated with BD. *Psychoeducation* involves teaching patients about the illness, its treatment, and recognition of signs of relapse so that early intervention can be sought before a full-blown episode of depression or mania occurs. *Family therapy* uses strategies to reduce the level of distress within the family that may contribute to or result from the patient's symptoms, improve family communication, and address unresolved feelings of fear, hurt, or loss. *Interpersonal and social rhythm therapy* helps patients both improve their interpersonal relationships and set regular daily routines and sleep schedules that may prevent manic episodes.

ECT is used mainly in patients who have suicidal tendencies or when medications have proven ineffective or intolerable. In fact, studies have repeatedly shown that ***ECT is the most effective treatment for depression that is not relieved with medications.*** In one study of 400 bipolar patients who received ECT, 313 showed significant clinical improvement. Under anesthesia and following treatment with a muscle relaxant to avoid injury to self, a small electrical current is briefly passed into the brain through electrodes placed on the head. The current profoundly affects brain metabolism and blood flow, produces a brain seizure, and is safe and often highly effective in controlling the symptoms of BD. A major favorable aspect of ECT is its more rapid onset of therapeutic response compared with medications—days instead of weeks. A major drawback of ECT is temporary memory loss.

Since no well-controlled research studies of bipolar treatment modalities in adolescents are available to provide evidence-based medical care, treatment of bipolar patients in this age group is modeled after treatments provided to adult patients as described above.

Anyone who has experienced two or more episodes of BD is considered a long-term bipolar patient and requires maintenance therapy, sometimes for life. No surgery is indicated for BD and special diets are rarely required. Patients in the depressed phase are encouraged to exercise, as regular physical activity may provide a key to surviving the disorder.

Disease Summary Question 2. Match the generic names of the antidepressants listed directly below with their appropriate and commonly used brand names.

A. bupropion	_____ Prozac
B. sertraline	_____ Paxil
C. paroxetine	_____ Wellbutrin
D. fluoxetine	_____ Zoloft

Disease Summary Question 3. Three of the four medications listed in Question 2 above are selective serotonin reuptake inhibitors (SSRIs), that is, they inhibit reuptake by nerve terminals so that serotonin is available for neurotransmission within the synapse. Which of the four agents is not an SSRI?

Serious Complications and Prognosis

Bipolar disorder currently has no cure and the presence of mood swings should be anticipated throughout life. The frequency and severity of episodes are not readily predictable. However, people with BD can lead healthy and productive lives when the illness is effectively treated. Patients may pursue a college education and careers with much success, and strong relationships may be developed. Without treatment, the natural course of BD tends to worsen. Over time patients may suffer more frequent and more severe attacks of mania and depression. In most cases, proper treatment can help reduce the frequency and severity of episodes and help patients maintain good quality of life.

Approximately 55% of patients with BD gain control of their symptoms. Forty-five percent experience more episodes and forty percent develop a persistent, recurring disorder. Factors that suggest a poorer prognosis include poor job history, alcohol abuse, psychotic

features, early age of onset, and male gender. Indicators of a better prognosis include late age of onset, few thoughts of suicide, few psychotic symptoms, and few medical problems.

Some of the more serious complications of BD include poor judgment and decision making, substance abuse, risky and reckless behaviors, aggressive behavior that may result in homicide, and suicide. The risk for self-destructive behavior and death presents a lifelong challenge.

Suggested Readings

Aloi M. Bipolar disorder. eMedicineHealth website. Available at: www.emedicinehealth.com/bipolar_disorder/article_em.htm. Date accessed: October 2007.

Ballas P. Bipolar disorder. MedlinePlus Medical Encyclopedia website, National Library of Medicine and National Institutes of Health. Available at: www.nlm.nih.gov/medlineplus/ency/article/000926.htm. Date accessed: November 2006.

Eisendrath SJ, Lichtmacher JE. Bipolar disorders. In: McPhee SJ, Papadakis MA, Tierney LM Jr., eds. 2007 Current Medical Diagnosis and Treatment. 46th Ed. New York: McGraw-Hill, 2007:1091.

Katz-Wise SL, Tank C. Bipolar disorder—Topic overview. WebMD website. Available at: www.webmd.com/bipolar-disorder/tc/Bipolar-Disorder-Topic-Overview. Date accessed: April 2006.

Mayo Clinic staff. Bipolar disorder. Mayo Clinic website. Available at: www.mayoclinic.com/health/bipolar-disorder/DS00356. Date accessed: November 2006.

MedicineNet website. Bipolar disorder (mania). Available at: www.medicinenet.com/bipolar disorder/article.htm. Date accessed: October 2007.

MedicineNet website. Definition of bipolar disorder. Available at: www.medterms.com/script/main/art.asp?articlekey=2468. Date accessed: June 2006.

Oakley LD. Bipolar disorder. In: Copstead LEC, Banasik JL, eds. Pathophysiology. 3rd Ed. St. Louis: Elsevier Saunders, 2005:1207–1212.

Pasch SK. Bipolar depression. In: Porth CM, ed. Pathophysiology—Concepts of Altered Disease States. 7th Ed. Philadelphia: Lippincott Williams & Wilkins, 2005:1278–1280.

Soreff S, McInnes LA. Bipolar affective disorder. eMedicine website. Available at: www.emedicine.com/med/topic229.htm. Date accessed: July 2007.

Takahashi LK. Mood disorders: Depression and mania. In: McCance KL, Huether SE, eds. Pathophysiology: The Biologic Basis for Disease in Adults and Children. 5th Ed. St. Louis: Elsevier Mosby, 2006:610–616.

Upadhyaya HP, Fields MC, Gray KM. Mood disorder: Bipolar disorder. eMedicine website. Available at: www.emedicine.com/ped/topic240.htm. Date accessed: October 2006.