TABLE 109-2 IMPORTANT PSYCHIATRIC SYNDROMES

SYNDROME	MAIN SYMPTOMS AND SIGNS	DISORDERS
Neurocognitive	Impairment in intellectual functions (e.g., level of consciousness, orientation, attention, memory, language, praxis, visuospatial, executive functions)	Neurocognitive disorders Intellectual disability (if onset in childhood)
Mood	Depressive: lowered mood, anhedonia, negativistic thoughts, neurovegetative symptoms Manic: elevated or irritable mood; grandiosity; goal-directed hyperactivity with increased energy; pressured speech; decreased sleep need	Neurocognitive disorders Mood disorders (bipolar or depressive) (primary or secondary) Psychotic disorders (schizoaffective disorder)
Anxiety	All include anxious mood and associated physiologic signs and symptoms (e.g., palpitations, tremors, diaphoresis) May include various types of dysfunctional thoughts (e.g., catastrophic fears, obsessions, flashbacks) and behaviors (e.g., compulsions, avoidance behaviors)	Neurocognitive disorders Mood disorders (bipolar or depressive) (primary or secondary) Psychotic disorders (primary or secondary) Anxiety disorders (primary or secondary)
Psychotic	Impairments in reality testing: hallucinations, thought process derailments	Neurocognitive disorders Mood disorders (bipolar or depressive) (primary or secondary) Psychotic disorders
Somatic symptom syndromes	Somatic symptoms with associated distressing thoughts, feelings, or behaviors	Mood disorders (bipolar or depressive) (primary or secondary) Anxiety disorders (primary or secondary) Obsessive-compulsive and related disorders Trauma-related disorders Somatic symptom disorders
Personality pathology	Dysfunctional enduring patterns of emotional regulation, thought patterns, interpersonal behaviors, impulse regulation	Neurocognitive disorders Personality change due to another medical condition Personality disorders

Data from American Psychiatric Association: Diagnostic and statistical manual of mental disorders, ed 5, Washington, D.C., 2013, American Psychiatric Association.

of the serotonin transporter protein, affect vulnerability to depressive episodes in the face of psychosocial stressors. Depression is polygenic and multifactorial, with genetic factors accounting for about 40% of the risk. Alterations in the functioning of brain serotonergic and noradrenergic systems and of the hypothalamic-pituitary-adrenal axis are found in depression. Neuroimaging studies show smaller hippocampal volumes and altered metabolic activity in several regions, including the anterior cingulate cortex. However, the information in these studies is not sufficient for making the clinical diagnosis, which depends on identification of the clinical syndrome. Dysfunctional, negativistic patterns of thinking, impaired social relationships, and stressful life events also contribute to depression.

Mild to moderate forms of major depression respond to focused psychotherapies or antidepressant medications (level A evidence) (Table 109-4). More severe forms of depression do not respond to psychosocial interventions alone. Severe or refractory depression may be treated safely and effectively with electroconvulsive therapy (level A). Other evidence-based somatic therapies include light therapy (for depression with a seasonal component) and vagal nerve stimulation (levels B and C). Data suggest that the dissociative anesthetic ketamine, an *N*-methyl-D-aspartate (NMDA) receptor antagonist, may rapidly improve patients with treatment-resistant depression, although the general clinical applicability of ketamine remains to be determined.

Bipolar disorder (i.e., bipolar I) is characterized by recurrent episodes of mania, usually with episodes of major depression. Manic episodes include elevated (euphoric) or irritable mood, goal-directed hyperactivity (often for pleasurable activities with poor judgment leading to substantial adverse consequences such as sexual, spending, or gambling sprees), pressured speech, increased energy level with a decreased need for sleep, and distractibility.

Compared with unipolar depression, bipolar disorder has a lower 12-month prevalence (approximately 0.6%) and a younger average age of onset (typically late teens to 20s). Unlike unipolar depression, bipolar disorder is slightly more common among males. Most patients return to baseline functioning between acute mood episodes, but some have a deteriorating course, and others have frequent debilitating episodes (i.e., rapid cycling of four episodes per year).

Genetic factors play a greater role in the pathogenesis of bipolar disorder than in major depressive disorder, accounting for approximately 50% of the risk and representing a greater than 50-fold increase over the population base rate. Bipolar disorder is polygenic and has been linked in individual families to different loci. The pathogenesis is unclear but likely involves dysregulation of frontostriatal systems. Structural neuroimaging studies show increased ventricular-to-brain ratios, suggesting parenchymal atrophy. Psychosocial stressors often play a role in precipitating episodes of mania and depression.

The mainstay of treatment for bipolar disorder is mood stabilizer medications (e.g., lithium, anticonvulsants such as valproic acid and carbamazepine) for acute episodes and maintenance therapy (level A evidence). The anticonvulsant lamotrigine may be particularly useful for bipolar depression. Antipsychotic medications are useful for acute manic episodes and may have a role in maintenance therapy. Benzodiazepines may be used to treat acute agitation and aggression while waiting for more definitive antimanic therapies to take effect. Antidepressants have long been used for depressive episodes, although they may precipitate manic episodes.

Electroconvulsive therapy is effective for refractory mania (level B evidence) and depression (level A). Psychosocial treatments alone do not effectively treat mania and may be less effective for bipolar depression, but psychoeducation and support to