Blueprints Psychiatry

3rd edition

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BLUEPRINTS PSYCHIATRY

Third Edition

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Preface

In 1997, the first five books in the *Blueprints* series were published as board review for medical students, interns and residents who wanted high-yield, accurate clinical content for USMLE Steps 2 & 3. Six years later, we are proud to report that the original books and the entire *Blueprints* brand of review materials have far exceeded our expectations.

The feedback we've received from our readers has been tremendously helpful and pivotal in deciding what direction the third edition of the core books will take. The student-to-student approach was highly acclaimed by our readers, so resident contributors have been recruited to ensure that the third edition of the series continues to provide content and an approach that made the original *Blueprints* a success. It was suggested that the review questions should reflect the current format of the Boards, so new board-format questions have been included in this edition with full explanations provided in the answers. Our readers asked for an enhanced art program, so a second color has been added to this edition to increase the usefulness of the figures and tables.

What we've also learned from our readers is that *Blueprints* is more than just Board review for USMLE, Steps 2 & 3. Students use the books during their clerkship rotations and subinternships. Residents studying for USMLE Step 3 often use the books for reviewing areas that were not their specialty. Students in physician assistant, nurse practitioner and osteopath programs use *Blueprints* either as a companion or in lieu of review materials written specifically for their areas.

However you use *Blueprints*, we hope that you find the books in the series informative and useful. Your feedback and suggestions are essential to our continued success. Please send any comments you may have about this book or any book in the *Blueprints* series to *blue@bos.blackwellpublishing.com*.

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Ronald L. Cowan, MD, PhD
Michael J. Murphy, MD, MPH

This third edition stands as a testimonial to the fine work of my colleagues, Drs. Cowan and Murphy, and to the lessons we have all learned from our patients and our students.

Lloyd I. Sederer, MD

Abbreviations

AA	Alcoholics Anonymous
ABG	Arterial blood gas
ACLS	Advanced cardiac life support
ADHD	Attention-deficit/hyperactivity disorder
ASP	Antisocial personality disorder
BAL	Blood alcohol level
BID	Twice daily
CBC	Complete blood count
CBT	Cognitive-behavioral therapy
CNS	Central nervous system
CO_2	Carbon dioxide
CPR	Cardiopulmonary resuscitation
CSF	Cerebrospinal fluid
CT	Computerized tomography
CVA	Cerebrovascular accident
DBT	Dialectical behavior therapy
DID	Dissociative identity disorder
DT	Delirium tremens
ECT	Electroconvulsive therapy
EEG	Electroencephalogram
ECG	Electrocardiogram
EPS	Extrapyramidal symptoms
EW	Emergency ward
FBI	Federal Bureau of Investigation
5HIAA	5-hydroxy indoleacetic acid
5HT	5-hydroxy tryptamine
GABA	Gamma-amino butyric acid
GAD	Generalized anxiety disorder
GHB	Gamma-hydroxybutyrate
GI	Gastrointestinal
HPF	High power field
HIV	Human immunodeficiency virus
ICU	Intensive care unit
IM	Intramuscular
IPT	Intrapersonal therapy
IQ	Intelligence quotient
IV	Intravenous
LP	Lumbar puncture
LSD	Lysergic acid diethylamine
MAOI	Monoamine oxidase inhibitor
MCV	Mean corpuscular volume
MDMA	Ecstasy
MR	Mental retardation
MRI	Magnetic resonance imaging
NIDA	National Institute on Drug Abuse
NMS	Neuroleptic malignant syndrome
OCD	Obsessive-compulsive disorder
PCA	Patient controlled analgesia
PMN	Polymorphonuclear leukocytes
PO	By mouth
PTSD	Posttraumatic stress disorder
QD	Each day
REM	Rapid eye movement
SES	Socioeconomic status
SSRI	Selective serotonin reuptake inhibitor
TD	Tardive dyskinesia
T4	Tetra-iodo thyronine
T3	Tri-iodo thyronine
TCA	Tricyclic antidepressant
TID	Three times daily
TSH	Thyroid-stimulating hormone
WBC	White blood cell count
WISC-R	Wechsler Intelligence Scale for Children–Revised

Chapter

1 Psychotic Disorders

Psychotic disorders are a collection of disorders in which **psychosis** predominates the symptom complex. Psychosis is defined as a gross impairment in reality testing. Specific psychotic symptoms include delusions, hallucinations, ideas of reference, and disorders of thought. Table 1-1 lists the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) classification of the psychotic disorders.

It is important to understand that psychotic disorders are different from mood disorders with **psychotic features**. Patients can present with a severe episode of depression and have delusions or with a manic episode with delusions and hallucinations. These patients do not have a primary psychotic disorder; rather, their psychosis is secondary to a mood disorder.

The diagnoses described below are among the most severely disabling of mental disorders. Disability is due in part to the extreme degree of social and occupational dysfunction associated with these disorders.

SCHIZOPHRENIA

Schizophrenia is a disorder in which patients have psychotic symptoms and social and/or occupational dysfunction that persists for at least 6 months.

Epidemiology

Schizophrenia affects 1% of the population. The typical age of onset is the early 20s for men and the late 20s for women. Women are more likely to have a "first break" later in life; in fact, about one third of women have an onset of illness after age 30. Schizophrenia is diagnosed disproportionately among the lower socioeconomic classes; although theories exist for this finding, none have been substantiated.

Etiology

The etiology of schizophrenia is unknown. There is a clear inheritable component, but familial incidence is sporadic and schizophrenia does occur in families with no history of the disease. Schizophrenia is widely believed to have a neurobiological basis. The most notable theory is the **dopamine hypothesis**, which posits that schizophrenia is due to hyperactivity in brain dopaminergic pathways. This theory is consistent with the efficacy of antipsychotics (which block dopamine receptors) and the ability of drugs (such as cocaine or amphetamines) that stimulate dopaminergic activity to induce psychosis. Postmortem studies also have shown higher numbers of dopamine receptors in specific subcortical nuclei of schizophrenics than in normal brains. More recent studies have focused on structural and functional abnormalities through brain imaging of schizophrenics and control populations. No one finding or theory to date is adequate in explaining the etiology and pathogenesis of this complex disease.

Clinical Manifestations

History and Mental Status Examination

Schizophrenia is a disorder characterized by what have been termed **positive** and **negative symptoms**, a pattern of **social and occupational deterioration**, and persistence of the illness for at least **6 months**. Positive symptoms are characterized by the **presence** of unusual thoughts, perceptions, and behaviors (e.g.,

TABLE 1-1

Psychotic Disorders

Schizophrenia	Brief psychotic disorder
Schizophreniform disorder	Shared psychotic disorder
Schizoaffective disorder	Delusional disorder

hallucinations, delusions, agitation); negative symptoms are characterized by the **absence** of normal social and mental functions (e.g., lack of motivation, isolation, anergia, and poor self-care). The positive versus negative distinction was made in a nosologic attempt to identify subtypes of schizophrenia and because some medications seem to be more effective in treating negative symptoms. Clinically, patients often exhibit both positive and negative symptoms at the same time. Table 1-2 lists common positive and negative symptoms.

To make the diagnosis, two (or more) of the following criteria must be met: hallucinations, delusions, disorganized speech, grossly disorganized or catatonic (mute and/or posturing) behavior, or negative symptoms. There must also be social and/or occupational dysfunction. The patient must be ill for at least 6 months.

Patients with schizophrenia generally have a history of abnormal premorbid functioning. The prodrome of schizophrenia includes poor social skills, social withdrawal, and unusual (although not frankly delusional) thinking. Inquiring about the premorbid history may help to distinguish schizophrenia from a psychotic illness secondary to mania or drug ingestion.

Schizophrenics are at high risk for suicide. Approximately one third will attempt suicide and 10% will complete suicide. Risk factors for suicide include male gender, age <30 years, chronic course, prior depression, and recent hospital discharge.

DSM-IV recognizes five subtypes of schizophrenia: paranoid, disorganized, catatonic, undifferentiated, and residual. The subtypes of schizophrenia are useful as descriptors but have not been shown to be reliable or valid. Table 1-3 describes these subtypes.

Differential Diagnosis

The differential diagnosis of an acute psychotic episode is broad and challenging (Table 1-4). Once a medical or substance-related condition has been ruled out, the task is to differentiate schizophrenia from a schizoaffective disorder, a mood disorder with psychotic features, a delusional disorder, or a personality disorder.

Management

Antipsychotic agents (also called neuroleptics) are primarily used in treatment. These medications are used to treat acute psychotic episodes and to maintain patients in remission or with chronic illness. Antipsychotic medications are discussed in Chapter 11. Combinations of several classes of medications are often prescribed in severe or refractory cases. **Psychosocial treatments**, including stable reality-

TABLE 1-2

Positive and Negative Symptoms of Schizophrenia

Negative symptoms	
Affective flattening	Decreased expression of emotion, such as lack of expressive gestures
Alogia	Literally "lack of words," including poverty of speech and of speech content in response to a question
Asociality	Few friends, activities, interests; impaired intimacy, little sexual interest
Positive symptoms	
Hallucinations	Auditory, visual, tactile, and/or olfactory hallucinations; voices that are commenting
Delusions	Often described by content; persecutory, grandiose, paranoid, religious; ideas of reference, thought broadcasting, thought insertion, thought withdrawal
Bizarre behavior	Aggressive/agitated, odd clothing or appearance, odd social behavior, repetitive-stereotyped behavior

Adapted from Andreasen NC, Black DW. Introductory textbook of psychiatry. 3rd ed. Washington, DC: American Psychiatric Publishing, 2001.

TABLE 1-3

Subtypes of Schizophrenia

Paranoid	Paranoid delusions, frequent auditory hallucination, affect *not* flat
Catatonic	Motoric immobility or excessive, purposeless motor activity, maintenance of a rigid echolalia
Disorganized	Disorganized speech, disorganized behavior, flat or inappropriate affect; not catatonic
Undifferentiated (probably most common)	Delusions, hallucinations, disorganized speech, catatonic behavior, negative symptoms *Criteria not met for paranoid, catatonic, or disorganized*
Residual	Met criteria for schizophrenia, now resolved, i.e., no hallucinations, no prominent delusions, etc., but *residual* negative symptoms or attenuated delusions, hallucinations, or thought disorder

Adapted from Andreasen NC, Black DW, Introductory textbook of psychiatry. 3rd ed. Washington, DC: American Psychiatric Publishing, 2001.

TABLE 1-4

Causes of Acute Psychotic Syndromes

- Major psychiatric disorders
  - Acute exacerbation of schizophrenia
  - Atypical psychoses (e.g., schizophreniform)
  - Depression with psychotic features
  - Mania
- Drug abuse and withdrawal
  - Alcohol withdrawal
  - Amphetamines and cocaine
  - Phencyclidine (PCP) and hallucinogens
  - Sedative-hypnotic withdrawal
- Prescription drugs
  - Anticholinergic agents
  - Digitalis toxicity
  - Glucocorticoids and adrenocorticotropic hormone (ACTH)
  - Isoniazid
  - L-Dopa and other dopamine agonists
  - Nonsteroidal anti-inflammatory agents
  - Withdrawal from MAOIs
- Other toxic agents
  - Carbon disulfide
  - Heavy metals
- Neurologic causes
  - AIDS encephalopathy
  - Brain tumor
  - Complex partial seizures
  - Early Alzheimer's or Pick's disease
  - Huntington's disease
  - Hypoxic encephalopathy
  - Infectious viral encephalitis
  - Lupus cerebritis
  - Neurosyphilis
  - Stroke
  - Wilson's disease
- Metabolic causes
  - Acute intermittent porphyria
  - Cushing's syndrome
  - Early hepatic encephalopathy
  - Hypo- and hypercalcemia
  - Hypoglycemia
  - Hypo- and hyperthyroidism
  - Paraneoplastic syndromes (limbic encephalitis)
- Nutritional causes
  - Niacin deficiency (pellagra)
  - Thiamine deficiency (Wernicke-Korsakoff syndrome)
  - Vitamin B_{12} deficiency

Reproduced with permission from Hyman SE, Arana GW, Rosenbaum JF. Handbook of psychiatric drug therapy. 4th ed. Philadelphia: Lippincott, Williams and Wilkins, 2000.

oriented psychotherapy, family support, psychoeducation, social and vocational skills training, and attention to details of living situation (housing, roommates, daily activities), are critical to the long-term management of these patients. Poorer prognosis occurs with early onset, a history of head trauma, or comorbid substance abuse.

KEY POINTS

1. Schizophrenia is characterized by psychosis and social/occupational dysfunction.
2. Symptoms must last for at least 6 months.
3. Schizophrenia has a 10% suicide rate (approximately one third attempt).
4. It is treated with antipsychotics and psychosocial support.

SCHIZOAFFECTIVE DISORDER

Patients with schizoaffective disorder have psychotic episodes that **resemble schizophrenia but with prominent mood disturbances**. Their psychotic symptoms, however, must persist for some time in the absence of any mood syndrome.

Epidemiology

Lifetime prevalence is estimated at 0.5% to 0.8%. Age of onset is similar to schizophrenia (late teens to early 20s). Schizoaffective patients are more likely than schizophrenics but less likely than mood-disordered patients to have a remission after treatment.

Etiology

The etiology of schizoaffective disorder is unknown. It may be a variant of schizophrenia, a variant of a mood disorder, a distinct psychotic syndrome, or simply superimposed mood disorder and psychotic disorder.

Clinical Manifestations

History and Mental Status Examination

Patients with schizoaffective disorder have the typical symptoms of schizophrenia and coincidentally a major mood disturbance, such as a manic or depressive episode. They must also have periods of illness in which they have psychotic symptoms **without a major mood disturbance**. Mood disturbances need to be present for a substantial portion of the illness.

There are two subtypes of schizoaffective disorder recognized in the DSM-IV, **depressive** and **bipolar**, which are determined by the nature of the mood-disturbance episodes.

Differential Diagnosis

Mood disorders with psychotic features, as in mania or psychotic depression, are different from schizoaffective disorder in that schizoaffective patients have persistence (for at least 2 weeks) of the psychotic symptoms after the mood symptoms have resolved. Schizophrenia is differentiated from schizoaffective disorder by the absence of a prominent mood disorder in the course of the illness.

It is important to distinguish the prominent negative symptoms of the schizophrenic from the **lack of energy** or **anhedonia** in the depressed patient with schizoaffective disorder. More distinct symptoms of a mood disturbance (such as depressed mood and sleep disturbance) should indicate a true coincident mood disturbance.

Management

Patients are treated with medications that target the psychosis and the mood disorder. Typically, these patients require the **combination of an antipsychotic medication and a mood stabilizer**. Mood stabilizers are described in Chapter 13. An antidepressant or electroconvulsive therapy may be needed for an acute depressive episode. Psychosocial treatments are similar for schizoaffective disorder and schizophrenia. Prognosis is better than for schizophrenia and worse than for bipolar disorder or major depression.

KEY POINTS

1. In schizoaffective disorder, there are mood disturbances *with* psychotic episodes and there are periods of psychosis *without* a mood disturbance.
2. Treatment is with antipsychotics and mood stabilizers.
3. The prognosis for schizoaffective disorder is better than for schizophrenia but worse than for a mood disorder.

SCHIZOPHRENIFORM DISORDER

Essentially, this is schizophrenia that fails to last for 6 months and does not involve social withdrawal.

Epidemiology

The validity of this diagnosis is under question. Outcome studies of this disorder indicate that most patients may go on to develop full-blown schizophrenia, whereas others appear to develop a mood disorder. The diagnosis of schizophreniform disorder may, however, help to avoid premature diagnosis of patients with schizophrenia before some other disorder, such as bipolar disorder, manifests itself.

Etiology

At this time, the etiology is unknown. At least one study found similarities in brain structure abnormalities between schizophrenics and those with schizophreniform disorder.

Clinical Manifestations

History and Mental Status Examination

Schizophreniform disorder is essentially **short-course schizophrenia** without the requirement of social withdrawal. Patients with this disorder have what appears to be a "full-blown" episode of schizophrenia, including delusions, hallucinations, disorganized speech, or negative symptoms, but the duration of illness including prodromal, active, and residual phases, is from 1 to 6 months. The diagnosis changes to schizophrenia once the symptoms have extended past 6 months, even if only residual symptoms are left.

Differential Diagnosis

Care must be taken to distinguish schizophreniform disorder from a manic or depressive episode with psychotic features. Other causes of an acute psychosis must be ruled out (substance-induced or due to a general medical condition).

Management

The disorder is by definition **self-limited**. When symptoms cause severe impairment, treatment is similar to that for the acute treatment of psychosis in schizophrenia.

KEY POINTS

1. Schizophreniform disorder resembles schizophrenia.
2. It resolves completely in less than 6 months.
3. It most often results in either schizophrenia or bipolar disorder.
4. It is self-limited.

DELUSIONAL DISORDER

Delusional disorder is characterized by nonbizarre delusions without other psychotic symptoms. It is rare, its course is chronic, and treatment is supportive.

Epidemiology

This disorder is rare, with a prevalence of <0.05%. Generally, onset is in middle to late life; it affects women more often than men. Its course is **chronic** and **unremitting**.

Etiology

The etiology is unknown. Often, psychosocial stressors appear to be etiologic, for example, following **migration**. In migration psychosis, the recently immigrated person develops persecutory delusions. Many patients with delusional disorder have a paranoid character premorbidly. Paranoid personality disorder has been found in families of patients with delusional disorder.

Clinical Manifestations

History and Mental Status Examination

This disorder is characterized by well-systematized **nonbizarre delusions** about things that could happen in real life (such as being followed, poisoned, infected, loved at a distance, having a disease, being deceived by one's spouse or significant other). The delusions must be present for at least 1 month. Other than the delusion, the patient's social adjustment may be normal.

The patient must not meet criteria for schizophrenia. Any mood disorder must be brief relative to the duration of the illness. The DSM-IV recognizes seven subtypes of delusional disorder (Table 1-5).

TABLE 1-5

Delusional Disorder Subtypes

Erotomanic	A person becomes falsely convinced that another person is in love with him or her.
Grandiose	A person becomes falsely convinced that he or she has special abilities or is in other ways much more important than reality indicates.
Jealous	A person becomes falsely convinced that his or her lover is unfaithful.
Persecutory	A person becomes falsely convinced that others are out to harm him or her and that he or she is being conspired against in general.
Somatic	A person becomes falsely convinced that he or she has a bodily function disorder, for example organ dysfunction, body odor, or parasite infection.
Mixed	Diagnosed when no single delusional theme predominates
Unspecified	Diagnosed when a single delusional theme cannot be determined or when the predominant delusional theme does not match subtype criteria

Differential Diagnosis

It is important to rule out other psychiatric or medical illnesses that could have caused the delusions. Thereafter, delusional disorder must be distinguished from major depression with psychotic features, mania, schizophrenia, and paranoid personality.

Management

Trials of antipsychotics are appropriate but are often ineffective. The primary treatment is **psychotherapy**, taking care to neither support nor refute the delusion but to maintain an **alliance** with the patient. Without such an alliance, most patients fall out of treatment; with an alliance, over time, the patient may relinquish the delusions.

KEY POINTS

1. Delusional disorder is characterized by nonbizarre delusions.
2. It is chronic and unremitting.
3. Treatment involves making a therapeutic alliance.

BRIEF PSYCHOTIC DISORDER

In brief psychotic disorder, the patient experiences a full psychotic episode that is **short-lived**. It can be temporally related to some stressor or occur postpartum, but is also seen **without any apparent antecedent**.

Epidemiology

There is insufficient data available to determine prevalence and sex ratio.

Etiology

Etiology is unknown. However, it seems to be associated with borderline personality disorder and schizotypal personality disorder.

Clinical Manifestations

History and Mental Status Examination

In brief psychotic disorder, the patient develops psychotic symptoms that last for at least 1 day but no more than 1 month, followed by eventual return to premorbid functioning. Patients can exhibit any combination of delusions, hallucinations, disorganized speech, or grossly disorganized behavior. There are three recognized subtypes: **with marked stressors** (formerly known as brief reactive psychosis), **without marked stressors**, and **postpartum**. Patients with the postpartum subtype typically develop symptoms within 1 to 2 weeks after delivery that resolve within 2 to 3 months.

Differential Diagnosis

It is important to rule out schizophrenia, especially if the disorder worsens or persists for more than a month (except for postpartum psychosis, which may last 2 to 3 months). A mood disorder such as mania or depression with psychotic features must be ruled out.

Management

Hospitalization may be necessary to protect the patient. Treatment with antipsychotics is common, although the condition is by definition self-limited and no specific treatment is required. The containing environment of the hospital milieu may be sufficient to help the patient recover.

KEY POINTS

1. Brief psychotic disorder is characterized by typical psychotic symptoms.
2. The condition is short-lived, lasting from 1 to 30 days.
3. It can be preceded by a stressor or can be postpartum.
4. It may occur without an antecedent.
5. The condition is self-limited.

Chapter

2 Mood Disorders

Mood disorders are among the most common diagnoses in psychiatry. Mood is a persistent emotional state (as differentiated from affect, which is the external display of feelings). There are three major categories of mood disorders according to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition: unipolar mood disorders (major depressive disorder, dysthymic disorder), bipolar mood disorders (bipolar I disorder, bipolar II disorder, and cyclothymic disorder), and mood disorders having a known etiology (substance-induced mood disorder and mood disorder due to a general medical condition) (Table 2-1).

The best available evidence suggests that mood disorders lie on a continuum with normal mood. Although mania and depression are often viewed as opposite ends of the mood spectrum, they can occur simultaneously in a single individual within a brief period, giving rise to the concept of mixed mood states.

UNIPOLAR DISORDERS

Unipolar disorders are major depressive disorder and dysthymic disorder.

Major Depressive Disorder

Major depressive disorder is diagnosed after a single episode of major depression (Table 2-2). It is characterized by emotional changes, primarily depressed mood, and by so-called vegetative changes, consisting of alterations in sleep, appetite, and energy levels.

Epidemiology

The lifetime prevalence (will occur at some point in a person's life) rate for major depressive disorder is 5% to 20%. The female-male ratio is 2:1. Race distributions appear equal, and socioeconomic variables do not seem to be a factor. The incidence (rate of new cases) is greatest between the ages of 20 and 40 and decreases after the age of 65.

Etiology

Psychological theories of depression generally view interpersonal losses (actual or perceived) as risk factors for developing depression. In fact, available evidence suggests that childhood loss of a parent or loss of a spouse are associated with depression. Classic psychoanalytic theories center on ambivalence toward the lost object (person), although more recent theories focus on the critical importance of the object relationship in maintaining psychic equilibrium and self-regard. The cognitive-behavioral model views cognitive distortions as the primary events that foster a negative misperception of the world, which in turn generate negative emotions. The learned helplessness model (based on animal studies) suggests that depression arises when individuals come to believe they have no control over the stresses and pains that beset them.

Biologic, familial, and genetic data support the idea of a biologic diathesis in the genesis of depression. Genetic studies show that depression is found to be concordant more often in monozygotic twins than in dizygotic twins. Unipolar depression in a parent leads to an increased incidence in the offspring of both unipolar and bipolar mood disorders.

Neurotransmitter evidence points to abnormalities in amine neurotransmitters as mediators of depressive states: The evidence is strongest for deficiencies in norepinephrine and serotonin.

Neuroendocrine abnormalities in the hypothalamic-pituitary-adrenal axis are often present in depression and suggest a neuroendocrine link.

TABLE 2-1

Classification of Mood Disorders

Unipolar	Bipolar	Etiologic
Major depressive disorder	Bipolar I disorder	Substance-induced mood disorder
Dysthymic disorder	Bipolar II disorder	Mood disorder due to general medical condition
	Cyclothymic disorder	

TABLE 2-2

Criteria for Major Depressive Episode

Mood: depressed mood most of the day, nearly every day

Sleep: insomnia or hypersomnia

Interest: marked decrease in interest and pleasure in most activities

Guilt: feelings of worthlessness or inappropriate guilt

Energy: fatigue or low energy nearly every day

Concentration: decreased concentration or increased indecisiveness

Appetite: increased or decreased appetite or weight gain or loss

Psychomotor: psychomotor agitation or retardation

Suicidality: recurrent thoughts of death, suicidal ideation, suicidal plan, suicide attempt

General criteria for a major depressive episode require five or more of the above symptoms to be present for at least 2 weeks; one symptom must be *depressed mood* or *loss of interest or pleasure*. These symptoms must be a change from prior functioning and cannot be due to a medical condition, cannot be substance-induced, and cannot be due to bereavement. The symptoms must also cause *distress or impairment*. Reprinted with permission from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Copyright 2000 American Psychiatric Association.

Sleep disturbances are near universal complaints in depressed persons. Objective evidence from sleep studies reveals that deep sleep (delta sleep, stages 3 and 4) is decreased in depression and that rapid-eye-movement (REM) sleep alterations include increased time spent in REM and earlier onset of REM in the sleep cycle (decreased latency to REM).

Clinical Manifestations

History and Mental Status Examination

A major depressive disorder is diagnosed if a patient has at least one episode of major depression and does not meet criteria for bipolar disorder or etiologic mood disorder. Major depression is characterized by emotional and vegetative changes. Emotional changes most commonly include depressed mood with feelings of sadness, hopelessness, guilt, and despair. Irritability may be the primary mood complaint in some cases. Vegetative symptoms include alterations in sleep, appetite, energy, and libido.

Major depression is frequently **recurrent**. The usual duration of an untreated episode (Fig. 2-1A) is 6 to 12 months. Fifteen percent of patients diagnosed with major depression die by suicide at some point in their lifetime.

Differential Diagnosis

Mood disorders secondary to (induced by) medical illnesses or substance abuse are the primary differential diagnoses. Psychotic depression must be differentiated from schizophrenia; negative symptoms of schizophrenia can mimic depression. Persons with major depression may eventually meet criteria for bipolar disorder.

Management

Depression is responsive to **psychotherapy** and **pharmacotherapy**. Milder cases may be treated with brief

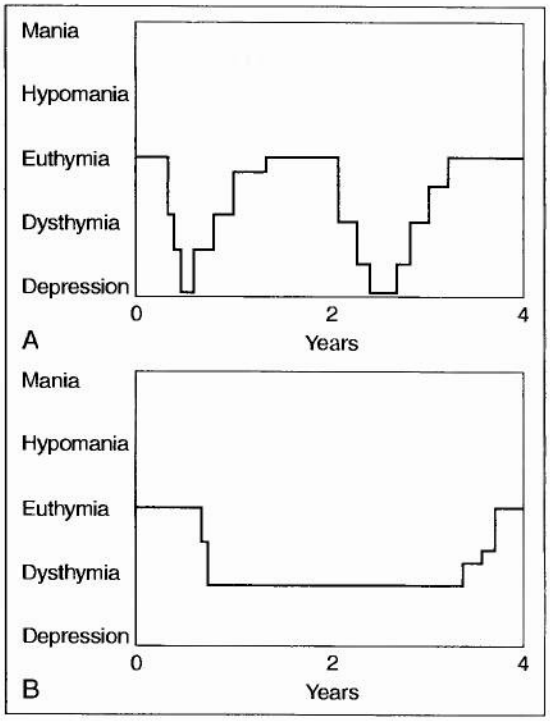

Figure 2-1 • Unipolar mood disorders. (A) Major depressive disorder and (B) dysthymic disorder.

psychotherapy interventions alone. For more severe cases, antidepressant medications combined with psychotherapy are superior to medications or psychotherapy alone. Among the psychotherapies, supportive, cognitive-behavioral, and brief interpersonal therapies have the most data to support their efficacy. There is a long tradition of psychodynamic psychotherapy in treating depression, although it has not been empirically well studied.

There are many classes of antidepressants available that are effective and are usually chosen according to side-effect profiles. Presently, available classes of antidepressants include tricyclic antidepressants, selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, and atypical antidepressants. In addition, lithium, thyroid hormone, and psychostimulants may be used as augmentative treatments. Electroconvulsive therapy (ECT) is used in psychotic, severe, or treatment refractory depressions or when medications are contraindicated (e.g., in the elderly or debilitated). Antipsychotic medications are an essential adjunct to antidepressants in psychotic depressions. Anxiolytics may be used as adjuncts to antidepressants in depression with high levels of anxiety, although more sedating antidepressants may suffice. Phototherapy can be used for seasonal mood disorders.

KEY POINTS

1. Major depression is a unipolar mood disorder.
2. It is often recurrent.
3. Major depression has a 15% suicide rate.
4. Combined psychotherapy and pharmacotherapy are the best treatment.

Dysthymic Disorder

Dysthymic disorder is a mild, chronic form of major depression.

Epidemiology

The lifetime prevalence is 6%.

Etiology

Because dysthymia is often conceptualized as a milder, chronic form of major depression, similar etiologies are generally attributed to dysthymia.

Clinical Manifestations

History and Mental Status Examination

Dysthymic disorder is a chronic and less severe form of major depression. The diagnosis of dysthymia requires a minimum of 2 years of chronically depressed mood most of the time (Fig. 2-1B). Associated symptoms and complaints may include change in appetite and sleep, fatigue, decreased concentration, and hopelessness. Dysthymia can be chronic and difficult to treat. At times, major depressive episodes may co-occur, giving rise to the term double depression.

Differential Diagnosis

Major depression and etiologic mood disorders are the major differential diagnostic considerations.

Management

Treatment is similar to major depression except that psychotherapy may play a larger role and the course of treatment may be more protracted.

KEY POINTS

1. Dysthymia is a unipolar mood disorder.
2. It is chronic, lasting at least 2 years.
3. It is often treatment refractory.

BIPOLAR DISORDERS

The bipolar disorders are bipolar I disorder, bipolar II disorder, and cyclothymia.

Bipolar I Disorder

Bipolar I disorder is the most serious of the bipolar disorders and is diagnosed after at least one episode of mania (Table 2-3). Patients with bipolar I disorder typically also have major depressive episodes in the course of their lives.

Epidemiology

The lifetime prevalence is 0.4% to 1.6% and the male-female ratio is equal. There are no racial variations in incidence.

Etiology

Genetic and familial studies reveal that bipolar I disorder is associated with increased bipolar I, bipolar II, and major depressive disorders in first-degree rel-

TABLE 2-3

Criteria for Manic Episode

Three to four of the following criteria are required during the elevated mood period:

Self-esteem: highly inflated, grandiosity

Sleep: decreased need for sleep, rested after only a few hours

Speech: pressured

Thoughts: racing thoughts and flight of ideas

Attention: easy distractibility

Activity: increased goal-directed activity

Hedonism: high excess involvement in pleasurable activities (sex, spending, travel)

General criteria for a manic episode require a clear period of persistently *elevated, expansive,* or *irritable mood* lasting 1 week or severe enough to require hospitalization. These symptoms must be a change from prior functioning and cannot be due to a medical condition and cannot be substance-induced. The symptoms also must cause distress or impairment.

Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Washington, DC: American Psychiatric Association, 2000.

atives. X linkage has been demonstrated in some studies but remains controversial. Mania can be precipitated by psychosocial stressors, and there is evidence that sleep/wake cycle perturbations may predispose a person to mania.

Clinical Manifestations

History and Mental Status Examination

Bipolar I disorder is defined by the occurrence of mania (or a mixed episode). A single manic episode is sufficient to meet diagnostic requirements; most patients, however, have recurrent episodes of mania typically intermixed with depressive episodes. The criteria for a manic episode are outlined in Table 2-3.

The first episode of mania usually occurs in the early 20s. Manic episodes are typically briefer than depressive episodes. The transition between mania and depression occurs without an intervening period of euthymia in about two of three patients (Fig. 2-2A). Lifetime suicide rates range from 10% to 15%.

Differential Diagnosis

Mania may be induced by antidepressant treatment, including antidepressant medications, psychostimulants, ECT, and phototherapy. When this occurs, the patient is diagnosed with substance-induced mood disorder, not bipolar disorder. Mood disorder due to a general medical condition is the other major differential consideration. Schizoaffective disorder, borderline personality disorder, and depression with agitation are also considerations.

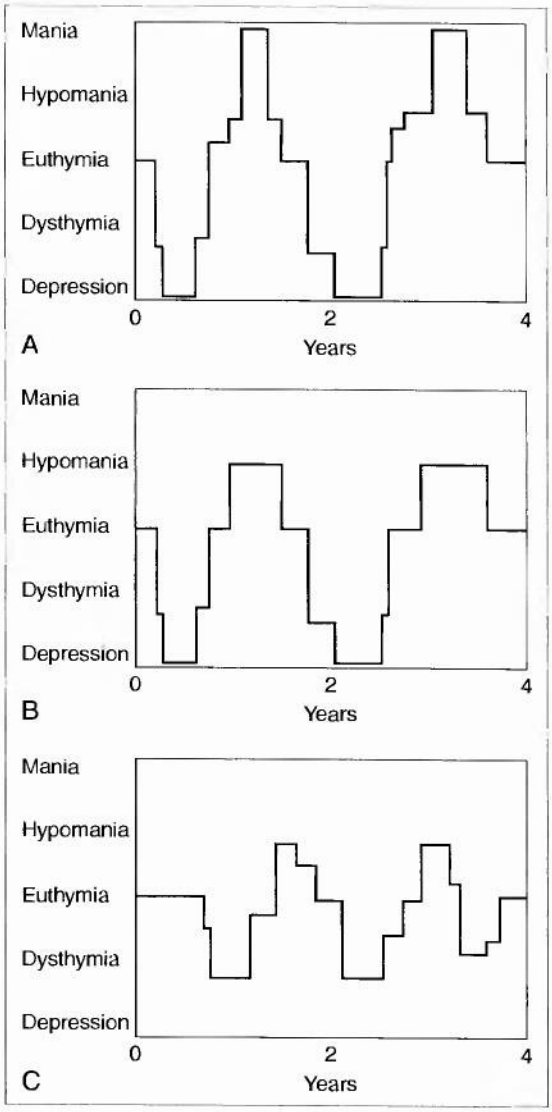

Figure 2-2 • Bipolar mood disorders. (A) bipolar I disorder, (B) bipolar II disorder, (C) cyclothymic disorder.

Management

Persons experiencing a manic episode often have poor insight and resist treatment. Pharmacological interventions for acute mania include **antipsychotics** in conjunction with **benzodiazepines** (for rapid tranquilization) and initiation of **mood stabilizer** medication. Antipsychotics are frequently used in mania

with and without psychotic features. Lithium is the most commonly used mood stabilizer, but valproic acid is quite effective and is more effective for the rapid-cycling variant of mania. Carbamazepine, lamotrigine, gabapentin, and long-acting benzodiazepines are used if first-line treatments fail. ECT is used in patients with medication intolerance and where a more immediate response is medically or psychiatrically needed.

Mood stabilizer maintenance therapy is essential in preventing the recurrence of mania and appears to decrease the recurrence of depression. Psychotherapy is used to encourage medication compliance, to help patients come to terms with their illness, and to help repair some of the interpersonal damage done while ill (e.g., infidelity, hostility, squandering money). Care must be taken when prescribing antidepressants for depression or dysthymia because of their role in prompting more severe or more frequent manic episodes.

KEY POINTS

1. Bipolar I disorder is a biphasic mood disorder.
2. It is cyclic.
3. It has a suicide rate of 10% to 15%.
4. Maintenance treatment with mood stabilizers is required.

Bipolar II Disorder

Bipolar II disorder is similar to bipolar I disorder except that mania is absent in bipolar II disorder and **hypomania** (a milder form of elevated mood than mania) is the essential diagnostic finding.

Epidemiology

Lifetime prevalence is about 0.5%. Bipolar II disorder may be more common in women.

Etiology

Current evidence implicates the same factors as for bipolar I.

Clinical Manifestations

History and Mental Status Examination

Bipolar II disorder is characterized by the occurrence of hypomania and episodes of major depression in an individual who has never met criteria for mania or a mixed state. Hypomania is determined by the same symptom complex as mania but the symptoms are less severe, cause less impairment, and usually do not require hospitalization. Bipolar II disorder is **cyclic** (see Fig. 2-2B for course of untreated bipolar II). Suicide occurs in 10% to 15%.

Differential Diagnosis

Same as for bipolar I.

Management

Treatment is the same as for bipolar I disorder, although hypomanic episodes typically do not require as aggressive a treatment regimen as mania. Care must be taken in prescribing antidepressants for depression or dysthymia because of their role in prompting more severe or frequent hypomanic episodes.

KEY POINTS

1. Bipolar II disorder is a biphasic mood disorder with hypomania.
2. It is recurrent.
3. It has a suicide rate of 10% to 15%.

Cyclothymic Disorder

Cyclothymic disorder is a recurrent, chronic, mild form of bipolar disorder in which mood typically oscillates between hypomania and dysthymia. It is not diagnosed if a person has had either a manic episode or a major depressive episode.

Epidemiology

The lifetime prevalence of cyclothymic disorder is 0.4% to 1%. The rate appears equal in men and women, though women more often seek treatment.

Etiology

Familial and genetic studies reveal an association with other mood disorders.

Clinical Manifestations

History and Mental Status Examination

Cyclothymic disorder is a milder form of bipolar disorder consisting of **recurrent mood disturbances** between hypomania and dysthymic mood. A single episode of hypomania is sufficient to diagnose cyclothymic disorder; however, most individuals also have dysthymic periods. The diagnosis of

cyclothymic disorder is never made when there is a history of mania or major depressive episode or mixed episode. The course of untreated cyclothymic disorder is depicted in Figure 2-2C.

Differential Diagnosis

The principal differential is among other unipolar and bipolar mood disorders, substance-induced mood disorder, and mood disorder due to a general medical condition. Personality disorders (especially borderline) with labile mood may be confused with cyclothymic disorder.

Management

Psychotherapy, mood stabilizers, and antidepressants are used. However, persons with cyclothymia may never seek medical attention for their mood symptoms.

KEY POINTS

1. Cyclothymic disorder is a biphasic mood disorder without frank mania or depression.
2. It is chronic and recurrent.

MOOD DISORDERS WITH KNOWN ETIOLOGY

Substance-Induced Mood Disorder

Substance-induced mood disorder is diagnosed when medications, other psychoactive substances, ECT, or phototherapy are proximate events and the likely cause of the mood disturbance. All aforementioned types of mood disorder (e.g., unipolar, bipolar) may occur.

Mood Disorder Due to a General Medical Condition

This category is for mood disturbances apparently caused by a medical illness. Endocrine disorders, such as thyroid and adrenal dysfunction, are common etiologies. Postpartum mood disorders are excluded from the criteria; they are modifiers of unipolar and bipolar mood disorders (see above).

SUBTYPES AND MODIFIERS

Various diagnostic specifiers can be applied to specific subtypes of mood disorders. These have prognostic and treatment implications and may prove to have etiologic implications.

Melancholic: Melancholic depression is a severe form of depression associated with guilt, remorse, loss of pleasure, and extreme vegetative symptoms.

Postpartum: Postpartum depression occurs within 4 weeks of delivery. The presence of one episode of postpartum mood disorder is strongly predictive of a recurrence.

Seasonal: Seasonal mood disorders show a consistent seasonal pattern of variation. The most common pattern is a worsening of depression during the fall and winter with improvement in the spring. The reverse is sometimes true. If the depression is a component of a bipolar disorder, the manic and hypomanic episodes may show a seasonal association.

Atypical: Atypical depressions show a pattern of hypersomnia, increased appetite or weight gain, mood reactivity, long-standing rejection sensitivity, anergia, and leaden paralysis.

Rapid Cycling: Patients with bipolar disorder may have frequent (rapid) cycles. To meet criteria for rapid cycling, four mood disturbances per year must be present. The suicide rate may be higher than in non-rapid cyclers.

Catatonic: The catatonic specifier is applied to mood disorders when there are pronounced movement abnormalities, including motoric immobility or excessive purposeless motor activity, maintenance of a rigid posture, mutism, stereotyped movement, echolalia (repetition of a word or phrase just spoken by another person), or echopraxia (repetition of movements made by another person).

Chapter

3 Anxiety Disorders

The term **anxiety** refers to many states in which the sufferer experiences a sense of impending threat or doom that is not well defined or realistically based. Anxiety can be adaptive or pathologic, transient or chronic, and has a variety of psychological and physical manifestations. **Anxiety disorders** are a heterogeneous group of disorders in which the feeling of anxiety is the major element. They are the most prevalent group of psychiatric disorders; according to the Epidemiological Catchment Area study, 7.3% of all Americans meet the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition (DSM-III; the DSM version used at the time) criteria at a given point in time (so-called point prevalence). Anxiety disorders listed in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) are shown in Table 3–1.

PANIC DISORDER AND AGORAPHOBIA

Panic disorder is characterized by recurrent unexpected panic attacks that can occur *with or without* agoraphobia. Agoraphobia is a disabling condition in which patients fear places in which escape might be difficult. Whether occurring as distinct disorders or together, panic disorder and agoraphobia are common, sometimes disabling, conditions.

Epidemiology

Panic disorder occurs more frequently in women, with a lifetime prevalence of 2% to 3%. The typical onset is in the 20s, with most cases beginning before age 30.

Agoraphobia also occurs more frequently in women, with a lifetime prevalence of between 2% and 6%. Only one third of patients with agoraphobia also have panic disorder. However, most patients with agoraphobia seen clinically also have panic disorder. This apparent contradiction is due to the fact that patients with agoraphobia alone are unlikely to seek treatment.

Etiology

The etiology of panic disorder is unknown. There are several popular biologic theories involving carbon dioxide (CO_2) hypersensitivity, abnormalities in lactate metabolism, an abnormality of the locus coeruleus (a region in the brain that regulates level of arousal), and elevated central nervous system catecholamine levels. The gamma-amino butyric acid (GABA) receptor also has been implicated as etiologic since patients respond well to benzodiazepines and panic is induced in patients with anxiety disorders using GABA antagonists.

Theorists posit that panic attacks are a conditioned response to a fearful situation. For example, a person has an automobile accident and experiences severe anxiety, including palpitations. Thereafter, palpitations alone, experienced during exercise or any sympathetic nervous system response, may induce the conditioned response of a panic attack.

Clinical Manifestations

History and Mental Status Examination

Panic disorder is characterized by **recurrent unexpected panic attacks** that can occur with or without

TABLE 3-1

Anxiety Disorders

Panic disorder *with agoraphobia*
Panic disorder *without agoraphobia*
Agoraphobia
Social phobia
Specific phobia
Obsessive-compulsive disorder
Generalized anxiety disorder
Acute stress disorder
Posttraumatic stress disorder
Substance-induced anxiety disorder
Anxiety disorder due to a general medical condition
Anxiety disorder not otherwise specified

agoraphobia (see below). Panic attacks typically come on suddenly, peak within minutes, and last 5 to 30 minutes. The patient must experience 4 of 13 typical symptoms of panic outlined in Table 3-2.

To warrant the diagnosis, one of the following must occur for at least 1 month: persistent concern about having additional attacks, worry about the implications of the attack (losing control, "going crazy"), or a significant change of behavior related to the attacks (e.g., restriction of activities).

Agoraphobia is a disabling complication of panic disorder but can also occur in patients with no history of panic disorder. It is characterized by an **intense fear of places or situations** in which escape might be difficult (or embarrassing). Patients with agoraphobia and panic disorder typically fear having a panic attack in a public place and being embarrassed or unable to escape. Those with agoraphobia alone (two thirds of those with agoraphobia) simply avoid public arenas but do not have panic attacks. Although some agoraphobic patients are so disabled that they are homebound, many are comforted by the presence of a companion, allowing them to enter some public places with less anxiety.

Differential Diagnosis

Panic attacks should be distinguished from the direct physiologic effects of a substance or a general medical condition. The panic attacks also cannot be accounted for by another mental disorder (such as social phobia or obsessive-compulsive disorder [OCD]).

TABLE 3-2

DSM-IV Criteria for Panic Attack

A discrete period of intense fear or discomfort, in which four (or more) of the following symptoms developed abruptly and reached a peak within 10 minutes:

- Palpitations, pounding heart, or accelerated heart rate
- Sweating
- Trembling or shaking
- Sensations of shortness of breath or smothering
- Feeling of choking
- Chest pain or discomfort
- Nausea or abdominal distress
- Feeling dizzy, unsteady, lightheaded, or faint
- Derealization (feelings of unreality) or depersonalization (being detached from oneself)
- Fear of losing control or going crazy
- Fear of dying
- Paresthesias
- Chills or hot flushes

Adapted from Diagnostic and statistical manual of mental disorders, 4th ed. Washington, DC: American Psychiatric Association, 1994:395.

Management

Not surprisingly, the main treatments for panic disorder are pharmacotherapy and cognitive-behavioral therapy or their combination. Specific **tricyclic antidepressants** (TCAs), specific **monoamine oxidase inhibitors** (MAOIs), **selective serotonin reuptake inhibitors** (SSRIs), and high-potency **benzodiazepines** have been shown to be effective in controlled studies. **Cognitive-behavioral therapy (CBT)** involves the use of relaxation exercises and desensitization combined with education aimed at helping patients to understand that their panic attacks are a result of misinterpreting bodily sensations. Patients can then learn that the sensations are innocuous and self-limited, which diminishes the panicky response. **Exposure therapy**, in which the patient incrementally confronts a feared stimulus, has been shown to be effective in treating agoraphobia.

KEY POINTS

1. Panic disorder is characterized by recurrent unexpected panic attacks.
2. Panic disorder can be seen with or without agoraphobia.
3. Panic disorder is treated with antidepressants and benzodiazepines and cognitive-behavioral techniques.
4. Agoraphobia is fear of not being able to (or being too embarrassed to) escape a place or situation.
5. Agoraphobia most often occurs alone (without panic).
6. Agoraphobia can be a complication of panic disorder.
7. Agoraphobia is treated with exposure therapy.

SPECIFIC PHOBIA

Specific phobia is an anxiety disorder characterized by intense fear of particular objects or situations (e.g., snakes, heights). It is the most common psychiatric disorder.

Epidemiology

Specific phobias are more prevalent in women than men and occur with a lifetime prevalence of 25%. Typical onset is in childhood, with most cases occurring before age 12.

Etiology

Phobic disorders, including specific phobia, tend to run in families. Behavioral theorists argue that phobias are learned by being paired with traumatic events.

Clinical Manifestations

History and Mental Status Examination

A phobia is an irrational fear of a specific object, place, activity, or situation that is out of proportion to any actual danger. To meet the DSM-IV criteria for specific phobia, a patient must experience a marked, persistent fear that is recognized by the patient to be excessive or unreasonable and is cued by the presence or anticipation of a specific object or situation. In addition, exposure to the stimulus must almost invariably provoke the anxiety reaction, and the avoidance of or distress over the feared situation must impair everyday activities or relationships. For those younger than age 18, symptoms must persist for at least 6 months.

Differential Diagnosis

The principal differential diagnosis is another mental disorder (such as avoidance of school in separation anxiety disorder) presenting with anxiety or fearfulness.

Management

Specific childhood phobias tend to remit spontaneously with age. When they persist into adulthood, they often become chronic. However, they rarely cause disability. Exposure therapy in the form of **systematic desensitization** or **flooding** is the treatment of choice. There is no role for medication.

KEY POINTS

1. Specific phobia is an intense fear of a certain object, place, activity, or situation.
2. It occurs in 25% of the population at some point in their lifetime, usually with onset before age 12.
3. It is treated with systematic desensitization and flooding.

SOCIAL PHOBIA

Social phobia is an anxiety disorder in which patients have an intense fear of being scrutinized in social or public situations (e.g., giving a speech, speaking in class). The disorder may be **generalized** or **limited** to specific situations.

Epidemiology

Social phobias occur equally among men and women and affect 3% to 5% of the population. The typical onset is in adolescence, with most cases occurring before age 25.

Etiology

Phobic disorders, including social phobia, tend to run in families. Behavioral theorists argue that phobias

are learned by being paired with traumatic events. Some theorists posit that hypersensitivity to rejection is a psychological antecedent of social phobia.

Clinical Manifestations

History and Mental Status Examination

Social phobias are characterized by the fear of situations in which the person is exposed to unfamiliar people or to possible scrutiny by others. Exposure to the feared social situation must almost invariably provoke an anxiety reaction. Avoidance of or distress over the feared situation must impair everyday activities or relationships. For those younger than age 18, symptoms must persist for at least 6 months. Social phobia can either be generalized (the patient fears nearly all situations) or limited to specific situations.

Differential Diagnosis

The principal differential diagnosis is another mental disorder (such as avoidance of school in separation anxiety disorder) presenting with anxiety or fearfulness.

Management

Mild cases of social phobia can be treated with CBT, but many cases require medication. MAOIs, beta-blockers, SSRIs, alprazolam, and gabapentin have proven successful in treating social phobia. CBT uses the exposure therapy techniques of flooding and systematic desensitization to reduce anxiety in feared situations. Supportive individual and group psychotherapy is helpful to restore self-esteem and to encourage venturing into feared situations.

KEY POINTS

1. Social phobia is fear of exposure to scrutiny by others.
2. It has a lifetime prevalence of 3% to 5%, typically occurring before age 25.
3. It can be generalized or limited.
4. It is treated with MAOIs, beta-blockers, SSRIs, alprazolam, or gabapentin and with CBT.

GENERALIZED ANXIETY DISORDER (GAD)

GAD is characterized by intense pervasive worry over virtually every aspect of life associated with **physical manifestations** of anxiety.

Epidemiology

The lifetime prevalence of GAD is approximately 5%. The typical age of onset is in the early 20s, but the disorder may begin at any age.

Etiology

Twin studies suggest that GAD has both inherited and environmental etiologies. Serotonergic, noradrenergic, and GABA-ergic neurotransmitter systems have been studied in relation to GAD, but the biologic etiology remains obscure. Cognitive-behavioral theorists posit that GAD is due to cognitive distortions in which patients misperceive situations as dangerous when they are not.

Clinical Manifestations

History and Mental Status Examination

Patients with generalized anxiety disorder worry excessively about virtually every aspect of their lives (job performance, health, marital relations, and social life). They do not have panic attacks, phobias, obsessions, or compulsions; rather, they experience pervasive anxiety and worry (apprehensive expectation) about a number of events or activities that occur most days for at least 6 months. They must also have difficulty controlling the worry, and it must be associated with at least three of the following symptoms: restlessness, easily fatigued, difficulty concentrating or mind going blank, irritability, muscle tension, and sleep disturbance.

Differential Diagnosis

The focus of the anxiety and worry in GAD must not be symptomatic of another axis I disorder. For example, the anxiety and worry cannot be about having a panic attack (as in panic disorder) or being embarrassed in public (as in social phobia).

Management

The pharmacologic treatment of GAD is with **benzodiazepines, buspirone** (a non-benzodiazepine anxiolytic), **gabapentin**, or **beta-blockers**. Although benzodiazepines are very effective, the duration of treatment is limited by the risk of tolerance and dependence. **Relaxation techniques** are also used in treatment with some success.

KEY POINTS

1. GAD is intense worry over every aspect of life.
2. GAD is characterized by difficulty controlling the worry.
3. It is associated with physical manifestations of anxiety.
4. GAD is treated with benzodiazepine, buspirone, beta-blockers, gabapentin, and relaxation techniques.

POSTTRAUMATIC STRESS DISORDER (PTSD)

PTSD is an anxiety disorder characterized by the **persistent reexperience of a trauma, efforts to avoid recollecting the trauma**, and **hyperarousal** (Table 3-3).

Epidemiology

The prevalence of PTSD is estimated at 0.5% among men and 1.2% among women. PTSD may occur at any age from childhood through adulthood and may begin hours, days, or even years after the initial trauma.

Etiology

The central etiologic factor in PTSD is the trauma. There may be some necessary predisposition to PTSD because not all people who experience similar traumas develop the syndrome. Magnetic resonance imaging studies support a finding of altered hippocampal volume in PTSD.

TABLE 3-3

Posttraumatic Stress Disorder
Reexperience of the trauma
Efforts to avoid recollection of the trauma
Hyperarousal

Clinical Manifestations

History and Mental Status Examination

People with PTSD have endured a traumatic event (e.g., combat, physical assault, rape, explosion) in which they experienced, witnessed, or were confronted with actual or potential death, serious physical injury, or a threat to physical integrity. The traumatic event is subsequently reexperienced through repetitive intrusive images or dreams or through recurrent illusions, hallucinations, or flashbacks of the event. In an adaptive attempt, these patients make efforts to avoid recollections of the event, often through psychological mechanisms (e.g., dissociation, numbing) or actual avoidance of circumstances that will evoke recall. They also experience feelings of detachment from others and exhibit evidence of autonomic hyperarousal (e.g., difficulty sleeping, exaggerated startle response).

Differential Diagnosis

Symptoms that resemble PTSD may be seen in depression, GAD, panic disorder, OCD, and dissociative disorder. When symptoms resemble PTSD, verify that there also are symptoms from all three categories; if not, consider one of the above diagnoses.

Management

Treatment is with a combination of **symptom-directed** psychopharmacologic agents and **psychotherapy** (individual or group). TCAs and MAOIs are the most commonly used medications in PTSD, especially when there is a comorbid major depression. SSRIs have also been used. Psychotherapy is typically tailored to the nature of the trauma, degree of coping skills, and the support systems available to the patient.

KEY POINTS

1. PTSD occurs in response to trauma.
2. PTSD is characterized by reexperience of the trauma, efforts to avoid recalling the trauma, and hyperarousal.
3. It is treated with medications directed at specific symptoms and with psychotherapy.

OBSESSIVE-COMPULSIVE DISORDER (OCD)

OCD is an anxiety disorder in which patients experience recurrent obsessions and compulsions that cause significant distress and occupy a significant portion of their lives.

Epidemiology

The lifetime prevalence of OCD is 2% to 3%. Typical onset of the disorder is between the late teens and early 20s, but one third of patients show symptoms of OCD before age 15.

Etiology

Behavioral models of OCD claim that obsessions and compulsions are produced and sustained through classic and operant conditioning. Interestingly, OCD is seen more frequently after brain injury or disease (e.g., head trauma, seizure disorders, Huntington's disease), and twin studies show that monozygotic twins have a higher concordance rate than dizygotic twins; these findings support a biologic basis for the disorder. The neurotransmitter **serotonin** has been implicated as a mediator in obsessive thinking and compulsive behaviors.

Clinical Manifestations

History and Mental Status Examination

Patients with OCD experience obsessions and compulsions. **Obsessions** are recurrent intrusive ideas, thoughts, or images that cause significant anxiety and distress; **compulsions** are repetitive purposeful physical or mental actions that are generally performed in response to obsessions. The compulsive "rituals" are meant to neutralize the obsessions, diminish anxiety, or somehow magically prevent a dreaded event or situation.

Differential Diagnosis

It is important to distinguish the *obsessional* thinking of OCD from the *delusional* thinking of schizophrenia or other psychotic disorders. Obsessions are usually unwanted, resisted, and recognized by patients as coming from their own thoughts, whereas delusions are generally regarded as distinct from patients' thoughts and are typically not resisted. For example, patients with depression often experience obsessive ruminations that can be distinguished from obsessions because they are transient, not considered unwanted, and not resisted.

Management

Clomipramine and SSRIs have been shown to be quite effective in treating OCD. Although poorly studied, the behavioural techniques of **systematic desensitization**, **flooding**, and **response prevention** have been used successfully to treat compulsive rituals. For example, someone who fears contamination from an object will hold the object repeatedly in therapy while simultaneously being prevented from carrying out the ritual associated with the dreaded object.

KEY POINTS

1. OCD is characterized by recurrent obsessions and compulsions.
2. OCD causes distress and wastes time by compelling patients to carry out various obsessions/compulsions/rituals.
3. Lifetime prevalence is 2% to 3%.
4. It is treated with clomipramine and SSRIs, and with systematic desensitization, flooding, and response prevention.

Chapter

4 Personality Disorders

Personality disorders are coded on Axis II in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV). Ten types of personality disorders are grouped into clusters based on similar overall characteristics. There are three recognized personality disorder clusters: **odd and eccentric, dramatic and emotional**, and **anxious and fearful** (Table 4–1).

DSM-IV general diagnostic criteria for personality disorders are outlined in Table 4–2. Criteria for individual personality disorders are discussed below. Personality disorders frequently overlap in symptoms.

CLUSTER A (ODD AND ECCENTRIC)

Paranoid Personality Disorder

People with paranoid personality disorder are distrustful and suspicious and anticipate harm and betrayal.

Epidemiology

Paranoid personality disorder has a lifetime prevalence of 0.5% to 2.5% of the general population. Relatives of chronic schizophrenics and patients with persecutory delusional disorders show an increased prevalence of paranoid personality disorder.

Etiology

Environmental precursors are unclear. Family studies suggest a link to delusional disorder (paranoid type). There appears to be a small increase in prevalence among relatives of schizophrenics.

Clinical Manifestations

History and Mental Status Examination

People with paranoid personality disorder are distrustful, suspicious and see the world as malevolent. They anticipate harm, betrayal, and deception. Not surprisingly, they are not forthcoming about themselves. They require emotional distance.

Differential Diagnosis

The key distinction is to separate paranoia associated with psychotic disorders from paranoid personality disorder, especially because paranoia associated with psychotic disorders is generally responsive to antipsychotic medications.

Schizoid Personality Disorder

Individuals with schizoid personality disorder are emotionally detached and prefer to be left alone.

Epidemiology

Estimates of lifetime prevalence range as high as 7.5% of the general population, but because people with schizoid personality disorder are avoidant of others, they are not commonly seen in clinical practice.

Etiology

There is some evidence to suggest increased prevalence of schizoid personality disorder in relatives of persons with schizophrenia or schizotypal personality disorder. Unloving or neglectful parenting is hypothesized to play a role.

Clinical Manifestations

History and Mental Status Examination

These people are loners. They are aloof and detached and have profound difficulty experiencing or expressing emotion. Although they prefer to be left alone and generally do not seek relationships, they

TABLE 4-1

Personality Disorders 1 Classification of Personality Disorders

Cluster A (Odd/Eccentric)	Cluster B (Dramatic/ Emotional)	Cluster C (Anxious/Fearful)
Paranoid	Antisocial	Avoidant
Schizoid	Borderline	Dependent
Schizotypal	Histrionic	Obsessive-compulsive
	Narcissistic	

TABLE 4-2

General Diagnostic Criteria for Personality Disorders

Personality disorder patients evidence an enduring pattern of inner experience and behavior, established by adolescence or early adulthood, that:

1. Deviates markedly from cultural expectations
2. Is inflexible and personally and socially pervasive
3. Causes distress or social or work impairment
4. Is a stable pattern of experience and behavior of long duration ("stably unstable")
5. Cannot be explained by another mental illness
6. Is not caused by substance use or medical condition

NOTE: Individuals with personality disorders usually maintain intact reality testing. However, they may have transient psychotic symptoms when stressed by real (or imagined) loss or frustration. Personality disorders are different from personality traits that are typically adaptive, culturally acceptable, and do not cause significant distress or impairment.

Source: Diagnostic and statistical manual of mental disorders, 4th ed. Washington, DC: American Psychiatric Association, 1994:327.

may maintain an important bond with a family member.

Differential Diagnosis

Schizoid personality disorder can be distinguished from avoidant personality disorder (see below) and social phobia by the fact that schizoid individuals do not desire relationships. Avoidant and socially phobic persons desire and may seek relationships, but their anxiety handicaps their capacity to achieve relatedness. Schizophrenia, autistic disorder, and Asperger's disorder (a less severe variant of autism) are also differential diagnostic conditions.

Schizotypal Personality Disorder

Individuals with schizotypal personality disorder have odd thoughts, affects, perceptions, and beliefs.

Epidemiology

Lifetime prevalence is 3% of the general population.

Etiology

Studies demonstrate interfamilial aggregation of this disorder, especially among first-degree relatives of schizophrenics.

Clinical Manifestations

History and Mental Status Examination

Schizotypal personality disorder is best thought of as similar to schizophrenia but less severe and without sustained psychotic symptoms. People with this disorder have few relationships and demonstrate oddities of thought, affect, perception, and belief. Many are highly distrustful and often paranoid, which results in a very constricted social world. The lifetime suicide rate among this population is 10%.

Differential Diagnosis

Schizophrenia, delusional disorder, and mood disorder with psychosis are the major differential diagnoses.

CLUSTER B (DRAMATIC AND EMOTIONAL)

Antisocial Personality Disorder (ASP)

Individuals with ASP repetitively disregard the rules and laws of society and rarely experience remorse for their actions.

Epidemiology

Antisocial personality disorder is present in 3% of men and 1% of women. About half have been arrested; about half of those in prison have ASP.

Etiology

ASP is more common among first-degree relatives of those diagnosed with ASP. In families of an individual with ASP, men show higher rates of ASP and substance abuse, whereas women have higher rates of somatization disorder. A harsh, violent, and criminal environment also predisposes people to this disorder.

Clinical Manifestations

History and Mental Status Examination

Individuals with ASP display either a flagrant or well-concealed disregard for the rules and laws of society. They are exploitative, lie frequently, endanger others, are impulsive and aggressive, and rarely experience remorse for the harm they cause others. Alcoholism is a frequently associated finding in this population. Many individuals with ASP are indicted or jailed for their actions. Their lifetime suicide rate is 5%.

Differential Diagnosis

Bipolar disorder and substance abuse disorder can prompt antisocial behaviors during the acute illness that remit when the disorder is controlled. The antisocial behavior of individuals with ASP, conversely, is not state dependent.

Borderline Personality Disorder

Individuals with borderline personality disorder suffer from instability in relationships, self-image, affect, and impulse control.

Epidemiology

Lifetime prevalence is 1% to 2% of the general population.

Etiology

Borderline personality disorder is about five times as common among first-degree relatives of borderline patients. In addition, this disorder shows increased rates in families of alcoholics and families of individuals with ASP, as well as in families with mood disorders. Females with borderline personality disorder frequently have suffered from sexual or physical abuse or both.

Clinical Manifestations

History and Mental Status Examination

Individuals with borderline personality disorder suffer from a legion of symptoms. Their relationships are infused with anger, fear of abandonment, and shifting idealization and devaluation. Their self-image is inchoate, fragmented, and unstable with consequent unpredictable changes in relationships, goals, and values. They are affectively unstable and reactive, with anger, depression, and panic prominent. Their impulsiveness can result in many unsafe behaviors, including drug use, promiscuity, gambling, and other risk-taking behavior. Their self-destructive urges result in frequent suicidal and parasuicidal behavior (such as superficial cutting or burning or nonfatal overdoses in which the intent is not lethal). They also demonstrate brief paranoia and dissociative symptoms. Suicide attempts can be frequent before the age of 30, and suicide rates approach 10% over a lifetime. The principal intrapsychic defenses they use are primitive with gross denial, distortion, projection, and splitting prominent. Patients may have a broad range of comorbid illnesses, including substance abuse, mood disorders, and eating disorders.

Differential Diagnosis

Mood disorders and behavioral changes due to active substance abuse are the principal differential diagnostic considerations. The diagnostic clues are unstable relationships, unstable self-image, unstable affect, and unstable or impulsive behaviors.

Histrionic Personality Disorder

Individuals with histrionic personality disorder have excessive superficial emotionality and a powerful need for attention.

Epidemiology

Lifetime prevalence is 2% to 3% of the general population. In clinical settings, the diagnosis is most frequently applied to women but may equally affect men in the general population.

Etiology

There appears to be a familial link to somatization disorder and to ASP.

Clinical Manifestations

History and Mental Status Examination

Individuals with histrionic personality disorder are characterized by their excessive and superficial emotionality and their profound need to be the center of attention at all times. Theatrical behavior dominates with lively and dramatic clothing, exaggerated emotional responses to seemingly insignificant events, and inappropriate flirtatious and seductive behavior across a wide variety of circumstances. Despite their apparent plethora of emotion, these individuals often have difficulty with intimacy, frequently believing their relationships are more intimate than they actually are.

Differential Diagnosis

Somatization disorder is the principal differential diagnostic consideration.

Narcissistic Personality Disorder

Individuals with narcissistic personality disorder appear arrogant and entitled but suffer from extremely low self-esteem.

Epidemiology

Lifetime prevalence is estimated at 1% in the general population and 2% to 16% in clinical populations. Fifty to 75% of those with this diagnosis are men.

Etiology

The etiology of this disorder is unknown.

Clinical Manifestations

History and Mental Status Examination

People with narcissistic personality disorder demonstrate an apparently paradoxical combination of self-centeredness and worthlessness. Their sense of self-importance is generally extravagant, and they demand attention and admiration. Concern or empathy for others is typically absent. They often appear arrogant, exploitative, and entitled. However, despite their inflated sense of self, below their brittle facade lies low self-esteem and intense envy of those whom they regard as more desirable, worthy, or able.

Differential Diagnosis

The grandiosity of narcissism can be differentiated from the grandiosity of bipolar disorder by the presence of characteristic mood symptoms in bipolar disorder.

CLUSTER C (ANXIOUS AND FEARFUL)

Avoidant Personality Disorder

Individuals with avoidant personality disorder desire relationships but avoid them because of the anxiety produced by their sense of inadequacy.

Epidemiology

Lifetime prevalence is 0.5% to 1% of the general population and appears to be of equal prevalence in men and women.

Etiology

There are no conclusive data. The pattern of avoidance may start in infancy.

Clinical Manifestations

History and Mental Status Examination

People with avoidant personality disorder experience intense feelings of inadequacy. They are painfully sensitive to criticism, so much so that they are compelled to avoid spending time with people. Their fears of rejection and humiliation are so powerful that to engage in a relationship they seek strong guarantees of acceptance. The essence of this disorder is inadequacy, hypersensitivity to criticism, and consequent social inhibition.

Differential Diagnosis

The major diagnostic distinction is between avoidant personality disorder and social phobia, generalized type.

Dependent Personality Disorder

Individuals with dependent personality disorder are extremely needy, relying on others for emotional support and decision making.

Epidemiology

Lifetime prevalence is 15% to 20%, 2% to 3% clinically.

Etiology

The etiology is unknown.

Clinical Manifestations

History and Mental Status Examination

These people yearn to be cared for. Because of their extreme dependence on others for emotional support and decision making, they live in great and continual fear of separation from someone they depend on, hence their submissive and clinging behaviors.

Differential Diagnosis

People with dependent personality disorder are similar to individuals with borderline personality disorder in their desire to avoid abandonment but do not exhibit the impulsive behavior, unstable affect, and poor self-image of the borderline patient.

Obsessive-Compulsive Personality Disorder

These individuals are perfectionists who require a great deal of order and control.

Epidemiology

The estimated prevalence is 1% in the general population. Men are diagnosed with obsessive-compulsive personality disorder twice as frequently as women.

Etiology

The etiology is unknown, but there may be an association with mood and anxiety disorders.

Clinical Manifestations

History and Mental Status Examination

Individuals with obsessive-compulsive personality disorder are perfectionists. They require order and control in every dimension of their lives. Their attention to minutiae frequently impairs their ability to finish what they start or to maintain sight of their goals. They are cold and rigid in relationships and make frequent moral judgments; devotion to work often replaces intimacy. They are serious and plodding; even recreation becomes a sober task.

Differential Diagnosis

Obsessive-compulsive personality disorder can be differentiated from obsessive-compulsive disorder (OCD) based on symptom severity.

MANAGEMENT

Because personality may have temperamental components and is developed over a lifetime of interacting with the environment, personality disorders are generally resistant to treatment. In general, **psychotherapy** is recommended for most personality disorders. **Psychodynamically based therapies** are commonly used, although they must be modified to each individual and each disorder. **Cognitive, behavioral**, and **family therapies** are also used to treat these disorders. Empirical studies validating the efficacy of various therapies are generally lacking. **Dialectical behavior therapy** (DBT) was developed specifically for the treatment of borderline personality disorder and has been validated in empirical studies. Group therapy incorporating various psychotherapeutic modalities is also used.

Pharmacotherapy is widely used in personality disorders, although no specific medication has been shown to treat any specific disorder. Instead, medications are targeted at the various associated symptoms of personality disorders. For example, mood stabilizers may be used for mood instability and impulsiveness. Benzodiazepines are commonly used for anxiety, although the potential for abuse and dependence is too often overlooked. Beta-blockers are also used frequently. For depression, obsessive-compulsive symptoms, and eating disturbances, selective serotonin reuptake inhibitors (SSRIs) and other antidepressants have been successfully used. Psychotic or paranoid symptoms are commonly treated with low-dose antipsychotics.

KEY POINTS

1. Personality disorders are categorized into three symptom clusters.
2. Personality disorders consist of an enduring pattern of experience and behavior.
3. They can produce transient psychotic symptoms during stress.
4. They are treated with psychotherapy and medications targeted at symptom relief.
5. Personality disorders are resistant to treatment.
6. They may have genetic associations with Axis I disorders.

Chapter

5 Substance-Related Disorders

Substance abuse is as common as it is costly to society. It is etiologic for many medical illnesses and is frequently comorbid with psychiatric illness. The *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) defines substance abuse and dependence independent of the substance. Hence, alcohol abuse and dependence is defined by the same criteria as heroin abuse and dependence. This chapter defines abuse and dependence and provides clinical descriptions of each substance-related disorder. The DSM-IV recognizes the different signs and symptoms associated with various drug addictions. We review the common substance-related disorders in sequence.

SUBSTANCE ABUSE

The DSM-IV defines substance abuse as a maladaptive pattern of substance use leading to clinically significant impairment or distress as manifested by one or more of the following:

- Failure to fulfill major role obligations at home, school, or work;
- Recurrent substance use in situations in which it is physically hazardous;
- Recurrent substance-related legal problems;
- Recurrent substance use despite persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance.

SUBSTANCE DEPENDENCE

Substance dependence is defined as a maladaptive pattern of substance use leading to clinically significant impairment or distress, as manifested by three or more of the following occurring at any time in a 12-month period:

1. Tolerance;
2. Withdrawal;
3. Repeated, unintended, excessive use;
4. Persistent failed efforts to cut down;
5. Excessive time spent trying to obtain the substance;
6. Reduction in important social, occupational, or recreational activities;
7. Continued use despite awareness that substance is the cause of psychological or physical difficulties.

Although each substance dependence disorder has unique features, these are considered the common features that define substance dependence. Each substance use disorder is discussed, with particular attention to their unique features.

ALCOHOL-RELATED DISORDERS

Alcohol Intoxication

Alcohol intoxication is defined by the presence of slurred speech, incoordination, unsteady gait, nystagmus, impairment in attention or memory, stupor or coma, and clinically significant maladaptive behavioral or psychological changes (inappropriate sexual or aggressive behavior, mood lability, impaired judgment, impaired social or occupational functioning) that develop during or shortly after alcohol ingestion.

The diagnosis of alcohol intoxication must be differentiated from other medical or neurologic states that may mimic intoxication, for example, diabetic

hypoglycemia; toxicity with various agents, including but not limited to ethylene glycol, lithium, and phenytoin; and intoxication with benzodiazepines or barbiturates. The diagnosis of alcohol intoxication can be confirmed by serum toxicologic screening, including a blood alcohol level (BAL).

Alcohol Dependence

Alcohol abuse becomes alcohol dependence when effects on one's life become more global and **tolerance** and **withdrawal** symptoms develop. The alcoholically dependent patient drinks larger amounts over longer periods of time than intended, spends a great deal of time attempting to obtain alcohol, and reduces participation in or eliminates important social, occupational, or recreational activities because of alcohol. In alcohol dependence, there also is a persistent desire or unsuccessful efforts to cut down or control alcohol intake.

Epidemiology

The percentage of Americans who abuse alcohol is thought to be high. Two thirds of Americans drink occasionally; 12% are heavy drinkers, drinking almost every day and becoming intoxicated several times a month. The Epidemiological Catchment Area study found a lifetime prevalence of alcohol dependence of 14%. The male-female prevalence ratio for alcohol dependence is 4:1.

Etiology

The etiology of alcohol dependence is unknown. Adoption studies and monozygotic twin studies demonstrate a partial genetic basis, particularly for men with alcoholism. Male alcoholics are more likely than female alcoholics to have a family history of alcoholism. Compared with control subjects, the relatives of alcoholics are more likely to have higher rates of depression and antisocial personality disorder (ASP). Adoption studies also reveal that alcoholism is multidetermined: genetics and environment (family rearing) both play a role.

Clinical Manifestations

History, Physical and Mental Status Examinations, and Laboratory Tests

The alcohol-dependent patient may deny and/or minimize the extent of drinking, making the early diagnosis of alcoholism difficult. The patient may present with accidents or falls, blackouts, motor vehicle accidents, or after an arrest for driving under the influence. Because denial is so prominent in the disorder, collateral information from family members is essential to the diagnosis. Early physical findings that suggest alcoholism include acne rosacea, palmar erythema, and painless hepatomegaly (from fatty infiltration).

Signs of more advanced alcoholism include cirrhosis, jaundice, ascites, testicular atrophy, gynecomastia, and Dupuytren's contracture. Cirrhosis can lead to complications including variceal bleeding, hepatocellular carcinoma, and hepatic encephalopathy. Medical disorders with an increased incidence in alcohol-dependent patients include pneumonia, tuberculosis, cardiomyopathy, hypertension, and gastrointestinal cancers (i.e., oral, esophageal, rectal, colon, pancreas, and liver).

There are also numerous neuropsychiatric complications of alcoholism. **Wernicke-Korsakoff syndrome** may develop in the alcohol-dependent patient because of **thiamine deficiency**. The Wernicke stage of the syndrome consists of the triad of **nystagmus, ataxia,** and **mental confusion**. These symptoms remit with the injection of thiamine (100 mg IM). Without thiamine, Wernicke's encephalopathy may progress to Korsakoff's psychosis (**anterograde amnesia** and **confabulation**), which is irreversible in two thirds of patients. Other neuropsychiatric complications of alcoholism include alcoholic hallucinosis, alcohol-induced dementia, peripheral neuropathy, substance-induced depression, and suicide. In the later stages of alcoholism, significant social and occupational impairment is likely: job loss and family estrangement are typical.

Various laboratory tests are helpful in making the diagnosis. BALs quantitatively confirm alcohol in the serum. They can also provide a rough measure of tolerance. In general, the higher the BAL without significant signs of intoxication, the more tolerant the patient has become of the intoxicating effects of alcohol. Alcohol-dependent patients also develop elevated high-density lipoprotein cholesterol and decreased low-density lipoprotein cholesterol, elevated mean corpuscular volume, elevated serum glutamic-oxaloacetic transaminase, and elevated serum glutamic-pyruvic transaminase. Thirty percent of alcohol-dependent patients, compared with 1% of control subjects, have evidence of old rib fractures on chest X ray.

Differential Diagnosis

The diagnosis of alcohol dependence is usually clear after careful history, physical and mental

status examination, and consultation with family or friends.

Management

Management is specific to the clinical syndrome. Alcohol intoxication is treated with supportive measures, including decreasing external stimuli and withdrawing the source of alcohol. Intensive care may be required in cases of excessive alcohol intake complicated by respiratory compromise. All suspected or known alcohol-dependent patients should receive oral vitamin supplementation with folate 1 mg/day and thiamine 100 mg/day. If oral intake is not possible, thiamine should be injected intramuscularly before any glucose is given (because glucose depletes thiamine stores).

Alcohol withdrawal syndromes include the following.

Minor Withdrawal

"The shakes" begin 12 to 18 hours after cessation of drinking and peak at 24 to 48 hours. Untreated, uncomplicated alcohol withdrawal lasts 5 to 7 days. It is characterized by tremors, nausea, vomiting, tachycardia, and hypertension. Minor withdrawal is treated with benzodiazepines, such as chlordiazepoxide (Librium) or oxazepam (Serax) titrated to the degree of withdrawal signs. The benzodiazepine is then tapered over a period of days. The goals of treatment are prevention of more serious complications and patient comfort.

Major Withdrawal

The risk of alcoholic seizures ("rum fits") begins 7 to 36 hours after cessation of drinking and peaks between 24 and 48 hours. One to six generalized seizures are common but rarely lead to status epilepticus. Alcoholic seizures precede delirium tremens in 30% of cases. Seizures are treated acutely with intravenous benzodiazepines. Prophylactic phenytoin (Dilantin) may be effective when administered during the high-risk period in patients with a history of withdrawal seizures.

Alcoholic hallucinosis has an onset within 48 hours of cessation of drinking and may last more than a week. It is characterized by vivid, unpleasant auditory hallucinations in the presence of a clear sensorium. Alcoholic hallucinosis may be treated with a neuroleptic (e.g., haloperidol [Haldol] 2–5 mg twice a day). On rare occasions, these hallucinations become chronic.

Alcohol withdrawal delirium (delirium tremens) is a life-threatening condition manifested by delirium (perceptual disturbances, confusion or disorientation, agitation), autonomic hyperarousal, and mild fever. It affects up to 5% of hospitalized patients with alcohol dependence and typically begins 2 to 3 days after abrupt reduction in or cessation of alcohol intake. It is treated with intravenous benzodiazepines and supportive care. Treatment may need to occur in an intensive care unit, particularly if there is significant autonomic instability (e.g., rapidly fluctuating blood pressure). The syndrome typically lasts 3 days but can persist for weeks.

Alcohol Rehabilitation

The two goals of rehabilitation are **sobriety** and treatment of **comorbid psychopathology**.

To have a lasting recovery, the patient must stop denying the illness and accept the diagnosis of alcohol dependence. Alcoholics Anonymous (AA), a worldwide self-help group for recovering alcohol-dependent patients, has been shown to be one of the most effective programs for achieving and maintaining sobriety. The program involves daily to weekly meetings that focus on 12 steps toward recovery. Members are expected to pursue the 12 steps with the assistance of a sponsor (preferably someone with several years of sobriety).

Alcohol appears to be a potent depressant, so treatment of depression should be geared to patients who remain depressed after 2 to 4 weeks of sobriety. Anxiety is also common in withdrawing or newly sober patients and should be assessed after at least 1 month of sobriety. Inpatient and residential rehabilitation programs use a team approach aimed at focusing the patient on recovery. Group therapy allows patients to see their own problems mirrored in and confronted by others. Family therapy allows the patient to examine the role of the family in alcoholism.

Disulfiram (Antabuse) can be helpful in maintaining sobriety in some patients. It acts by inhibiting the second enzyme in the pathway of alcohol metabolism, aldehyde dehydrogenase, so that acetaldehyde accumulates in the bloodstream, causing flushing, nausea, vomiting, palpitations, and hypotension. In theory, disulfiram should inhibit drinking by making it physiologically unpleasant; however, because the effects can be fatal in rare cases, patients must be committed to abstinence and fully understand the danger of drinking while taking disulfiram. The usual dose of disulfiram is 250 mg daily.

Naltrexone (Revia) is an opiate antagonist medication that has the strongest empirical support in

reducing alcohol intake among medications available in the United States. Naltrexone reduces both the amount of alcohol intake and the frequency of alcohol intake. Naltrexone is usually dosed at 50 mg per day, but higher doses may potentially be more effective. Unlike disulfiram, patients can continue taking naltrexone if they relapse to alcohol intake. Naltrexone, as an opioid antagonist, may work in part by reducing the reinforcing high of alcohol ingestion.

Many studies have demonstrated benefits from rehabilitation programs, but nearly half of all treated alcohol-dependent patients will relapse, most commonly in the first 6 months.

KEY POINTS

1. In alcohol dependence, denial and minimization are common.
2. Benzodiazepines are used in acute detoxification to prevent life-threatening complications of withdrawal.
3. Peak incidence of alcoholic seizures is within 24 to 48 hours.
4. Rehabilitation is aimed at abstinence and treating comorbid disorders.
5. Rehabilitation involves AA and group and family therapies.
6. Fifty percent of treated alcoholics will relapse.
7. Wernicke-Korsakoff syndrome is due to thiamine deficiency.
8. Wernicke's triad consists of nystagmus, ataxia, and mental confusion.
9. Korsakoff's symptoms are anterograde amnesia and confabulation.

SEDATIVE, HYPNOTIC, AND ANXIOLYTIC SUBSTANCE USE DISORDERS

Sedative, hypnotic, and anxiolytic drugs are widely used. They are all **cross-tolerant** with each other and with alcohol. Included in this class are barbiturates and benzodiazepines. Of these, the benzodiazepines are the most widely prescribed and available.

Epidemiology

Approximately 15% of the general population is prescribed a benzodiazepine in a given year. Some patients abuse these drugs.

Clinical Manifestations

History, Physical and Mental Status Examinations, and Laboratory Tests

Sedative-hypnotic drug abuse and dependence are associated with syndromes of intoxication, withdrawal, and withdrawal delirium that resemble those of alcohol.

Intoxication only can be distinguished from alcohol intoxication by the presence (or absence) of alcohol on the breath, or in the serum or urine. Barbiturates, when taken orally, are much more likely than benzodiazepines to cause clinically significant respiratory compromise. Intoxication can be confirmed through quantitative or qualitative serum or urine toxicologic analyses. Serum toxicologic screens can identify the presence of benzodiazepines and barbiturates and their major metabolites.

Withdrawal symptoms are listed in Table 5-1. Withdrawal delirium (confusion, disorientation, and visual and somatic hallucinations) has an onset of 3 to 4 days after abstinence. Dependence requires the presence of three or more of the seven symptoms listed in Table 5-1.

Management

Treatment of sedative-hypnotic withdrawal may be on an outpatient or inpatient basis. Generally, inpatient detoxification is required when there is comorbid medical or psychiatric illness, prior treatment failures, or lack of support by family or friends. On an inpatient unit, benzodiazepines or barbiturates may be administered and tapered in a controlled manner. Withdrawal from short-acting substances is generally more severe, whereas withdrawal from longer-acting substances is more prolonged.

TABLE 5-1

Signs and Symptoms of Sedative-Hypnotic Withdrawal

Minor Withdrawal	More Severe Withdrawal
Restlessness	Coarse tremors
Apprehension	Weakness
Anxiety	Vomiting
	Sweating
	Hyperreflexia
	Nausea
	Orthostatic hypotension
	Seizures

Withdrawal from barbiturates is more dangerous than from benzodiazepines: it can (much more easily) lead to hyperpyrexia and death. Withdrawal is managed by scheduled dosing and tapering of a benzodiazepine or barbiturate (diazepam or phenobarbital).

In patients who have been abusing alcohol and benzodiazepines or barbiturates, it may be necessary to perform a **pentobarbital challenge test**. This test allows for the quantification of tolerance to perform a controlled taper, thereby reducing the problems of withdrawal.

Treatment of sedative-hypnotic dependence resembles that for alcohol dependence. After detoxification, the patient can enter a residential rehabilitation program or a day or evening treatment program. Referral to AA is appropriate because the addiction issues and recovery process are similar. Families may be referred to Al-Anon, an AA focused family education and support group.

KEY POINTS

1. Sedatives and hypnotic drugs are cross-tolerant with alcohol.
2. They have intoxicating effects and result in withdrawal states similar to alcohol.
3. Tolerance can be measured by a pentobarbital challenge test.
4. Treatment resembles that for alcoholism.

OPIOID USE DISORDERS

Opiates include morphine, heroin, codeine, meperidine, and hydromorphone. Heroin is only available illegally in the United States. Opiates are commonly used for pain control.

Epidemiology

Opiate use and abuse are relatively uncommon in the United States. Lifetime prevalence in 1991 was 0.9% and point prevalence was less than 0.1%, although more recent surveys indicate opiate use and abuse has been increasing during the past decade. Many of those who use opiates recreationally become addicted. The number of opiate addicts in the United States is estimated at 500,000.

Clinical Manifestations

History, Physical and Mental Status Examinations, and Laboratory Tests

Most heroin and morphine users take opiates intravenously, which produces flushing and an intensely pleasurable, diffuse bodily sensation that resembles orgasm. This initial "rush" is followed by a sense of well-being. Psychomotor retardation, drowsiness, inactivity, and impaired concentration ensue. Signs of intoxication occur immediately after the addict "shoots up" and include pupillary constriction, respiratory depression, slurred speech, hypotension, bradycardia, and hypothermia. Nausea, vomiting, and constipation are common after opiate use. Opiate use can be confirmed by urine or serum toxicologic measurements.

Opiate abuse is defined by the criteria for substance abuse noted above. In opiate dependence, tolerance to the effects of opiates occurs. Addicts "shoot up" three or more times per day.

Withdrawal symptoms usually begin 10 hours after the last dose. Withdrawal from opiates can be highly uncomfortable but is rarely medically complicated or life-threatening. Withdrawal symptoms are listed in Table 5-2.

Opiate addicts often have comorbid substance use disorders, antisocial or borderline personality disorders, and mood disorders. Opiate addicts are more prone to commit crimes because of the high cost of opiates. Opiate addiction also is associated with high mortality rates from inadvertent overdoses, accidents, and suicide. Opiate addicts are also at higher risk of medical problems because of poor nutrition and use of dirty needles. Common medical disorders include

TABLE 5-2

Symptoms of Opiate Withdrawal

Mild Withdrawal	More Severe Withdrawal
Dysphoric mood, anxiety, and restlessness	Nausea
Lacrimation or rhinorrhea	Vomiting
Pupillary dilatation	Muscle aches
Piloerection	Seizures (in meperidine withdrawal)
Sweating	
Hypertension	Abdominal cramps
Tachycardia	Hot and cold flashes
Fever	Severe anxiety
Diarrhea	
Insomnia	
Yawning	

serum hepatitis, HIV infection, endocarditis, pneumonia, and cellulitis.

Differential Diagnosis

The diagnosis of opiate addiction is usually obvious after a careful history and mental status and physical examinations.

Management

Patients addicted to opiates should be gradually withdrawn using methadone. **Methadone** is a weak agonist of the *mu* opiate receptor and has a longer half-life (15 hours) than heroin or morphine. Thus, it causes relatively few intoxicant or withdrawal effects. Generally, the initial dose of methadone (typically 5–20 mg) is based on the profile of withdrawal symptoms. Withdrawal from short-acting opiates lasts 7 to 10 days; withdrawal from longer-acting meperidine lasts 2 to 3 weeks.

Clonidine, a centrally acting alpha 2 receptor agonist that decreases central noradrenergic output, can also be used for acute withdrawal syndromes. It is remarkably effective at treating the autonomic symptoms of withdrawal but does little to curb the drug craving. Risks of sedation and hypotension limit clonidine's usefulness in outpatient settings. Additional medications can be used to relieve uncomfortable symptoms of withdrawal, such as dicyclomine for abdominal cramping, promethazine for nausea, and quinine for muscle aches.

Rehabilitation generally involves referral to an intensive day treatment program and to Narcotics Anonymous, a 12-step program similar to AA. Methadone maintenance, daily administration of 60–100 mg of methadone in government-licensed methadone clinics, is used widely for patients with demonstrated physiologic dependence. Long-term administration of methadone can alleviate drug hunger and minimize drug-seeking behavior.

KEY POINTS

1. Recreational use of opiates often leads to addiction.
2. Opiate addicts are at increased risk of HIV, pneumonia, endocarditis, hepatitis, and cellulitis.
3. High mortality occurs from accidental overdose, suicide, and accidents.
4. Opiate withdrawal begins 10 hours after last dose.
5. Withdrawal is uncomfortable but not usually medically complicated.

CENTRAL NERVOUS SYSTEM (CNS) STIMULANT USE DISORDERS

Cocaine and amphetamines are readily available in the United States. The patterns of use and abuse of and dependence on cocaine and amphetamines are similar because both are **CNS stimulants** with similar psychoactive and sympathomimetic effects.

In the United States, cocaine is available in two forms: as cocaine hydrochloride powder, which is typically snorted, and as cocaine alkaloid crystal ("crack"), which is typically smoked. Cocaine has an extremely rapid onset of action (when snorted or smoked) and a short half-life, requiring frequent dosing to remain "high."

In the United States, an amphetamine (dextroamphetamine) and methylphenidate are available in pill form by prescription for the treatment of obesity, narcolepsy, and attention-deficit/hyperactivity disorder. Various forms of amphetamine are used illicitly including a very pure form of methamphetamine, called crystal methamphetamine, which can be snorted or smoked. Amphetamines have a longer half-life than cocaine and hence are taken less frequently.

Clinical Manifestations

Cocaine or amphetamine intoxication is characterized by

1. Maladaptive behavioral changes (e.g., euphoria or hypervigilance);
2. Tachycardia or bradycardia;
3. Pupillary dilatation;
4. Hyper- or hypotension;
5. Perspiration or chills;
6. Nausea or vomiting;
7. Weight loss;
8. Psychomotor agitation or retardation;
9. Muscular weakness, respiratory depression, chest pain, cardiac dysrhythmias;
10. Confusion, seizures, dyskinesia, or coma.

Cocaine intoxication can cause tactile hallucinations ("coke bugs"). Both cocaine and amphetamine intoxication can lead to agitation, impaired judgement, and **transient psychosis** (e.g., paranoia, visual hallucinations). Cocaine and amphetamine dependence is defined by the criteria outlined above for substance dependence.

Withdrawal of cocaine or amphetamines leads to fatigue, depression, nightmares, headache, profuse

sweating, muscle cramps, and hunger. Withdrawal symptoms peak in 2 to 4 days.

Management

Withdrawal from amphetamines or other (CNS) stimulants is self-limited and usually does not require inpatient detoxification. Psychosis from amphetamine intoxication or withdrawal is generally self-limited, requiring only observation in a safe environment. Antipsychotic medications can be used for agitation.

Ultimately, the goal is rehabilitation. Narcotics Anonymous, treatment of comorbid psychopathology, drugs to reduce craving, and family therapy are the essential features of cocaine rehabilitation.

KEY POINTS

1. Cocaine and amphetamines are CNS stimulants.
2. CNS stimulants can cause transient psychosis (e.g., "coke bugs" or paranoia).
3. Withdrawal symptoms (fatigue, depression, nightmares, etc.) peak in 2 to 4 days.
4. Withdrawal from CNS stimulants is self-limited.

CANNABIS AND MISCELLANEOUS SUBSTANCE USE DISORDERS

Cannabis

Cannabis is widely used throughout the world in the forms of marijuana and hashish. The drug is usually smoked and causes a state of euphoria. Complications of cannabis include impaired judgement, poor concentration, and poor memory. Serious complications include delirium and/or psychosis.

Club Drugs

Club Drugs are a group of drugs classified by the National Institute on Drug Abuse (NIDA) according to their popularity in dance clubs and other party venues. These drugs are of a wide variety of chemical classes, but are linked by their frequent use in social groups and the fact that they are commonly taken together. Because of their popularity and tendency for users to show up in emergency rooms, we review some of the more widely used Club Drugs below.

Ecstasy (MDMA) – Ecstasy is a widely popular drug with mixed stimulant and hallucinogenic properties. Many users report a stimulant and euphoric effect, and MDMA appears to specifically enhance the user's desire for intimacy with others. As such, its use has been associated with an increased frequency of unsafe sexual activity. Acute use of MDMA has been associated with deaths from various causes. Long-term MDMA use appears to lead to a permanent loss of fine-diameter serotonin axons throughout the brain.

Methamphetamine – Also known as crystal or crank, methamphetamine is a psychostimulant which is neurotoxic to dopamine neurons. Methamphetamine is often produced locally in small labs and can therefore vary greatly in purity.

GHB – Gamma-hydroxybutyrate is a compound used in lower doses by bodybuilders and others seeking to gain muscle mass (GHB promotes the release of growth hormone). In higher doses, GHB is used to produce a high, and is common among the club and party scene. GHB is easily overdosed, and can lead to death from respiratory arrest.

Ketamine – Also known as Special K, ketamine is a dissociative anesthetic mostly used in veterinary medicine. It is used for its hallucinatory, dissociative effect.

Rohypnol – Rohypnol is a benzodiazepine approved for clinical use in some countries outside the United States. Rohypnol produces classic benzodiazepine effects of sedation. However, it has strong amnestic properties, and may be a frequent culprit in drugging others for the purpose of theft or sexual assault.

LSD – Lysergic acid diethylamide is famous for its hallucinogenic properties. Acute use can produce a highly euphoric ("good trip") or a highly dysphoric ("bad trip") hallucinatory experience. Long-term LSD use can lead to psychosis or hallucinogen persisting perception disorder.

Chapter

6 Eating Disorders

Eating disorders are characterized by disturbances in eating behavior and an overconcern with body image or size. Although eating disorders are classified into two discrete diagnostic categories in the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV), many symptoms overlap. The principal diagnostic distinction is based on ideal body weight. When abnormal eating behavior causes body weight to fall below a defined percentage of expected body weight, a diagnosis of anorexia nervosa is made. If ideal body weight is maintained in the presence of abnormal eating behaviors, a diagnosis of bulimia nervosa is made. Eating disorders likely lie along a continuum of disturbances in eating behavior and often are associated with mood disorders and other psychiatric illnesses (Table 6-1).

ANOREXIA NERVOSA

Anorexia nervosa is a severe eating disorder characterized by **low body weight**. Anorexia nervosa is diagnosed when a person's body weight falls below 85% of the ideal weight for that individual. The weight loss must be due to behavior directed at maintaining low weight or achieving a particular body image.

Epidemiology

The point prevalence of anorexia nervosa is between 0.5% and 1% in women, and more than 90% of patients with anorexia nervosa are women. The prevalence in men is not clear. Average age of onset is 17; onset is rare before puberty or after age 40. Anorexia nervosa is more common in industrial societies and higher socioeconomic classes.

Etiology

Eating disorders and their subtypes likely share many common bases of origin. Psychological theories of anorexia nervosa remain speculative. Patients with anorexia nervosa generally have a high fear of losing control, difficulty with self-esteem, and commonly display "all or none" thinking. Although it is not specific to eating disorders, past physical or sexual abuse may be a risk factor. Contemporary theories focus on the need to control one's body.

Social theories propose that societal opinions, which equate low body weight with attractiveness, drive women to develop eating disorders. Although this fact may be responsible for some cases (e.g., anorexia nervosa is more common among dancers and models), historically anorexia nervosa has been present during periods when societal mores for beauty were different.

Biologic, familial, and genetic data support a biologic and heritable basis for anorexia. Family studies reveal an increased incidence of mood disorders and anorexia nervosa in first-degree relatives of patients with anorexia nervosa. Twin studies show higher concordance for monozygotic versus dizygotic twins. Neuroendocrine evidence supporting a biologic contribution to anorexia includes alterations in corticotropin-releasing factor, reduced central nervous system norepinephrine metabolism, and that amenorrhea (caused by decreased luteinizing hormone and follicle-stimulating hormone release) sometimes precedes the onset of anorexia nervosa.

TABLE 6-1

Classification of Eating Disorders

Anorexia Nervosa	Bulimia Nervosa
Restricting type	Nonpurging type
Binge eating/purging type	Purging type

Clinical Manifestations

History and Mental Status Examination

DSM-IV criteria for the diagnosis of anorexia nervosa include refusal to keep body weight at greater than 85% of ideal, an intense fear of weight gain, preoccupation with body size and shape, a disproportionate influence of body weight on personal worth, and the denial of the medical risks of low weight. Patients with anorexia nervosa generally do not have a loss of appetite; they refuse to eat out of fear of gaining weight. Amenorrhea is also a diagnostic criteria in postmenarchal females (delay of menarche may occur in premenarchal girls). In some cases, amenorrhea precedes the development of anorexia nervosa; however, in most cases, it appears to be a consequence of starvation.

Individuals with anorexia nervosa commonly exercise intensely to lose weight and alter body shape. Some restrict food intake as a primary method of weight control; others use binging and purging (use of laxatives, enemas, diuretics, or induced vomiting) to control weight.

The behavioral repertoire used to control body weight is used to further classify anorexia nervosa into two subtypes: restricting type and binging/purging type. In the restricting type, the major methods of weight control are food restriction and exercise. In the binging/purging type, food restriction and exercise may be present, but binge eating and subsequent purging behaviors also are present.

The natural course of anorexia is not well understood, but many cases become chronic. The long-term mortality of anorexia nervosa secondary to suicide or medical complications is greater than 10%.

Differential Diagnosis

Conditions that can resemble anorexia should be ruled out. These include major depression with loss of appetite and weight, some psychotic disorders where nutrition may not be adequate, body dysmorphic disorder, and a variety of general medical (especially neuroendocrine) conditions. Anorexia nervosa is differentiated from bulimia nervosa by the presence of low weight in the former.

Management

The management of anorexia nervosa is directed at the presenting symptoms. When medical complications are present, these must be carefully treated and followed. If ipecac use to induce vomiting is suspected, ipecac toxicity must be ruled out.

During starvation, psychotherapy is of little value because of the cognitive impairment produced by starvation. When patients are less medically ill, a therapeutic program including supervised meals; weight and electrolyte monitoring; psychoeducation about the illness, starvation, and nutrition; individual psychotherapy, and family therapy can begin. Psychopharmacology management often includes antidepressants, especially the selective serotonin reuptake inhibitors (SSRIs) to treat comorbid depression. Psychopharmacologic treatments are used principally to treat any comorbid psychiatric illness and have little or no effect on the anorexia per se.

KEY POINTS

1. Anorexia nervosa is a severe eating disorder characterized by low body weight.
2. It is diagnosed more than 90% of the time in women.
3. Anorexia nervosa can cause serious medical complications and has a greater than 10% long-term mortality rate.

BULIMIA NERVOSA

Bulimia nervosa is an eating disorder characterized by **binge eating** with the **maintenance of body weight**.

Epidemiology

The estimated point prevalence of bulimia nervosa is 1% to 3% of women. The male-female ratio is 1 : 10. This illness occurs disproportionately among whites in the United States.

Etiology

Many of the factors in the genesis of anorexia nervosa are also implicated in bulimia nervosa. Familial and genetic studies support similar familial linkages in both disorders. Psychological theories for bulimia nervosa stress an addiction or obsessive-compulsive behavioral model. Biologic, neurologic, and endocrine findings are less prominent in theories of causation of bulimia nervosa. Abnormal serotonin metabolism is thought to play more of a role in bulimia nervosa than in anorexia nervosa.

Clinical Manifestations

History and Mental Status Examination

Bulimia nervosa is diagnosed in individuals who engage in binge eating and behaviors designed to avoid weight gain but who maintain their body weight. In addition, these are people whose self-evaluation is overly influenced by their body weight and shape.

Food binges in bulimia nervosa may be precipitated by stress or altered mood states. Once a binge begins, the individual typically feels out of control and continues to eat large quantities of food, often to the point of physical discomfort. **Purging** may follow and most often consists of vomiting, usually induced mechanically by stimulating the gag reflex or using ipecac. Other purging methods used to avoid weight gain include laxative and diuretic abuse and enemas. Bulimic individuals often exercise and restrict their food intake. As in anorexia nervosa, patients with bulimia nervosa are overly concerned with body image and are preoccupied with becoming fat. Bulimia nervosa is classified into two subtypes: nonpurging type or purging type (see Table 6-1) according to whether purging behavior is present.

Differential Diagnosis

Bulimia nervosa should be distinguished from the binge eating and purging subtype of anorexia nervosa. If body weight is less than 85% of ideal, a diagnosis of anorexia nervosa is made. Binge eating can occur in major depression and in borderline personality disorder, but is not tied to a compulsion to reduce weight.

Management

The treatment for bulimia nervosa is similar to that for anorexia nervosa. Although medical complications of starvation are not present, other medical complications can require careful medical management and, at times, hospitalization. Psychotherapy focuses at first on achieving control of eating behavior. Cognitive therapy may be useful in treating overconcern with body image. Self-esteem and interpersonal relationships become the focus of therapy as the behavioral problems abate. Antidepressants, especially SSRIs, are more effective in the treatment of bulimia nervosa than in anorexia nervosa (including those patients who do not have comorbid depression).

KEY POINTS

1. Bulimia nervosa is a severe eating disorder characterized by binge eating and purging.
2. It is also characterized by maintenance of normal body weight.
3. Bulimia nervosa is more common in women than in men.
4. It can have serious medical complications.

MEDICAL COMPLICATIONS

Eating disorders, when persistent, can have serious medical consequences. The lifetime mortality from anorexia nervosa is approximately 10%; it is unknown for bulimia nervosa. Table 6-2 lists the common medical complications of eating disorders. The most serious of these, gastric or esophageal rupture, cardiomyopathy from ipecac toxicity, and cardiac arrhythmias secondary to electrolyte imbalance, can be fatal. Other complications parallel those of chronic medical illness, take a severe toll on the patient's overall functioning, and cause tremendous suffering and burden for their families. In addition to these medical complications, secondary psychiatric and neurologic sequela include cognitive decline, metabolic encephalopathy, and severe mood disturbance, all with profound consequences for patients and their families.

TABLE 6-2

Medical Complications of Eating Disorders

Behavior	Medical Complication
Binge eating	Gastric dilatation or rupture
Vomiting	Esophageal rupture Parotiditis with hyperamylasemia Hypokalemic, hypochloremic, metabolic alkalosis (with cardiac arrhythmias) Ipecac toxicity (cardiac and skeletal myopathies)
Laxative use	Constipation (due to laxative dependence) Metabolic acidosis Dehydration
Diuretic use	Electrolyte abnormalities (with cardiac arrhythmias) Dehydration
Starvation	Leukopenia, anemia Increased ventricular/brain ratio Hypotension, bradycardia Hypothermia Hypercholesterolemia Edema Dry skin, lanugo hair

Chapter

7 Disorders of Childhood and Adolescence

Many disorders seen in adults can occur in children. However, there is a group of disorders usually first diagnosed in children. Table 7-1 lists these disorders according to the *Diagnostic and Statistical Manual of Mental Disorders,* 4th edition (DSM-IV). This chapter reviews only the more common disorders.

Child psychiatric assessment requires attention to details of a child's stage of development, family structure and dynamics, and normative age-appropriate behavior. Consulting with parents and obtaining information from schools, teachers, and other involved parties (e.g., Department of Social Services/Youth Services) are essential to proper assessment.

Children, especially young children, usually express emotion in a more **concrete** (less abstract) way than adults. Consequently, child interviews require more concrete queries (Do you feel like crying? *instead of* Are you sad?). Playing games, taking turns telling stories, and imaginative play are often used to gain insight into the child's emotional and interpersonal life. During play, observations are also made regarding activity level, motor skills, and verbal expression. Children are much more likely than adults to have **comorbid** mental disorders, making diagnosis and treatment more complicated.

The complexities of diagnosis in child psychiatry often require the use of psychological testing. Tests of general intelligence include the Stanford-Binet Intelligence Scale (one of the first intelligence tests developed and often used in young children) and the Wechsler Intelligence Scale for Children–Revised (WISC-R). The WISC-R is the most widely used intelligence test for assessing school-age children. It yields a verbal score, a performance score, and a full-scale score (both verbal and performance) or **intelligence quotient (IQ)**.

There are many other tests and objective rating scales designed to measure behavior (e.g., impulsiveness, physical activity), perceptual-motor skills (by drawing people, placing pegs in appropriately shaped holes), and personality style (by describing what is happening in an ambiguous scene).

Because seizure activity or subtle electroencephalographic abnormalities are common in certain child psychiatric disorders, an **electroencephalogram (EEG)** may be warranted. The evaluation of mental retardation usually involves a search for possible causes.

MENTAL RETARDATION

Patients with mental retardation have subnormal intelligence (as measured by IQ) combined with deficits in adaptive functioning. IQ is defined as the mental age (as assessed using a WISC-R) divided by the chronologic age and multiplied by 100. If mental age equals chronologic age, then the ratio equals one and the IQ is "100." An IQ of less than 70 is required for the diagnosis of mental retardation. Severity ranges from mild to profound and is based on IQ (Table 7-2).

TABLE 7-1

Disorder Usually First Diagnosed in Infancy, Childhood, or Adolescence

- Mental Retardation
- Learning Disorders
  - Reading Disorder
  - Mathematics Disorder
  - Disorder of Written Expression
- Motor Skills Disorder
  - Developmental Coordination Disorder
- Communication Disorders
  - Expressive Language Disorder
  - Mixed Receptive-Expressive Language Disorder
  - Phonological Disorder
  - Stuttering
- Pervasive Developmental Disorders
  - Autistic Disorder
  - Rett's Disorder
  - Childhood Disintegrative Disorder
  - Asperger's Disorder
- Attention-Deficit and Disruptive Behavior Disorders
  - Attention-Deficit/Hyperactivity Disorder
  - Conduct Disorder
  - Oppositional Defiant Disorder
- Feeding and Eating Disorders of Infancy or Early Childhood
  - Pica
  - Rumination Disorder
  - Feeding Disorder of Infancy or Early Childhood
- Tic Disorders
  - Tourette's Disorder
  - Chronic Motor or Vocal Tic Disorder
  - Transient Tic Disorder
- Elimination Disorders
  - Encopresis
  - Enuresis
- Other Disorders of Infancy, Childhood, or Adolescence
  - Separation Anxiety Disorder
  - Selective Mutism
  - Reactive Attachment Disorder of Infancy or Early Childhood
  - Stereotypic Movement Disorder

Epidemiology

Mental retardation affects 1% to 2% of the population and has a male-female ratio of 2 : 1. Milder forms of mental retardation occur more frequently in families with low socioeconomic status (SES); more severe forms of mental retardation are independent of SES. Most patients with mental retardation have mild or moderate forms (see Table 7-2).

TABLE 7-2

Mental Retardation

Degree of MR	IQ	Percentage of Total MR Population
Mild	50–70	85%
Moderate	35–50	10%
Severe	20–35	3–4%
Profound	<20	1–2%

Etiology

Mental retardation can be thought of as a final common pathway of a number of childhood or perinatal disorders. The most common cause of mental retardation is **Down syndrome** (trisomy 21). **Fragile X syndrome** is the most common cause of *heritable* mental retardation. Inborn errors of metabolism, perinatal or early childhood head injuries, maternal diabetes, substance abuse, toxemia, or rubella can all cause mental retardation. Overall, there are more than 500 genetic abnormalities associated with mental retardation. In 30% to 40% of patients with mental retardation, no clear etiology can be determined.

Clinical Manifestations

History, Physical and Mental Status Examinations, and Laboratory Tests

Most mentally retarded children have physical malformations that identify them at birth as being at high risk for mental retardation (such as the characteristic facies of the child with Down syndrome). Infants can show signs of significantly subaverage intellectual functioning. Young children with mental retardation may be identified by parents or pediatricians after failure to meet developmental milestones in a number of functional areas (e.g., delayed speech, social skills, or self-care skills capacity) or on scoring an IQ less than 70 on the Stanford-Binet (usually only for very young children) or WISC–R (standard for school-age children).

The onset of symptoms must be before age 18. The patient must have both an IQ less than or equal to 70 and concurrent deficits or impairments in several areas of adaptive functioning (e.g., communication, self-care, interpersonal skills). Laboratory

findings may suggest metabolic or chromosomal etiology.

Differential Diagnosis

Attention-deficit/hyperactivity disorder (ADHD), learning disorders, depression, schizophrenia, and seizure disorder can all resemble mental retardation. These disorders can also be comorbid conditions. Children suspected of having mental retardation should have a thorough medical and neurologic evaluation, including IQ testing, an EEG, and brain imaging (CT or MRI).

Management

Management depends on the degree of retardation, the course, and the particular abilities of the child and the parents. Most children with mental retardation progress through normal milestones (standing, walking, talking, learning to recognize letters and numbers) in a similar pattern to normal children but at a slower rate. Growth and development occur in children with mental retardation. They can have developmental spurts, like normal children, that could not have been predicted at an earlier age.

In mild mental retardation, the child is typically considered educable. The child can usually learn to read, write, and perform simple arithmetic. With family support and special education, most of these children will be able to live with their parents. The long-term goal of treatment is to teach the child to function in the community and to hold some type of job.

In moderate mental retardation, the child is typically considered trainable. With training, the child can learn to talk, to recognize his or her name and a few simple words, and to perform activities of daily living (bathing, dressing, handling small change) without assistance. The long-term goal of treatment is typically to enable the child to live and function in a supervised group home.

Children with severe or profound mental retardation almost invariably require care in institutional settings, usually beginning very early in life. These forms of mental retardation are often associated with specific syndromes (e.g., Tay-Sachs disease) in which there is progressive physical deterioration leading to premature death.

KEY POINTS

1. Mental retardation is defined by IQ less than or equal to 70 and functioning in specific areas.
2. It is more common in males (2:1).
3. It is most commonly caused by Down syndrome (trisomy 21).
4. Mental retardation is managed in developmentally appropriate settings.

LEARNING DISORDERS

Learning disorders are characterized by performance in a specific area of learning (e.g., reading, writing, arithmetic) substantially below the expectation of a child's chronologic age, measured intelligence, and age-appropriate education. The (DSM-IV) identifies three learning disorders: **reading disorder, mathematics disorder**, and **disorder of written expression.**

Etiology

Specific learning disorders often occur in families. They are presumed to result from **focal cerebral injury** or from a **neurodevelopmental defect**.

Epidemiology

Learning disorders are relatively common. Reading disorder affects 4% of school-age children and mathematics disorder is estimated at 1%. The incidence of disorder of written expression is not yet known. Learning disorders are two to four times more common in boys than girls.

Clinical Manifestations

History, Mental Status Examination, and Laboratory Tests

Specific learning disorders are typically diagnosed after a child has exhibited difficulties in a specific academic area. Because reading and arithmetic are usually not taught before the first grade, the diagnosis is seldom made in preschoolers. Some children may not be diagnosed until fourth or fifth grade, particularly if they have a high IQ and can mask their deficits. The diagnosis of learning disorder is con-

firmed through specific intelligence and achievement testing. Children with learning disorders do not obtain achievement test scores consistent with their overall IQs.

Differential Diagnosis

It is important to establish that a low achievement score is not due to some other factor such as lack of opportunity to learn, poor teaching, or cultural factors (e.g., English as a second language). Physical factors (such as hearing or vision impairment) must also be ruled out.

Finally, it is important to consider and test for more global disorders such as pervasive developmental disorder, mental retardation, and communication disorders. It is not uncommon to find that several of these disorders coexist. A specific learning disorder diagnosis is made when the full clinical picture is not adequately explained by other comorbid conditions.

Management

Children with these disorders often need remedial education, especially if their diagnosis was made late. They also need to be taught learning strategies to overcome their particular deficits. Acceptable skills in the disordered area can often be achieved with steady supportive educational assistance, though patients may be affected by these disorders throughout adulthood.

KEY POINTS

1. There are three types of learning disorders: mathematics, reading, and written expression.
2. They tend to be familial and are probably due to cerebral injury or maldevelopment.
3. Reading disorder is the most common and all three disorders occur more in boys (2–4:1).
4. The diagnosis is confirmed through achievement tests.
5. Physical or social factors must be ruled out.
6. Management involves remedial education and learning strategies.

AUTISTIC DISORDER

Autistic disorder is the most common of the pervasive developmental disorders of childhood onset. It is characterized by the triad of impaired social interactions, impaired ability to communicate, and restricted repertoire of activities and interests.

Etiology

Autistic disorder is familial. Genetic studies demonstrate incomplete penetrance (36% concordance rate in monozygotic twins), although a specific genetic defect has not been discovered. A small percentage of those with autistic disorder have a fragile X chromosome, and a high rate of autism exists with **tuberous sclerosis.**

Epidemiology

Autistic disorder is rare. It occurs in two to five children per 10,000 live births. The male-female sex ratio is 3–4:1.

Clinical Manifestations

History, Mental Status Examination, and Laboratory Tests

Abnormal development is usually first noted soon after birth. Commonly, the first sign is **impairment in social interactions** (failure to develop a social smile, facial expressions, or eye-to-eye gaze). Older children often fail to develop nonverbal forms of communication (e.g., body postures and gestures) and may seem to have no desire or to lack the skills to form friendships. There is also a lack of seeking to share enjoyment (i.e., not showing, sharing, or pointing out objects they find interesting). By definition, findings must be present before age 3.

Autistic disorder is also characterized by a **marked impairment in communication.** There may be delay in or total lack of language development. Those children who do develop language show impairment in the ability to initiate and sustain conversations and use repetitive or idiosyncratic language. Language may also be abnormal in pitch, intonation, rate, rhythm, or stress.

Finally, there are usually **restrictive, repetitive, or stereotyped patterns of behavior, interests, or activities.** There may be an encompassing preoccupation with one or more stereotyped and restricted patterns of interest (e.g., amassing baseball trivia), an inflexible adherence to specific nonfunctional routines or rituals (e.g., eating the same meal in the same place at the same time each day), stereotyped or repetitive motor mannerisms (e.g., whole-body rocking), and a persistent preoccupation with the parts of objects (e.g., buttons).

Approximately 25% of children with autistic disorder have comorbid seizures; approximately 75% have mental retardation (the moderate type is most common). EEGs and intelligence testing are typically part of the initial evaluation. Rarely, specific special skills are present, e.g., calendar calculation.

Differential Diagnosis

The diagnosis is usually clear after careful history, mental status examination, and developmental monitoring. However, childhood psychosis, mental retardation (alone), language disorders, and congenital deafness or blindness must all be ruled out.

Management

Autistic disorder is a chronic lifelong disorder with relatively severe morbidity. Very few individuals with autism will ever live independently. Once the diagnosis is made, parents should be informed that their child has a neurodevelopmental disorder (not a behavioral disorder that they might feel responsible for creating). They will have to learn **behavioral management techniques** designed to reduce the rigid and stereotyped behaviors of the disorder and improve social functioning. Many children with autism require special education or specialized day programs for behavior management.

Autistic children with a comorbid seizure disorder are treated with anticonvulsants. Low doses of **neuroleptics** (e.g., haloperidol) and some mood stabilizers and antidepressants have been shown to help decrease aggressive or self-harming behaviors.

ATTENTION-DEFICIT/ HYPERACTIVITY DISORDER (ADHD)

ADHD is characterized by a persistent and dysfunctional pattern of overactivity, impulsiveness, inattention, and distractibility.

KEY POINTS

1. Autistic disorder is a rare pervasive developmental disorder characterized by impaired social interactions, impaired ability to communicate, and restricted repertoire of activities and interests.
2. It is familial and is associated with fragile X syndrome and tuberous sclerosis.
3. The comorbid seizure rate is 25%.
4. The mental retardation rate is 75%.
5. Autistic disorder is managed by behavioral techniques and neuroleptics.

Etiology

The disorder runs in families and cosegregates with mood disorders, substance use disorders, learning disorders, and antisocial personality disorder. Families with a child diagnosed with ADHD are more likely than those without ADHD offspring to have family members with the above-mentioned disorders.

The etiology of the disorder is unknown, but perinatal injury, malnutrition, and substance exposure have all been implicated. Many children with ADHD have abnormalities of sleep architecture (decreased rapid eye movement latency, increased delta latency), EEG, and soft neurologic signs.

Epidemiology

The prevalence of ADHD in school-age children is estimated to be 3% to 5%. The boy-girl ratio ranges from 4 : 1 in the general population to 9 : 1 in clinical settings. Boys are much more likely than girls to be brought to medical attention.

Clinical Manifestations

History, Mental Status Examination, and Laboratory Tests

To meet criteria for ADHD, a child must evidence the onset of inattentive or hyperactive symptoms before age 7; symptoms must also be present in two or more settings (e.g., school, home). Symptoms in only one setting suggest an environmental or psychodynamic cause.

Preschool-age children are usually brought for evaluation when they are unmanageable at home.

Typically, they stay up late, wake up early, and spend most of their waking hours in various hyperactive and impulsive activities. Children with a great deal of hyperactivity may literally run about the house, cause damage, and wreak havoc.

When these children enter school, their difficulties with attention become more obvious. They appear to not follow directions, forget important school supplies, fail to complete homework or in-class assignments, and attempt to blurt out answers to teachers' questions before being called on. As a result of their inattention and hyperactivity, these children often become known as "troublemakers." They fall behind their peers academically and socially.

Evaluation of the child involves gathering a careful history from parents and teachers (the latter usually through report cards and written reports). The child's behavior with and without the parent is carefully observed during psychiatric assessment. Informal testing is carried out by having the child attempt to complete a simple puzzle, write the letters of the alphabet, distinguish right from left, and recognize letters traced on the palms (graphesthesia). Physical examination, particularly focusing on neurologic function, is imperative. No specific laboratory or cognitive tests are helpful in making the diagnosis.

Differential Diagnosis

It is important to distinguish symptoms of ADHD from age-appropriate behaviors in active children (running about, being noisy, etc.). Children can also appear inattentive if they have a low or a high IQ and the environment is overstimulating or understimulating, respectively. In either instance, IQ testing and careful evaluation of the school program will clarify the diagnosis.

Children with oppositional defiant disorder may resist work or school tasks because of an unwillingness to comply with others' demands but not out of difficulty in attention.

Children with other mental disorders (e.g., mood disorder, anxiety disorder) can exhibit inattention but typically not before age 7. The child's history of school adjustment is not usually characterized by teacher or parent reports of inattentive, disruptive behavior.

Symptoms that resemble ADHD can occur in children before age 7, but the etiology is typically a side effect of a medication (e.g., bronchodilators) or a psychotic or pervasive developmental disorder; these children are not considered to have ADHD. Of course, ADHD may be comorbid with any of the above disorders. A dual diagnosis is made only when it is needed to explain the full clinical picture.

Management

The management of ADHD involves a combination of somatic and behavioral treatments. Most children with ADHD respond favorably to **psychostimulants**. Methylphenidate is the first-line agent, followed by D-amphetamine. Clinicians try to use the smallest effective dose and to restrict use to periods of greatest need (i.e., the school day) because psychostimulants have undesirable long-term physical effects (weight loss and inhibited body growth). Some children can be treated effectively with agents that raise norepinephrine in the brain, such as bupropion (Wellbutrin) or atomoxetine (Strattera) a norepinephrine reuptake inhibitor.

Behavioral management techniques include positive reinforcement, firm limit setting, and techniques for reducing stimulation (e.g., one playmate at a time; short, focused tasks).

KEY POINTS

1. ADHD disorder is characterized by inattentiveness and hyperactivity occurring in multiple settings.
2. Symptoms must begin before age 7.
3. ADHD is more common in boys (4:1).
4. For a diagnosis of ADHD, other causes of inattentiveness or hyperactivity must be ruled out.
5. ADHD is managed with psychostimulants and behavioral techniques.

CONDUCT DISORDER AND OPPOSITIONAL DEFIANT DISORDER

Conduct disorder is defined as a repetitive and persistent pattern of behavior in which the basic rights of others or important age-appropriate societal norms or rules are violated. Disordered behaviors include aggression toward people or animals, destruction of property, deceitfulness, theft, or serious vio-

lations of rules (school truancy, running away). Conduct disorder is the childhood equivalent of adult antisocial personality disorder (ASP). It is the most common disorder seen in outpatient psychiatric clinics and is frequently seen comorbidly with ADHD or learning disorders. Adoption studies show a genetic predisposition, but psychosocial factors play a major role. Parental separation or divorce, parental substance abuse, severely poor or inconsistent parenting, and association with a delinquent peer group have been shown to have some relationship to the development of conduct disorder.

Treatment involves individual and family therapy. Some children may need to be removed from the home and placed in foster care. Parents who retain custody of a child with conduct disorder are taught limit setting, consistency, and other behavioral techniques. Medications are used only to treat a comorbid ADHD or mood disorder but not for the conduct disorder itself. The long-term outcome depends on the severity of the disorder and the degree and type of comorbidity. Twenty-five percent to 40% of children with conduct disorder go on to have adult ASP.

Oppositional defiant disorder is diagnosed in children with annoying, difficult, or disruptive behavior when the frequency of the behavior significantly exceeds that of other children his or her mental age (or that is less tolerated in the child's particular culture). It is a relatively new diagnosis that is meant to describe children with behavior problems that do not meet criteria for full-blown conduct disorder. Management emphasizes individual and family counseling.

KEY POINTS

1. Conduct disorder is the childhood equivalent of ASP.
2. It is defined by observable measurable behaviors.
3. Conduct disorder is the most common diagnosis in outpatient child psychiatric clinics.
4. It is managed by limit setting, consistency, and behavioral techniques.
5. Oppositional defiant disorder is a less severe form of conduct disorder.

TOURETTE'S DISORDER

Tourette's disorder is a rare disorder in which the child demonstrates multiple involuntary motor and vocal tics. A **tic** is a sudden, rapid, recurrent, nonrhythmic, stereotyped motor movement or vocalization.

Epidemiology

Tourette's disorder affects 0.4% of the population. There is a 3 : 1 male-female ratio.

Etiology

Tourette's disorder is highly familial and appears to frequently co-occur with obsessive-compulsive disorder. Despite evidence of genetic transmission in some families, no gene (or genes) has yet been discovered to explain the etiology of the disorder.

Clinical Manifestations

History, Mental Status and Physical Examinations

The patient or family usually describes an onset in childhood or early adolescence before age 18. Vocal tics are usually loud grunts or barks but can involve shouting words; the words are sometimes obscenities (coprolalia). The patient describes being aware of shouting the words, being able to exert some control over them, but being overwhelmed by an uncontrollable urge to say them. Motor tics can involve facial grimacing, tongue protrusion, blinking, snorting, or larger movements of the extremities or whole body. Motor tics typically antedate vocal tics; barks or grunts typically antedate verbal shouts. The motor tics are not painful.

Differential Diagnosis

A careful neurologic evaluation should be performed to rule out other causes of tics. Wilson's disease and Huntington's disease are the principal differential diagnostic disorders. An EEG should be performed to rule out a seizure disorder. Careful evaluation for other comorbid psychiatric illnesses should be performed. Stimulants used to treat other psychiatric disorders may unmask tics.

Management

Treatment typically involves the use of low doses of high-potency neuroleptics such as haloperidol or pimozide. The child and his or her family should receive education and **supportive psychotherapy** aimed at minimizing the negative social consequences (e.g., embarrassment, shame, isolation) that occur with this disorder.

KEY POINTS

1. Tourette's disorder is a tic disorder.
2. It is rare and more common in males (3:1).
3. When diagnosing Tourette's disorder, Wilson's and Huntington's diseases must be ruled out.
4. It is treated with high-potency neuroleptics and patient/family support.

Chapter

8 Cognitive Disorders

The cognitive disorders are delirium, dementia, and amnestic disorders. Table 8-1 lists the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition, classification of cognitive disorders.

DELIRIUM

Delirium is a reversible state of global cortical dysfunction characterized by alterations in **attention** and **cognition** and produced by a definable precipitant. Delirium is categorized by its etiology (see Table 8-1) as due to general medical conditions, substance-related, or multifactorial in origin.

Etiology

Delirium is a syndrome with many causes. Most frequently, delirium is the result of a general medical condition; substance intoxication and withdrawal also are common causes. Structural central nervous system lesions can also lead to delirium. Table 8-2 lists common general medical and substance-related causes of delirium. Delirium is often multifactorial and may be produced by a combination of minor illnesses and minor metabolic derangements (e.g., mild anemia, mild hyponatremia, mild hypoxia, and urinary tract infection, especially in an elderly person). Common medical causes of delirium include metabolic abnormalities such as hyponatremia, hypoxia, hypercapnia, hypoglycemia, and hypercalcemia. Infectious illnesses, especially urinary tract infections, pneumonia, and meningitis, are often implicated. The common substance-induced causes of delirium are alcohol or benzodiazepine withdrawal and benzodiazepine and anticholinergic drug toxicity, although a great number of commonly used medications, prescribed and over the counter, can produce delirium. Other conditions predisposing to delirium include old age, fractures, and preexisting dementia.

Epidemiology

The exact prevalence in the general population is unknown. Delirium occurs in 10% to 15% of general medical patients older than age 65 and is frequently seen postsurgically and in intensive care units. Delirium is equally common in males and females.

Clinical Manifestations

History and Mental Status Examination

History is critical in the diagnosis of delirium, particularly in regard to the time course of development of the delirium and to the prior existence of dementia or other psychiatric illness. Key features of delirium are

1. Disturbance of consciousness, especially attention and level of arousal;
2. Alterations in cognition, especially memory, orientation, language, and perception;
3. Development over a period of hours to days; and
4. Presence of medical or substance-related precipitants.

In addition, sleep-wake cycle disturbances and psychomotor agitation may occur. Delirium is often difficult to separate from dementia, in part because dementia is a risk factor for delirium (and thus they frequently co-occur) and in part because there is a great deal of symptom overlap, as outlined in

TABLE 8-1

Cognitive Disorders

Delirium	Dementia	Amnestic
General medical	Alzheimer's type	General medical
Substance-related	Vascular origin	Substance-related
Multifactorial	HIV-related	
	Head trauma-related	
	\Parkinson's-related	
	Huntington's-related	
	Pick's-related	
	Creutzfeldt-Jakob–related	
	General medical origin	
	Substance-related	
	Multifactorial	

TABLE 8-2

Common Causes of Delirium

General Medical	Substance-Related
Infectious	Intoxication
Urinary tract infections	Alcohol
Meningitis	Hallucinogens
Pneumonia	Opioids
Sepsis	Marijuana
Metabolic	Stimulants
Hyponatremia	Sedatives
Hepatic encephalopathy	Withdrawal
Hypoxia	Alcohol
Hypercarbia	Benzodiazepines
Hypoglycemia	Barbiturates
Fluid imbalance	Medication-induced
Uremia	Anesthetics
Hypercalcemia	Anticholinergics
Postsurgical	Meperidine
Hyper/hypothyroidism	Antibiotics
Ictal/postictal	Toxins
Head trauma	Carbon monoxide
Miscellaneous	Organophosphates
Fat emboli syndrome	
Thiamine deficiency	
Anemia	

Table 8-3. Key differentiating factors are the time course of development of the mental status change (especially if the patient did not have a prior dementia) and the presence of a likely precipitant for the mental status change. Individuals with delirium may also display periods of complete lucidity interspersed with periods of confusion, whereas in dementia, the deficits are generally more stable. In both conditions, there may be nocturnal worsening of symptoms with increased agitation and confusion ("sundowning").

The diagnosis of delirium is complicated by the fact that there are no definitive tests for delirium. The workup for delirium includes a thorough history and mental status examination, a physical examination, and laboratory tests targeted at identifying general medical and substance-related causes. These should include urinalysis, complete chemistry panel, complete blood count, and oxygen saturation. Additional workup might entail chest X ray, arterial blood gas (ABG), neuroimaging, or electroencephalogram (EEG). EEG may reveal nonspecific diffuse slowing. The presence of a delirium is associated with a 1-year mortality rate of 40% to 50%.

Differential Diagnosis

Delirium should be differentiated from dementia (although both can be present at the same time), psychotic or manic disorganization, and status complex partial epilepsy.

Management

The treatment of delirium involves keeping the patient safe from harm while addressing the delirium. In the case of delirium due to a general medical illness, the underlying illness must be treated; in substance-related delirium, treatment involves removing the offending drug (either drugs of abuse or medications) or the appropriate replacement and tapering of a cross-reacting drug to minimize withdrawal. Delirium in the elderly is frequently multifactorial

TABLE 8-3

Delirium versus Dementia

	Delirium	Dementia
Onset	Hours to days	Weeks to years
Course/duration	Fluctuates within a day. May last hours to weeks*	Stable within a day. May be permanent, reversible, or progressive over weeks to years
Attention	Impaired	May be impaired
Cognition	Impaired memory, orientation, language	Impaired memory, orientation, language, executive function
Perception	Hallucinations, delusions, misinterpretations	Hallucinations, delusions
Sleep/wake	Disturbed, may have complete day/night reversal	Disturbed, may have no pattern
Mood/emotion	Labile affect	Labile affect; mood disturbances
Sundowning	Frequent	Frequent
Identified precipitant	Likely precipitant is present	Identifiable precipitant not required

*DSM-IV does not specify a limit for the duration for delirum; clinical experience suggests resolution within days to weeks, in most cases.

and requires correction of a multitude of medical conditions.

In addition to addressing the cause of a delirium, oral, intramuscular, or intravenous haloperidol is of great use in treating agitation. Low doses of short-acting benzodiazepines can be used sparingly. Providing the patient with a brightly lighted room with orienting cues such as names, clocks, and calendars is also useful.

KEY POINTS

1. Delirium is a disorder of attention and cognition.
2. It has an abrupt onset and a variable course.
3. It has an identifiable precipitant.
4. 1-year mortality rate is greater than 40%.

DEMENTIA

Dementia is characterized by the presence of **memory impairment** in the presence of other **cognitive defects**. Dementia is categorized according to its etiology (see Table 8-1). It can arise as a result of a specific disease, for example Alzheimer's disease or HIV infection; a general medical condition; or a substance-related condition; or it can have multiple etiologies. The definitive cause may not be determined until autopsy.

Etiology

Generally, the etiology of dementia is brain neuronal loss that may be due to neuronal degeneration or to cell death secondary to trauma, infarction, hypoxia, infection, or hydrocephalus. Table 8-1 lists the major discrete illnesses known to produce dementia. In addition, there are a large number of general medical, substance-related, and multifactorial causes of dementia.

Epidemiology

The prevalence of dementia of all types is about 2% to 4% after age 65, increasing with age to a prevalence of about 20% after age 85. Specific epidemiologic factors relating to disease-specific causes of dementia are listed in Table 8-4.

Clinical Manifestations

History and Mental Status Examination

Dementia is diagnosed in the presence of multiple cognitive defects not better explained by another diagnosis. The presence of memory loss is required; in addition, one or more cognitive defects in the categories of aphasia, apraxia, agnosia, and disturbance in executive function must be present. Table 8-3 compares characteristics of dementia to those of delirium. Dementia often develops insidiously over the course of weeks to years (although it may be

TABLE 8-4

Specific Diseases Associated with Dementia

Disease	Description
Alzheimer's	Most common cause of dementia, accounts for greater than 50% of all cases. Risk factors are familial, Down syndrome, prior head trauma, increasing age. Clinically, it is a diagnosis of exclusion. Post-mortem pathology reveals cortical atrophy, neurofibrillary tangles, amyloid plaques, granulovacuolar degeneration, loss of basal forebrain cholinergic nuclei. Course is progressive, death occurs 8–10 years after onset.
Vascular	Second most common cause of dementia. Risk factors are cardiovascular and cerebrovascular disease. Neuroimaging reveals multiple areas of neuronal damage. Neurological exam reveals focal findings. Course can be rapid onset or more slowly progressive. Deficits are not reversible, but progress can be halted with appropriate treatment of vascular disease.
HIV	Limited to those cases caused by direct action of HIV on the brain; associated illnesses, such as meningitis, lymphoma, toxoplasmosis producing dementia are categorized under dementia due to general medical conditions. Primarily affects white matter and cortex.
Head trauma	Most common among young males. Extent of dementia is determined by degree of brain damage. Deficits are stable unless there is repeated head trauma.
Parkinson's	Occurs in 20–60% of individuals with Parkinson's disease. The most likely pathological finding on autopsy is Lewy body disease. Bradyphrenia (slowed thinking) is common. Some individuals also have pathology at autopsy consistent with Alzheimer's dementia.
Huntington's	Risk factors are familial, autosomal dominant on chromosome 4. Onset commonly in mid 30s. Emotional lability is prominent. Caudate atrophy is present on autopsy.
Pick's	Onset at age 50–60. Frontal and temporal atrophy are prominent on neuroimaging. The dementia responds poorly to psychotropic medicine.
Creutzfeldt-Jakob	Ten percent of cases are familial. Onset age 40–60. Prion is thought to be agent of transmission. Clinical triad of dementia, myoclonus, and abnormal EEG. Rapidly progressive. Spongiform encephalopathy is present at autopsy.

abrupt after head trauma or vascular insult). Individuals with dementia usually have a stable presentation over brief periods of time, although they may also have nocturnal worsening of symptoms ("sundowning"). Memory impairment is often greatest for short-term memory. Recall of names is frequently impaired, as is recognition of familiar objects. Executive functions of organization and planning may be lost. Paranoia, hallucinations, and delusions are often present. Eventually, individuals with dementia may become mute, incontinent, and bedridden.

Differential Diagnosis

Dementia should be differentiated from delirium. In addition, dementia should be differentiated from those developmental disorders (such as mental retardation) with impaired cognition. Individuals with major depression and psychosis can appear demented; they warrant a diagnosis of dementia only if their cognitive deficits cannot be fully attributed to the primary psychiatric illness.

A critical component of differential diagnosis in dementia is to distinguish pseudodementia associated with depression. Although there are many precise criteria for separating the two disorders, neuropsychological testing may be needed to make an accurate diagnosis. In pseudodementia, mood symptoms are prominent and patients may complain extensively of memory impairment. They characteristically give "I don't know" answers to mental status examination queries but may answer correctly if pressed. Memory is intact with rehearsal in pseudodementia, but not in dementia.

Management

Dementia from reversible, or treatable, causes should be managed first by treating the underlying cause of the dementia; rehabilitation may be required for residual deficits. Reversible (or partially reversible) causes of dementia include normal pressure hydrocephalus; neurosyphilis; HIV infection; and thiamine,

folate, vitamin B_{12}, and niacin deficiencies. Vascular dementias may not be reversible, but their progress can be halted in some cases. Nonreversible dementias are usually managed by placing the patient in a safe environment and by medications targeted at associated symptoms. Tacrine, an acetylcholinesterase inhibitor, has some efficacy in treating memory loss in dementia of the Alzheimer's type. High-potency antipsychotics (in low doses) are used when agitation, paranoia, and hallucinations are present. Low-dose benzodiazepines and trazodone are often used for anxiety, agitation, or insomnia.

KEY POINTS

1. Dementia is a disorder of memory impairment coupled with other cognitive defects.
2. It has a gradual onset and progressive course.
3. It may be caused by a variety of illnesses.
4. Dementia predisposes to delirium.

AMNESTIC DISORDERS

Amnestic disorders are isolated disturbances of memory without impairment of other cognitive functions. They may be due to a general medical condition or substance related.

Etiology

Amnestic disorders are caused by general medical conditions or substance use. Common general medical conditions include head trauma, hypoxia, herpes simplex encephalitis, and posterior cerebral artery infarction. Amnestic disorders often are associated with damage of the mammillary bodies, fornix, and hippocampus. Bilateral damage to these structures produces the most severe deficits. Amnestic disorders due to substance-related causes may be due to substance abuse, prescribed or over-the-counter medications, or accidental exposure to toxins. Alcohol abuse is a leading cause of substance-related amnestic disorder. Persistent alcohol use may lead to thiamine deficiency and induce Wernicke-Korsakoff's syndrome. If properly treated, the acute symptoms of ataxia, abnormal eye movements, and confusion may resolve, leaving a residual amnestic disorder called Korsakoff's psychosis (alcohol-induced persistent amnestic disorder).

Epidemiology

Individuals affected by a general medical condition or alcoholism are at risk for amnestic disorders.

Clinical Manifestations

History and Mental Status Examination

Amnestic disorders present as **deficits in memory**, either in the inability to recall previously learned information or the inability to retain new information. The cognitive defect must be limited to memory alone; if additional cognitive defects are present, a diagnosis of dementia or delirium should be considered. In addition to defect in memory, there must be an identifiable cause for the amnestic disorder (i.e., the presence of a general medical condition or substance use).

Differential Diagnosis

Delirium and dementia are the major differential diagnostic considerations. Amnestic disorders are distinguished from dissociative disorders on the basis of etiology. By definition, amnestic disorders are due to a general medical condition or substance.

Management

The general medical condition is treated whenever possible to prevent further neurologic damage; in the case of a substance-related amnestic disorder, avoiding reexposure to the substance responsible for the amnestic disorder is critical. Pharmacotherapy may be directed at treating associated anxiety or mood difficulties. Patients should be placed in a safe, structured environment with frequent memory cues.

KEY POINTS

1. Amnestic disorders are disorders in memory alone.
2. They are caused by identifiable precipitants.
3. Amnestic disorders are reversible in some cases.

Chapter

9 Miscellaneous Disorders

Miscellaneous disorders does not refer to any official *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) classification but rather to psychiatric diagnoses not covered elsewhere in this book. Generally, these diagnoses are either less common, less understood, or less frequently the focus of psychiatric practice than the disorders previously covered. Although many of these disorders are not uncommon, they seldom come to psychiatric attention for a variety of reasons (e.g., they may be treated by other medical specialists, patients may not mention them, or they may not be detected adequately). Table 9-1 lists the categories of disorders discussed in this chapter.

DISSOCIATIVE DISORDERS

Dissociative disorders are characterized by disturbances in the integration of mental functions. These disturbances are manifested by loss of memory for personal information or identity, division of consciousness and personality into separate parts, and altered perception of the environment or sense of reality. Table 9-2 lists DSM-IV defined dissociative disorders.

Dissociative Amnesia

In dissociative amnesia, an individual develops a temporary inability to recall important personal information. The amnesia is more extensive than forgetfulness and is not caused by another medical or psychiatric condition (e.g., head trauma). The inability to recall information may take several forms. In **localized amnesia,** information is lost for a specific time period (e.g., a time associated with trauma). In **selective amnesia,** some information during a given time period is retained but other information is lost. In **generalized amnesia,** personal information is lost for the entire life span. In **continuous amnesia,** there is an inability to recall information from a single point in time to the present. In **systematized amnesia,** particular categories of information are lost to retrieval.

Dissociative amnesia is more common in people exposed to trauma, for example, exposure to battle or natural disaster.

Dissociative Fugue

Dissociative fugue is an amnestic disorder characterized by an individual's sudden unexplained travel away from home, coupled with amnesia for his or her identity. In this condition, patients do not appear mentally ill or otherwise impaired in any other mental function, including memory. In fact, patients are quite capable of negotiating the complexities of travel and interaction with others. In rare cases, an individual will establish a completely new identity in the new home. Dissociative fugue is typically precipitated by a severe trauma or stressor and eventually remits without treatment.

Dissociative Identity Disorder

Dissociative identity disorder (formerly called multiple personality disorder) is a controversial diagnosis in psychiatry. The diagnosis of dissociative identity disorder requires the presence of two or more separate personalities (alters) that recurrently take control of an individual's behavior. Individuals with this disorder often have amnesia for important personal information (also known as "losing time"). The various personalities (the average number by available surveys is seven distinct personalities) may be

TABLE 9-1

Miscellaneous Disorders

Dissociative disorders
Somatoform disorders
Adjustment disorders
Sexual and gender identity disorders
Sleep disorders
Factitious disorders/malingering

TABLE 9-2

Dissociative Disorders

Dissociative amnesia	Temporary inability to recall important personal information; more serious than simple forgetfulness
Dissociative fugue	Amnesia for one's identity coupled with sudden unexplained travel away from home
Dissociative identity disorder	Presence of two or more separate personalities that recurrently take control of a person's behavior
Depersonalization disorder	Pervasive sense of being detached from or being outside of one's body

unaware of each other's existence and thus may be quite confused as to how they arrived at certain places or why they cannot recall personal events. At other times, one or more personalities may be aware of the others, a condition known as coconsciousness. Some personalities may display conversion symptoms or self-mutilating behavior. The alters may be of varying ages and different genders and demeanors.

Dissociative identity disorder is most common in females and has a chronic course. Individuals with dissociative identity disorder are highly suggestible and easily hypnotized. Most report a childhood history of severe physical or sexual abuse. Satanic or cult abuse reports are also common. In many cases, these reports of abuse cannot be verified, leading many clinicians to believe that individuals with dissociative identity disorder may suffer from memories of events that did not occur. Whether these memories are true or false, they cause a great deal of suffering.

Disagreement over the very nature of dissociative identity disorder has led to divergent treatment opinions. Some clinicians believe that ignoring the different personalities will cause them to recede, based on the notion that the easy suggestibility of these patients will lead to reinforcement of alters if they are discussed. Others believe that long-term psychotherapy, exploring the various personalities and integrating them into a whole person, is the treatment of choice.

Depersonalization Disorder

Depersonalization disorder is characterized by "persistent or recurrent experiences of feeling detached from and as if one is an outside observer of one's mental processes or body" (DSM-IV). Individuals with this disorder may complain of a sense of detachment, of feeling mechanical or automated, and of absence of affect or sensation. Individuals with depersonalization disorder are easily hypnotized and prone to dissociate.

SOMATOFORM DISORDERS

Somatoform disorders are characterized by the presence of physical signs or symptoms without medical cause. In addition, they are *not* willfully produced by the individual. The somatoform disorders are listed and defined in Table 9-3 (adapted from DSM-IV).

Somatization Disorder

Somatization disorder is diagnosed when an individual has multiple medical complaints that are not the result of medical illness. The specific DSM-IV criteria are narrow and specific, requiring

- Pain in four different body sites or involving four different body functions;
- Two gastrointestinal symptoms (other than pain);
- One sexual symptom (other than pain); and
- One pseudoneurologic symptom (other than pain).

In addition, some symptoms must have begun before age 30 and persisted for several years. Individuals with somatization disorder often have a history of complex medical and surgical treatments that may actually lead to iatrogenic complications of treatment. Patients with somatoform disorder frequently have multiple physicians, make frequent office and hospital visits, and may seek disability because of their conviction that they are severely and chronically medically ill.

TABLE 9-3

Somatoform Disorders	
Somatization disorder	Chronic multiple medical complaints that include pain, gastrointestinal disturbance, sexual symptoms, and pseudoneurologic symptoms that are not due to a medical illness.
Undifferentiated somatoform disorder	A less severe form of somatization disorder; involves fewer complaints and briefer course.
Conversion disorder	Complaints involving sensory (such as numbness) and voluntary motor (such as paralysis) function that are not due to neurologic dysfunction.
Pain disorder	Pain is the major complaint. If medical causes are present, psychological factors have a major role in mediating the expression and impact of pain.
Hypochondriasis	Preoccupation with having a serious disease based on a misinterpretation of bodily function and sensation.
Body dysmorphic disorder	Excessive concern with a perceived defect in appearance.

This disorder is more common in females (approximately 80% of cases), and its incidence is increased in first-degree relatives of those with somatization disorder. Familial and genetic studies have also shown that male relatives of individuals with somatization disorder have an increased incidence of antisocial personality disorder (ASP) and substance abuse. Adoption studies suggest genetic influences in this disorder.

Various theories have been proposed to explain this disorder. Early psychoanalytic work focused on repressed instincts as causative; more modern theorists propose that somatization symptoms may represent a means of nonverbal interpersonal communication. Biologic findings have revealed abnormal cortical function in some individuals with this disorder.

ADJUSTMENT DISORDERS

According to DSM-IV, adjustment disorders are symptoms (changes in emotional state or behaviors) that arise in response to an identified psychosocial stressor that is out of proportion to what is expected in usual human experience. Adjustment disorder is not diagnosed if the symptoms occurred in response to a psychosocial stressor so severe that an individual meets criteria for another axis I disorder (e.g., major depression). Symptoms in response to bereavement do not meet criteria for the diagnosis of adjustment disorder. Adjustment disorders occur within 3 months of the identified stressor and usually resolve within 6 months, unless the stressor becomes chronic.

SEXUAL AND GENDER IDENTITY DISORDERS

The DSM-IV classifies these disorders into sexual dysfunctions, paraphilia, and gender identity disorders.

Sexual Dysfunctions

Sexual dysfunctions are sexual disorders associated with alterations in the sexual response cycle (Table 9-4) or with pain associated with sexual activity. The specific sexual dysfunctions are defined in Table 9-5.

Paraphilias

Paraphilias include sexual disorders related to culturally unusual sexual activity (Table 9-6). A key cri-

TABLE 9-4

Sexual Response Cycle	
Desire	Initial stage of sexual response; consists of sexual fantasies and the urge to have sex.
Excitement	Consists of physiologic arousal and feeling of sexual pleasure.
Orgasm	Peaking sexual pleasure; usually associated with ejaculation in males.
Resolution	Physiologic relaxation associated with sense of well-being. In males, there is usually a refractory period for further excitement and orgasm.

TABLE 9-5

Specific Sexual Dysfunctions

Sexual desire disorders	
Hypoactive sexual desire disorder	Sexual fantasy and desire for sex are very low or absent.
Sexual aversion disorder	Aversion to genital sexual contact with another person.
Sexual arousal disorders	
Female sexual arousal disorder	Inadequate vaginal lubrication and inadequate engorgement of external genitalia.
Male erectile disorder	Inability to attain or maintain an erection.
Orgasmic disorders	
Female orgasmic disorder	Orgasm is absent or delayed. Sexual excitement phase is normal.
Male orgasmic disorder	Orgasm is absent or delayed. Sexual excitement phase is normal.
Premature ejaculation	Orgasm and ejaculation occur early and with minimal stimulation.
Sexual pain disorders	
Dyspareunia	Genital pain in association with sexual intercourse.
Vaginismus	Involuntary contraction of external vaginal musculature as a result of attempted penetration.
Sexual dysfunction due to a general medical condition	
Substance-induced sexual dysfunction	

TABLE 9-6

Paraphilias

Exhibitionism	Sexual excitement is derived from exposing one's genitals to a stranger.
Fetishism	Nonliving objects are the focus of intense sexual arousal in fantasy or behavior.
Frotteurism	Sexual excitement is derived by rubbing one's genitals against or by sexually touching a nonconsenting stranger.
Pedophilia	Sexual excitement is derived from fantasy or behavior involving sex with prepubescent children.
Sexual masochism	Sexual excitement is derived from fantasy or behavior involving being the recipient of humiliation, bondage, or pain.
Sexual sadism	Sexual excitement is derived from fantasy or behavior involving inflicting suffering/humiliation on another.
Transvestic fetishism	Sexual excitement (in heterosexual males) is derived from fantasy or behavior involving wearing women's clothing.
Voyeurism	Sexual excitement is derived from fantasy or behavior involving the observation of unsuspecting individuals undressing, naked, or having sex.

terion for the diagnosis of a paraphilia (as in all psychiatric disorders) is that the disorder must cause an individual to experience significant distress or impairment in social or occupational functioning. In other words, an individual with unusual sexual practices who does not suffer significant distress or impairment would not be diagnosed with a psychiatric illness.

Gender Identity Disorder

Gender identity disorder remains a controversial diagnosis in psychiatry. Individuals with this disorder experience distress and interpersonal impairment as a result of their desire to be a member of the opposite sex. Criteria for the diagnosis require a pervasive cross-gender identification and persistent discomfort with one's assigned sex. In addition, the diagnosis is made only in those individuals who do not have an

intersex condition (e.g., ambiguous genitalia). Children with this disorder may engage in gender-atypical play; adults may assume the societal role, dress, and behavior associated with the opposite sex. In addition, patients with gender identity disorder may seek sex reassignment surgery and hormonal supplements. Individuals with gender identity disorder appear to have the same range of sexual orientations as do persons without this disorder.

SLEEP DISORDERS

Sleep disorders are illnesses related to alterations in the sleep-wake cycle (Table 9-7) and often have effects on mood, cognitive, somatic, and general performance. Table 9-8 outlines the DSM-IV classification of sleep disorders. Sleep disorders are categorized into primary and secondary sleep disorders. Primary sleep disorders are those disorders occurring as a direct result of disturbances in the sleep-wake cycle. They are divided into two categories: dyssomnias and parasomnias. Secondary sleep disorders are a consequence of other mental disorders (e.g., depression) due to general medical conditions (e.g., somatic pain) or substance use (e.g., caffeine).

Dyssomnias

Dyssomnias are five primary sleep disorders consisting of disturbances in initiating and maintaining sleep, feeling rested or refreshed after sleep, or sleeping excessively. Table 9-9 defines the DSM-IV determined key characteristics of each disorder.

Parasomnias

Parasomnias are a triad of sleep disorders associated with complex behavioral events that occur during sleep or that arouse a person from sleep. The disorders are defined in Table 9-9.

FACTITIOUS DISORDERS

A factitious disorder is one in which an individual willfully produces signs or symptoms of a medical or psychiatric illness to assume the sick role and its associated gratifications. This should be differentiated from **somatoform disorders** (which are not willful) and **malingering**, which is simply lying about signs or symptoms to obtain gains different from those obtained by assuming the sick role (e.g., to avoid the military or for monetary gain).

TABLE 9-7

Sleep Stages

Nonrapid eye movement (NREM)	
Stage 0	Awake.
Stage 1	Very light* sleep, transition from wakefulness to sleep. Drowsy.
Stage 2	Medium depth of sleep, occupies about half the night in adults. Serves as a transition stage between rapid eye movement (REM) and delta sleep. EEG demonstrates sleep spindles and k-complexes.
Delta	Slow wave sleep, composed of stages 3 and 4, occupies 10–30% of total sleep.
Stage 3	Consists of a moderate amount of delta wave activity; deeper sleep than stage 2.
Stage 4	Increased delta wave activity over stage 3. Very deep stage of sleep.
REM	Dream sleep. EEG is active, mimicking that of the waking stage. Depth of sleep is greater than stage 2 but probably less than delta.

* Depth of sleep as used here is not a precise term but generally refers to ease of arousability (i.e., how hard would it be to awaken an individual from a particular stage). However, the ease of arousability is due in part to the type of stimulus used (e.g., noise versus touch).

TABLE 9-8

Sleep Disorders

Primary Sleep Disorders	Secondary Sleep Disorders
Dyssomnias	Sleep disorder related to another mental disorder
Primary insomnia	Sleep disorder due to a general medical condition
Primary hypersomnia	Substance-induced sleep disorder
Narcolepsy	
Breathing-related sleep disorder	
Circadian rhythm sleep disorder	
Parasomnias	
Nightmare disorder	
Sleep terror disorder	
Sleepwalking disorder	

TABLE 9-9

Primary Sleep Disorders

Dyssomnias	
Primary insomnia	Difficulty falling asleep or staying asleep, or sleeping but feeling as if one has not rested during sleep.
Primary hypersomnia	Excess sleepiness, either sleeping too long at one setting or persistent daytime sleepiness not relieved by napping.
Narcolepsy	Sleep attacks during the daytime coupled with REM sleep intrusions or cataplexy (sudden, reversible bilateral loss of skeletal muscle tone). Daytime naps relieve sleepiness.
Breathing-related sleep disorder	Abnormal breathing during sleep leads to sleep disruption and daytime sleepiness.
Circadian rhythm sleep disorder	Sleep disturbance due to a mismatch between a person's intrinsic circadian rhythm and external sleep-wake demands.
Parasomnias	
Nightmare disorder	Repeated episodes of scary dreams that wake a person from sleep, usually occur during *REM* sleep.
Sleep terror disorder	Repeated episodes of apparent terror during sleep; individuals may sit up, scream, or cry out and appear extremely frightened. They do not usually awaken during the attack. Occurs during *delta* sleep.
Sleepwalking disorder	Recurrent sleepwalking, often coupled with other complex motor activity.

Chapter

10 Special Clinical Settings

SUICIDE ATTEMPTS

Epidemiology

Suicide is the eighth leading cause of death in the United States. Approximately 75 people commit suicide each day in the United States (25,000 per year). Many more people attempt suicide. The overall suicide rate has remained stable in the United States during the past 15 years. Although the rate of suicide in teen-agers aged 15 to 19 is low compared with the general adult population, the rate of teen suicide has risen dramatically in the last 50 years.

Risk Factors

Studies have demonstrated that the overwhelming majority of people who commit suicide have a mental illness (most often a mood disorder or chronic alcoholism). The first-degree relatives of people who have committed suicide are at a much higher risk of committing suicide themselves. Gay and lesbian youth have 2 to 3 times the rate of attempted suicide compared to heterosexual adolescents. Suicide risk increases with age. In men, suicides peak after age 45; in women, most suicides occur after age 55. The elderly account for 25% of the suicides, although they represent only 10% of the population. Overall, men are more successful at completing suicide, perhaps because of their more lethal methods (shooting, hanging, jumping); women often overdose or attempt drowning. Married people have a lower risk of suicide than singles. Suicide is more common among higher social classes, whites, and certain professional groups (physicians, dentists, musicians, law enforcement officers, lawyers, and insurance agents). Biologic risk factors include low levels of 5-hydroxyindoleacetic acid in the cerebrospinal fluid of patients who have committed suicide by violent means. Among psychological risk factors, hopelessness has been shown to be one of the most reliable indicators of long-term suicide risk.

Clinical Manifestations

History and Mental Status Examination

Most often, a suicide attempt is self-evident at presentation, either because the patient or family indicates that such an event has occurred or because there is an acute medical or surgical emergency (i.e., overdose or wrist laceration). Always obtain further details from the patient and any witnesses to provide a full history of the antecedents and the act itself. Occasionally, a patient will present to a physician in a more subtle way, with nonspecific complaints. Careful inquiry may reveal that the patient has taken an overdose of a medication with delayed lethality (such as acetaminophen).

Patients who have attempted suicide deserve thorough psychiatric evaluation. Psychiatric history and mental status examination should explicitly address depressive symptoms, such as suicidal thoughts, intent, and plans. The details of the suicide attempt are critical to understanding the risk of a future suicide. Patients who carefully plan the attempt, use particularly violent means, and isolate themselves so as not to be found alive are at particularly high risk of future suicide completion.

Differential Diagnosis

Patients who attempt suicide most commonly suffer from depression, schizophrenia, alcoholism, or personality disorders (or comorbidities of the above). However, patients who do not meet criteria for any of these disorders can and do attempt or commit

suicide, especially if they have any of the risk factors (e.g., hopelessness).

Management

Suicidal ideation should always be taken seriously. Suicidal patients often are fraught with ambivalence over whether to live or die, and intervention and effective treatment can be lifesaving. Most actively suicidal patients require hospitalization on a locked unit for their own safety. Potentially lethal items should be held securely by nursing staff, and the patient should be observed carefully for the risk of elopement. Treatment of the underlying disorder or distress derives from accurate diagnosis (antidepressants or electroconvulsive therapy [ECT] for depression; antipsychotics and/or mood stabilizers for bipolar disorder, psychotic depression, or schizophrenia).

Patients at lower risk of suicide can often be managed as outpatients if close follow-up is available, family members are supportive, and a treatment alliance exists. Frequent meetings with treatment providers, eliminating the means of suicide (firearms, potentially toxic prescription pills), and enlisting spouses, partners, or other family members are essential elements of outpatient treatment.

SPOUSAL ABUSE

Abuse between spouses or partners can take several forms: physical, sexual, and emotional. Physical abuse or battering is most often perpetrated by a male on a female partner, but women do batter men and abuse also occurs in same-sex relationships.

Epidemiology

Spousal abuse is estimated to occur in 2 to 12 million U.S. households. Some studies estimate that nearly one third of all women have been beaten by their husband at least once during their marriage. Many battered women are eventually murdered by their spouses or boyfriends.

Risk Factors

There is a strong association between alcohol abuse and domestic violence. More than 50% of abusers, and many of the abused, have a history of alcohol or other drug abuse. As children, most abusers lived in violent homes where they either witnessed or were themselves victims of battering. The victims of abuse, more often than not, are also products of violent homes. Pregnant women are at elevated risk for spousal abuse, often directed at their abdomens.

Clinical Manifestations

History and Physical and Mental Status Examination

Many victims of abuse are reluctant to report abusive episodes because they fear retaliation, believe they are deserving of abuse, or do not believe that help will be effective. Victims of abuse are often mistreated in ways that prevent them from escaping the abusive relationship. They are intimidated, maligned, coerced, and isolated by the abuser. Attempts to leave an abusive relationship may be thwarted by financial concerns, the welfare of children, fear of being alone, or the threat of further battering.

Patients may present in the company of their abuser for the treatment of "accidental" lacerations, contusions, fractures, or more severe trauma. Unless the patient is asked tactfully in the absence of the abuser, she or he is unlikely to reveal the true cause of injuries.

Physical examination should include examination of the skin for contusions (especially of the face and breasts) and a genital examination. The mental status examination should take into account the appropriateness of the patient's and partner's reactions to the "accident."

Management

The goal of treatment is to end the violence (i.e., both partners must agree to treatment) or to enable the victim to leave the relationship. Either option is difficult to achieve. Social agencies must be enlisted to aid in child protection and custody if the latter option is chosen.

Patients who refuse help should be told what emergency services are available and how to access them. Unfortunately, women are most at risk for serious injury or homicide when they attempt to leave the abusive relationship.

ELDER ABUSE

Approximately 10% of those older than 65 are abused. Victims usually live with their assailants, who

are often their children. Mistreatment includes abuse and neglect and takes physical, psychological, financial, and material forms. The abuser may withhold food, clothing, or other necessities or beat, sexually molest, or emotionally abuse the victim.

As with spousal abuse, the elder person is often reluctant to reveal the abuse. Clinicians should be alert to the signs of abuse. Treatment involves appropriate medical and psychiatric services and social and legal services. Some states mandate reporting of elder abuse.

Chapter

11 Antipsychotics

Antipsychotic medications are used commonly in medical and psychiatric practice. As a class, antipsychotics have in common their blockade of dopamine receptors and their potential for serious side effects if used inappropriately or without careful monitoring. The most commonly prescribed typical and atypical antipsychotics are listed in Table 11-1. Their relative potencies, relative side-effect profiles, and major adverse reactions also are described. **Typical antipsychotics** (also called neuroleptics for their tendency to cause movement disorders) are generally equally effective, although they differ in side-effect profiles and potencies. **Atypical antipsychotics** (e.g., risperidone and clozapine) have fewer extrapyramidal side effects at therapeutic doses compared to typical antipsychotics. Clozapine (and perhaps risperidone) are more effective than typical antipsychotics for the treatment of refractory psychotic disorders.

INDICATIONS

Antipsychotics generally are effective in treating positive psychotic symptoms (e.g., hallucinations, bizarre behavior, delusions) regardless of diagnostic category (Table 11-2). For example, hallucinations in schizophrenia, in Alzheimer's disease, or secondary to cerebral toxicity or injury all respond to antipsychotic medications. Antipsychotics are thought to be less effective (with the exception of clozapine and possibly risperidone, olanzapine, and quetiapine) in treating negative psychotic symptoms (e.g., amotivation, akinesia, affective blunting, social withdrawal). In addition to their role in treating psychotic symptoms, antipsychotics are used to treat some forms of nonpsychotic behavioral dyscontrol (e.g., organic brain syndromes, Alzheimer's, mental retardation), delirium, Tourette's syndrome, and transient psychotic symptoms as they appear in personality disorder patients.

Mechanism of Action

The most prominent theory on the mechanism of action of antipsychotics is the **dopamine hypothesis** of schizophrenia. This hypothesis purports that dopaminergic hyperactivity leads to psychosis. Evidence supporting a role for hyperdopaminergic states in schizophrenia (and presumably other psychotic states) is as follows: antipsychotic potency of traditional antipsychotics correlates highly with their potency of dopamine receptor blockade, individuals with schizophrenia have an increased number of brain dopamine receptors, and dopamine agonist drugs (e.g., amphetamine) can induce or exacerbate existing psychosis.

The action mechanism of antipsychotics is often much broader than simple dopamine blockade, which accounts for the numerous side effects and the finding that their action in the brain is not well correlated with regions thought to give rise to psychotic symptoms. In addition, newer drugs such as clozapine and risperidone have prominent serotonin (5-hydroxytryptamine-2 [$5HT_2$]) receptor blockade. It is unclear whether this serotonin blockade imparts antipsychotic efficacy or simply helps prevent extrapyramidal side effects.

Typical Antipsychotics (Dopamine Antagonists)

The antipsychotic potency of typical antipsychotics (and risperidone) correlates with their affinity for the D_2 receptor. Figure 11-1 is a schematic diagram of the proposed brain pathways affected by typical antipsy-

TABLE 11-1

Commonly Prescribed Antipsychotics

Drug	Therapeutic Dosage Range[1]	Potency[2]	Sedative	Hypotensive	Anticholinergic	EPS	Other Adverse Reactions
Typical antipsychotics (dopamine antagonists)							
Thioridazine (Mellaril)	150–800 mg	100	High	High	High	Low	Pigmentary retinopathy*
Chlorpromazine (Thorazine)	200–800 mg	100	High	High	Med	Low	
Perphenazine (Trilafon)	8–32 mg	10	Low	Low	Low	Med	
Trifluoperazine (Stelazine)	5–20 mg	5	Med	Low	Low	High	
Thiothixene (Navane)	5–30 mg	5	Low	Low	Low	High	
Haloperidol (Haldol)	5–30 mg	2	Low	Low	Low	High	
Fluphenazine (Prolixin)	2–60 mg	2	Med	Low	Low	High	
Atypical antipsychotics (serotonin/dopamine antagonists)							
Clozapine(Clozaril)	150–600 mg	100	High	High	High	Low	Agranulocytosis
Quetiapine (Seroquel)	150–800 mg	150	High	Med	Med	Low	
Ziprasidone (Geodon)	40–180 mg		Low	Low	Low	Low	QT interval prolongation
Aripiprazole (Abilify)	15–30 mg		Low	Low	Low	Low	
Olanzapine (Zyprexa)	10–20 mg	12	Med	Low	Med	Low	
Risperidone (Risperdal)	1–6 mg	1	Low	Med	Low	Low	

[1] Recommended initial starting dosages are lower than the therapeutic dosage. Dosages are generally lower for geriatric patients and for those who are taking interacting medications or have other medical problems. Adapted from Meltzer HY and Fatemi SH, "Treatment of Schizophrenia" in Ed. Schatzberg AF and Nemeroff CB, in Textbook of psychopharmacology, 2nd ed. American Psychiatric Press, 1998.

[2] Potency is indicated as relative milligram dose equivalents (i.e., 100 mg of thioridazine is equivalent to 2 mg of haloperidol) and should not be confused with efficacy. All typical antipsychotics are thought to be equally efficacious. Efficacy for clozapine and risperidone is at least equal to and may be superior to that of typical antipsychotics.

* Due to fatal cardiac events, now approved only for refractory schizophrenia.

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TABLE 11-2

Indications for Antipsychotic Drugs

- Short-term use (<3 Mos.)
  - Effective
    - Exacerbations of schizophrenia
    - Acute mania
    - Depression with psychotics features (combined with antidepressant)
    - Other acute psychoses (e.g schizophreniform psychoses)
    - Acute deliria and organic psychoses
    - Drug-induced psychoses due to hallucinogens and psychostimulants (not phencyclidine)
    - Non-psychiatric uses: nausea and vomiting; movement disorders
  - Possibly effective
    - Brief use for episodes of severe dyscontrol or apparent psychosis in personality disorders
- Long-term use
  - Effective
    - Schizophrenia
    - Tourette's syndrome
    - Treatment-resistant bipolar disorder
    - Huntington's disease and other movement disorders
    - Chronic organic psychoses
  - Possibly effective
    - Paranoid disorders
    - Childhood psychoses

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chotics. Dopamine-containing axons arising from brain stem nuclei (the ventral tegmental area and substantia nigra) project to the basal ganglia, frontal cortex, and limbic areas. Typical antipsychotics block D_2 receptors (as does risperidone). Blockade of dopamine in the cortical and limbic areas results in reduction in psychotic symptoms, whereas blockade of dopamine in the basal ganglia produces extrapyramidal side effects. Although antipsychotics, especially lower potency medications, may have an initial sedative effect, their antipsychotic action is not immediate and takes several days to several weeks to peak.

Atypical Antipsychotics (Serotonin/Dopamine Antagonists)

At present, in the United States five atypical antipsychotics are marketed; others are likely to be available soon. Antipsychotics are classified as atypical when they produce fewer movement side effects than typical antipsychotics. In addition to dopamine receptor blockade, atypical antipsychotics block serotonin receptors of the $5HT_2$ subtype. Figure 11-2 depicts the similarities and differences of the serotonin and dopamine systems. Serotonin receptor blockade conveys some protection against extrapyramidal side effects and may impart antipsychotic efficacy. Risperidone is similar to typical neuroleptics in that it is a very potent blocker of the D_2 receptor. Clozapine, conversely, is a potent blocker of the D_4 receptor and has much less D_2 affinity. The D_4 receptor blockade may account for clozapine's broader therapeutic qualities.

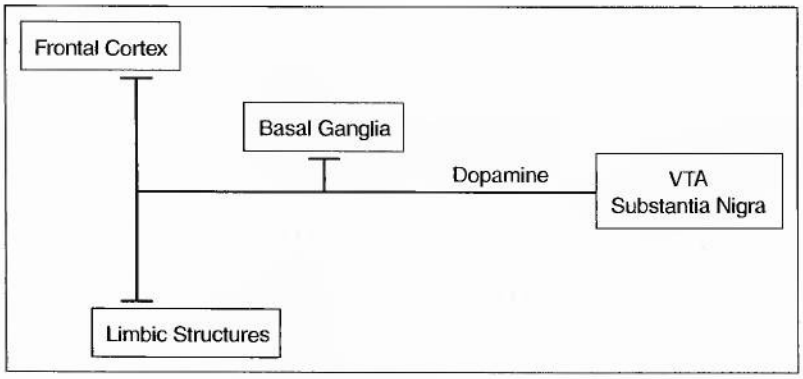

Figure 11-1 • Pathways affected by typical antipyschotics.

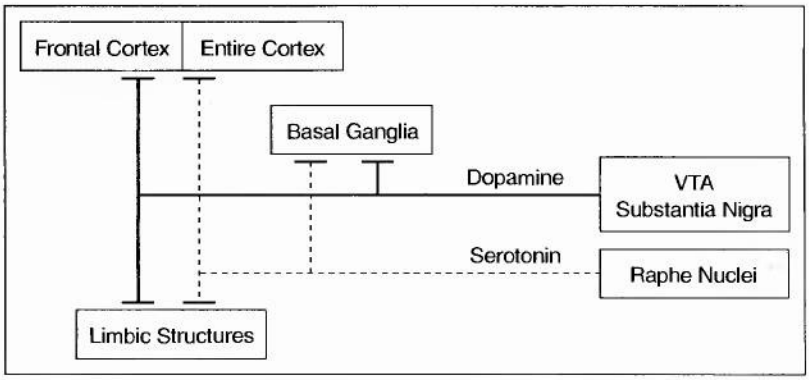

Figure 11-2 • Pathways affected by atypical antipsychotics.

Choice of Medication

Because all antipsychotics are considered efficacious, choice of medication should be based on prior patient or family member response, side-effect profile (patient tolerance), and available form (i.e., elixir or IM, or IM depot availability). At present, **clozapine** is the only medication clearly shown to be superior in patients for whom typical antipsychotics have failed. Fluphenazine and haloperidol are available in depot preparations, which are given intramuscularly every 2 to 4 weeks.

Therapeutic Monitoring

Patients on antipsychotics should be monitored closely for adverse drug reactions. Particularly important are neurologic side effects such as akathisia (restlessness), neuroleptic malignant syndrome (NMS), and extrapyramidal symptoms (EPS). Patients taking antipsychotics that lower seizure threshold should be carefully monitored for seizure activity. Individuals taking clozapine must have weekly white blood cell counts to monitor for the development of agranulocytosis. Clozapine must be discontinued immediately in patients demonstrating this potentially fatal reaction.

Blood levels have generally been of little use in monitoring antipsychotic efficacy, in part because some medications have many active metabolites. Haloperidol levels have some utility in patients who have side effects at low doses or who fail to respond to high doses. Clozapine levels are also frequently used. Noncompliance is often the cause of apparent therapeutic failure.

The duration of therapy depends on the nature and severity of the patient's illness. Many disorders, such as schizophrenia, require maintenance antipsychotic therapy. Because of the serious sequela associated with long-term antipsychotic use, maintenance therapy should be used only after a careful risk-to-benefit analysis with the patient and involved family.

Side Effects and Adverse Drug Reactions

Side effects of antipsychotics are a major consideration in physician prescribing. Patients who cannot bear the side effects of medications are noncompliant and suffer greater rates of relapse and recurrence. Certain side effects such as sedation can be useful to a patient with insomnia or severe agitation but can also limit functioning. A comparison of side-effect profiles for commonly used antipsychotics is provided in Table 11-1. Common side effects are described below. Further discussion of neurologic side effects is found in Chapter 16.

Anticholinergic Side Effects

Low-potency antipsychotics have the greatest anticholinergic side effects such as dry mouth, constipation, urinary retention, and blurred vision. The anticholinergic properties, however, counter the EPS. In some cases, anticholinergic delirium may occur, especially in the elderly, those with organic brain syndromes, or patients on other anticholinergic agents.

Reduced Seizure Threshold

Low-potency typical antipsychotics and clozapine are associated with lowering seizure threshold. Seizures resulting from antipsychotic therapy are treated by changing medications, lowering the dose, or adding an antiseizure medication.

Hypotension

Orthostatic hypotension is particularly common with low-potency agents and risperidone. The hypotensive effect of antipsychotics is generally due to alpha-receptor blockade.

Agranulocytosis

Agranulocytosis has been associated most commonly with clozapine. Because of the potentially fatal nature of this adverse effect, clozapine distribution is regulated and requires a weekly complete blood count with differential to monitor for neutropenia.

Cardiac Side Effects

Ziprasidone, low-potency antipsychotics (particularly thioridazine), and risperidone may cause QT prolongation (with risk of torsade de pointes). Nonspecific electrocardiographic changes may also occur with certain antipsychotic medications (particularly with clozapine and olanzapine).

Metabolic Effects

Although patients with psychotic disorders are known to have higher rates of obesity and diabetes mellitus independent of medication therapy, studies have indicated that certain atypical antipsychotic medications (particualarly olanzapine and clozapine) are associated with high rates of weight gain, dyslipidemia, and may be associated with adult-onset diabetes. All patients taking these medications should have regular metabolic indices checked including lipid levels, fasting blood sugar, and body mass index.

Movement Disorders

Movement disorders such as dystonia, EPS, akathisia, NMS, and tardive dyskinesia may occur and are discussed further in Chapter 16.

Other Side Effects

Skin and ocular pigmentation are common side effects of neuroleptics, as is increased photosensitivity. Thioridazine can cause pigmentary retinopathy at high doses. Increased prolactin levels (and sequela) may also occur. Quetiapine may increase the risk of developing cataracts.

KEY POINTS

1. Antipsychotics are used to treat the psychotic symptoms of a wide range of disorders.
2. These drugs are equally effective but differ in potency (with the exception of clozapine and possibly risperidone).
3. Antipsychotics can have serious side effects.

Chapter

12 Antidepressants, ECT, and Phototherapy

Antidepressants are used commonly in medical and psychiatric practice. As a class, antidepressants have in common their ability to treat major depressive illness. Most antidepressants are also effective in the treatment of panic disorder and other anxiety disorders. Some antidepressants effectively treat obsessive-compulsive disorder (OCD) and a variety of other conditions (see indications below).

The most commonly prescribed antidepressants are listed in Table 12-1. Antidepressants are subdivided into groups based on structure or prominent functional activity: selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants (TCAs), monoamine oxidase inhibitors (MAOIs), and other antidepressant compounds with a variety of mechanisms of action. Antidepressants are typically thought to act on either the serotonin or norepinephrine systems, or both. Choice of medications typically depends on diagnosis, history of response (in patient or relative), and the side-effect profile of the medication. Antidepressant effects are typically not seen until 2 to 4 weeks into treatment. Side effects must be carefully monitored, especially for TCAs and MAOIs.

INDICATIONS

Table 12-2 lists the indications for antidepressants. The main indication for antidepressant medications is major depressive disorder as defined by the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV). Antidepressants are used in the treatment of all subtypes of depression, including depressed phase of bipolar disorder, psychotic depression (in combination with an antipsychotic medication), atypical depression, and seasonal depression (see Chapter 2). Antidepressants also are indicated for the prevention of recurrent depressive episodes.

Antidepressant medications may be effective in the treatment of patients with dysthymic disorder, especially when there are clear neurovegetative signs or a history of response to antidepressants.

Panic disorder with or without agoraphobia has been shown to respond to SSRIs, MAOIs, TCAs, and high-potency benzodiazepines (alprazolam and clonazepam).

OCD has been shown to respond to the serotonin-selective tricyclic clomipramine (Anafranil) and to SSRIs at high doses (e.g., fluoxetine at 60–80 mg/day). Obsessions tend to be more responsive to pharmacotherapy than compulsions. Symptoms of OCD respond more slowly than symptoms of major depression. Trials of 12 weeks or more are needed before a medication can be ruled a failure for an OCD patient.

The binging and purging behavior of bulimia has been shown to respond to SSRIs, TCAs, and MAOIs in several open and controlled trials. Because SSRIs have the most benign side-effect profile of these medications, they are often the first-line psychopharmacologic treatment.

MECHANISMS OF ACTION

Antidepressants are thought to exert their effects at particular subsets of neuronal synapses throughout the brain. Their major interaction is with the

TABLE 12-1

Commonly Prescribed Antidepressants

Drug (Brand Name)	Suggested Starting Dosage*	Suggested Maximum Dosage*
SSRIs		
Fluoxetine (Prozac)	5–20 mg/day	80 mg/day
Sertraline (Zoloft)	25–50 mg/day	200 mg/day
Paroxetine (Paxil)	10–20 mg/day	50 mg/day
Fluvoxamine (Luvox)	25–50 mg qhs	300 mg qhs
Citalopram (Celexa)	20 mg/day	60 mg/day
Escitalopram (Lexapro)	10 mg/day	40 mg/day
TCAs		
Nortriptyline (Pamelor)	10–25 mg/day	150 mg/day
Imipramine (Tofranil)	10–50 mg/day	300 mg/day
Desipramine (Norpramin)	10–25 mg/day	300 mg/day
Clomipramine (Anafranil)	25–50 mg/day	250 mg/day
MAOIs		
Tranylcypromine (Parnate)	10–20 mg/day	60 mg/day
Phenelzine (Nardil)	15–30 mg/day	90 mg/day
Other antidepressants		
Bupropion (Wellbutrin)	75–100 mg/day	150 mg tid
Bupropion SR (Wellbutrin SR, Zyban)	150 mg/day	200 mg bid
Nefazodone (Serzone)	50–100 mg bid	600 mg/day
Venlafaxine (Effexor)	25 mg tid	125 mg tid
Mirtazapine (Remeron)	15 mg qhs	45 mg qhs
Trazodone (Desyrel)	50 mg qhs	400 mg qhs

* Geriatric patients generally require lower doses.
Modified from Stoll A, Psychopharmacology reference card I.1996.

TABLE 12-2

Common Indications for Antidepressant Usage

Effective
- Major depression (unipolar)
- Bipolar depression
- Prophylaxis against recurrence of major depression (unipolar)
- Panic disorder
- Social phobia
- Depression with psychotic features in combination with antipsychotic
- Bulimia
- Neuropathic pain (tricyclic drugs)
- Enuresis (imipramine best studied)
- Obsessive-compulsive disorder (clomipramine and SSRIs)
- Atypical depression (SSRIs or monoamine oxidase inhibitors)

Probably effective
- Attention-deficit/hyperactivity disorder
- Cataplexy due to narcolepsy
- Dysthymia (chronic depression)
- Generalized anxiety disorder
- Organic mood disorders
- Posttraumatic stress disorder
- Pseudobulbar affect (pathological laughing or crying)

Possibly effective
- School phobia and separation anxiety
- Personality disorders

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monoamine neurotransmitter systems (dopamine, norepinephrine, and serotonin). Dopamine, norepinephrine, and serotonin are released throughout the brain by neurons that originate in the ventral brainstem, locus ceruleus and the raphe nuclei, respectively. These neurotransmitters interact with numerous receptor subtypes in the brain that are associated with the regulation of global state functions including appetite, mood states, arousal, vigilance, attention, and sensory processing.

SSRIs act by binding to presynaptic serotonin reuptake proteins, thereby inhibiting reuptake and increasing the levels of serotonin in the synaptic cleft. TCAs act by blocking presynaptic reuptake of both serotonin and norepinephrine. MAOIs act by inhibiting the presynaptic enzyme (monoamine oxidase) that catabolizes norepinephrine, dopamine, and serotonin, thereby increasing the levels of these neurotransmitters presynaptically.

These immediate mechanisms of action are not sufficient to explain the delayed antidepressant effects (typically 2 to 4 weeks). Other unknown mechanisms must play a role in the successful psychopharmacologic treatment of depression.

CHOICE OF MEDICATION

Many textbooks and articles assert that all antidepressants have roughly the same efficacy in treating depression. More recent data looking at rates of remission of depression indicate that venlafaxine and tricyclic antidepressants may be more effective than the SSRIs at achieving depression remission. The higher rates of remission are believed to be related to the combined actions of venlafaxine and TCAs on serotonin and noradrenergic systems. These effects may be achieved also by combinations of antidepressants such as bupropion and SSRIs. This complicates medication choice, as before medication choice was based mainly on symptom profile and diagnosis, prior patient response, side-effect profile, and patient tolerance. Another factor to consider when treating depression is remission rates with a particular drug. More research must be done in this area to determine the relative rates of depression remission, particularly in subtypes of patients with depression. SSRIs, bupropion, venlafaxine, mirtazapine, and nefazadone are the most well tolerated antidepressants and are generally thought of as first-line agents for major depression. Compared with TCAs and MAOIs, these medications have very low sedative, anticholinergic, and orthostatic hypotensive effects. These agents should be considered for use especially in patients with cardiac conduction disease, constipation, glaucoma, or prostatic hypertrophy.

Among the tricyclic antidepressants, nortriptyline and desipramine have the least sedative, anticholinergic, and orthostatic hypotensive effects. They can be used as first-line agents in younger, healthier people, especially if cost is a consideration (tricyclics are much less expensive than SSRIs, bupropion, or venlafaxine).

Because of the necessary diet restrictions and the risk of postural hypotension, the MAOIs (phenelzine and tranylcypromine) should be used most selectively. They can be quite effective, however, and are used in patients for whom SSRIs and tricyclics have failed, in patients with a concomitant seizure disorder (MAOIs and SSRIs do not lower the seizure threshold), or in those with atypical depressions or social phobia (MAOIs or SSRIs are most effective). High-dose SSRIs and clomipramine (despite its high sedative, anticholinergic, and orthostatic hypotensive effects) are the treatments of choice for OCD.

THERAPEUTIC MONITORING

Approximately 50% of patients who meet DSM-IV criteria for major depression will recover with a single adequate trial (at least 6 weeks at a therapeutic dosage) of an antidepressant. The most common reasons for failed trials are inadequate dose and inadequate trial length. However, dosage and length of trial are often limited by side effects (or noncompliance).

Patients on antidepressants should be monitored carefully for side effects or adverse drug reactions (listed below). Generally, antidepressant therapy of a first episode of unipolar depression should continue for 6 months. Patients with recurrent or chronic depression require longer or perhaps lifelong maintenance treatment. Increasing the dose, augmentation with lithium or T3 (Cytomel), or a psychostimulant (e.g., methylphenidate), switching antidepressants, or addition of a second antidepressant is helpful in treating refractory depression. Patients on most TCAs require serum level measurements to determine appropriate dosing.

SIDE EFFECTS AND ADVERSE DRUG REACTIONS

SSRIs

Although specific SSRIs have slightly different side-effect profiles, as a group their main side effects are nausea, headache, neuromuscular restlessness (resembling akathisia), insomnia or sedation, and delayed ejaculation/anorgasmia. SSRIs combined with MAOIs are dangerous: a fatal serotonin syndrome may result.

TCAs

TCAs in many patients are quite well tolerated but overall are less tolerated by patients for their side effects than the SSRIs, bupropion, or venlafaxine. The major side effects associated with TCAs are orthostatic hypotension, anticholinergic effects, cardiac toxicity, and sexual dysfunction. Specific TCAs have relative degrees of each of these effects.

Orthostatic hypotension is the most common serious side effect of the TCAs. This is particularly worrisome in elderly patients, who may be more prone to falls. Anticholinergic toxicity can be mild, including dry mouth, constipation, blurred near vision, and urinary hesitancy, or more severe, with agitation, motor restlessness, hallucinations, delirium, and seizures.

Cardiac toxicity may limit the use of TCAs in some patients. TCAs have quinidine-like effects on the heart, potentially causing sinus tachycardia; supraventricular tachyarrhythmias; ventricular tachycardia; ventricular fibrillation; prolongation of PR, QRS, and QT intervals; bundle branch block; first-, second-, and third-degree heart block; or ST and T-wave changes. Major complications from TCAs are rare in patients with normal hearts. TCAs should be avoided in patients with conduction system disease.

Sexual dysfunction includes impotence in men and decreased sexual arousal in women.

MAOIs

Patients who take MAOIs are at risk for hyperadrenergic crises from the ingestion of sympathomimetic amines (such as tyramine) that fail to be detoxified because of inhibition of the gastrointestinal monoamine oxidase system. Improper diet can lead to severe hypertensive crises (tyramine crisis) with potential myocardial infarction or stroke. Foods that must be avoided include cured meats or fish, beer, red wine, all cheese except cottage and cream cheeses, and overripe fruits. Many over-the-counter cold and pain remedies must also be avoided. Treatment of hypertensive crisis, if severe, may require emergency medical attention, including IV phentolamine (an alpha blocker) or continuous IV nitroprusside infusion.

MAOIs cause a dose-related orthostatic hypotension: tranylcypromine can cause insomnia and agitation; phenelzine can cause daytime somnolence.

Other Antidepressants

Venlafaxine (Effexor, Effexor XR) is a serotonin and noradrenergic reuptake inhibitor with a better side effect profile than TCAs or MAOIs. A May 2002 meta-analysis of prior antidepressant trials suggested that venlafaxine and TCAs may have a greater remission rate than SSRIs. Further study is needed including more head-to-head comparison trials. Nefazodone (Serzone) and trazodone (Desyrel) are serotonin-modulating antidepressants. Trazodone is prescribed rarely as a sole antidepressant but is often prescribed as an adjunct to an SSRI for sleep because it has strong sedative properties (at higher doses it serves as an antidepressant). In addition to sedation, trazodone can on rare occasions induce priapism (prolonged, painful penile erection) that can cause permanent damage. Patients must be instructed to seek emergency treatment should such an erection occur. Nefazodone is similar to trazodone but is less sedating at therapeutic doses. It appears to have a low rate of sexual dysfunction.

Bupropion (Wellbutrin, Wellbutrin SR, Zyban) appears to work by inhibiting the uptake of dopamine and norepinephrine. Bupropion has a low incidence of sexual side effects. In addition to its efficacy in treating major depression, bupropion has been shown to be effective in smoking cessation (marketed as Zyban) and attention deficit disorder. Bupropion has a higher than average risk of seizures compared with other antidepressants. The risk of seizures is greatest above a daily dose of 450 mg or after a single dose of greater than 150 mg of immediate-release bupropion.

Mirtazapine (Remeron) is classified as a modulator of norepinephrine and serotonin. It is quite sedating in some individuals, and has a low incidence of sexual dysfunction.

Phototherapy

Phototherapy consists of the controlled administration of bright light to treat specific psychiatric illnesses. Phototherapy is administered using specially designed bright light boxes and has been shown effective in the treatment of the seasonal subtype of major depression (also known as seasonal affective disorder). Phototherapy is also used in the treatment of the delayed sleep phase syndrome and jet lag. In the treatment of seasonal affective disorder, early morning bright light therapy is superior to evening light in most individuals. Light intensity of 2,500 to 10,000 lux is most effective. Light therapy can induce mania in susceptible individuals.

Electroconvulsive Therapy (ECT)

ECT (formerly known as electric shock therapy) is one of the oldest and most effective treatments for major depression. ECT also has some efficacy in refractory mania and in psychoses with prominent mood components or catatonia. ECT appears to work via the induction of generalized seizure activity in the brain. The peripheral manifestations of seizure activity are blocked by the use of paralytics, and memory for the event is blocked by the use of anesthetics and by seizure activity. Modern ECT produces short-term memory loss and confusion. Bilateral ECT is more effective than unilateral ECT but produces more cognitive side effects.

KEY POINTS

1. Antidepressants have multiple indications including various forms of depression, anxiety disorders, bulimia, and OCD, among others.
2. Antidepressants act on serotonergic and noradrenergic receptor systems.
3. Some antidepressants have been shown to be efficacious for particular disorders; for major depression, all approved antidepressants reduce symptoms to a significant degree, TCAs and venlafaxine may more frequently induce remission. Antidepressants for depression are often chosen based on side-effect profile and symptom constellation.
4. Some antidepressants, particularly TCAs, require monitoring of serum levels.
5. The effects of antidepressants can be augmented by the addition of lithium, thyroid hormone, or psychostimulants.
6. Antidepressants have side effects that vary according to class.

Chapter

13 Mood Stabilizers

The mood stabilizers most commonly used are lithium, valproate, and carbamazepine. Table 13-3 lists mood stabilizers and their dosage and therapeutic levels. Other medications, such as calcium channel blockers, benzodiazepines, and antipsychotics, have some utility in refractory bipolar disorder.

INDICATIONS

Mood stabilizers are indicated acutely (in conjunction with antipsychotics) for the treatment of mania. They are indicated for long-term maintenance prophylaxis against depression and mania in bipolar individuals. Anticonvulsant medications (valproate and carbamazepine) may also be useful in individuals having seizure-related mood instability. Mood stabilizers are also used for treatment of impulsive behavior in individuals without bipolar disorders.

The choice of mood stabilizer is based on a patient's particular psychiatric illness (i.e., subtype of bipolar disorder) and other clinical factors such as side effects, metabolic routes, patient tolerance, and a history of patient or first-degree relative drug responsiveness. Table 13-1 lists the major mood stabilizers and their most common indications. Some common and more serious side effects of mood stabilizers are listed in Table 13-2.

The mechanism of action of mood stabilizers in bipolar illness is unclear. The range of neurotransmitters affected by these medications and their disparate modes of action suggest that mania may be controlled by altering the function of several different neurotransmitter systems. Conversely, they may share a common mechanism of action that is yet to be elucidated.

LITHIUM

Mechanism of Action

The mechanism of action of lithium in the treatment of mania is not well determined. Lithium alters at least two intracellular second messenger systems (the adenyl cyclase, cyclic AMP system, and the G protein-coupled phosphoinositide systems) and as an ion, can directly alter ion channel function. Because norepinephrine and serotonin in the central nervous system (CNS) use G protein-coupled receptors as one of their mechanisms of action, their function is altered by lithium. Lithium also alters GABA (gamma-amino-butyric acid) metabolism.

Choice of Medication

Lithium is indicated as a first-line treatment for regular cycling bipolar disorder in individuals with normal renal function. Lithium also is used to augment other antidepressants in unipolar depression. Lithium is renally cleared and can easily reach toxic levels in persons with altered renal function (e.g., especially the elderly). It is less effective in the treatment of the rapid cycling variant of bipolar disorder.

Therapeutic Monitoring

Lithium levels should be monitored regularly until a stable dosing regimen has been obtained. Additional monitoring is necessary in a patient with variable compliance or altered renal function. In addition, patients should be warned about toxicity and be regularly assessed for side effects. Thyroid-stimulating hormone and creatinine should also be monitored at

TABLE 13-1

Psychiatric Indications for Mood Stabilizers

Drug	Indications
Lithium	Acute mania Long-term prophylaxis in bipolar disorder Augmentation of antidepressant medications Impulse dyscontrol
Valproate	Acute mania Rapid-cycling bipolar disorder Mixed features bipolar disorder Impulse dyscontrol
Carbamazepine	Acute mania Rapid-cycling bipolar disorder Mixed features bipolar disorder Impulse dyscontrol

regular intervals to check thyroid and kidney function, respectively.

Side Effects

Lithium has several minor but troublesome side effects, including tremor, polyuria, gastrointestinal distress, minor memory problems, acne exacerbation, and weight gain. Approximately 5% of patients on long-term lithium therapy develop hypothyroidism because the lithium interferes with thyroid hormone production. At toxic levels, ataxia, coarse tremor, confusion, coma, sinus arrest, and death can occur. Lithium has a narrow therapeutic window, and patients can become toxic at prescribed doses, especially if they undergo an abrupt change in renal function.

VALPROATE

Mechanism of Action

The mechanism of action of valproate is thought to be due in part to augmentation of GABA function in the CNS. Valproate increases GABA synthesis, decreases GABA breakdown, and enhances its postsynaptic efficacy.

Choice of Medication

Valproate is indicated in acute mania and in prophylaxis against mania in bipolar disorder (Table 13-1). It is more effective than lithium for the rapid-cycling and mixed variants of bipolar disorder. It may not provide prophylaxis against depression in bipolar disorder or augment antidepressants. It is used in treating impulse dyscontrol.

Therapeutic Monitoring

Valproate levels should be monitored regularly until a stable blood level and dosing regimen have been obtained. Liver function tests should be checked at baseline and frequently during the first 6 months, especially because the idiosyncratic reaction of fatal hepatotoxicity is most frequent in this time frame.

Side Effects

At therapeutic levels, valproate produces a variety of side effects, including sedation, mild tremor, mild ataxia, and gastrointestinal distress. Thrombocytopenia and impaired platelet function may also occur. At toxic levels, confusion, coma, cardiac arrest, and death can occur. Valproate usage carries with it the risk of idiosyncratic but serious side effects. These include fatal hepatotoxicity, fulminant pancreatitis, and agranulocytosis.

CARBAMAZEPINE

Mechanism of Action

The mechanism of action of carbamazepine in bipolar illness is unknown. Carbamazepine blocks sodium channels in neurons that have just produced an action potential, blocking the neuron from repetitive firing. In addition, carbamazepine decreases the amount of transmitter release at presynaptic terminals. Carbamazepine also appears to indirectly alter central GABA receptors.

Choice of Medication

Carbamazepine is generally considered to be a second-line drug (after lithium and valproate) for the treatment of mania (Table 13-1). It is used in acute mania, prophylaxis against mania in bipolar disorder, and may be more effective than lithium in rapid-cycling and mixed mania. Carbamazepine's efficacy in the prophylaxis and treatment of depression is not clear. It is also used in treating impulse dyscontrol.

TABLE 13-2

Drugs Used as Mood Stabilizers

Drug	Side-Effect Profile	
Lithium	Therapeutic levels	
	CNS:	sedation, cognitive clouding, fine tremor
	Endocrine:	abnormal TSH, clinical hypothyroidism
	Cardiac:	T-wave change, sinus arrhythmia
	Renal:	polyuria
	Dermatologic:	acne, psoriasis
	GI:	nausea, vomiting, diarrhea
	Hematologic:	benign leukocytosis
	Other:	weight gain, fluid retention
	Toxic levels	
	CNS:	ataxia, coarse tremor, confusion, seizure, coma, death
	Cardiac:	sinus arrest
Valproate	Therapeutic levels	
	CNS:	somnolence, ataxia, tremor
	Endocrine:	menstrual irregularities, thyroid abnormalities
	Dermatologic:	alopecia, rash
	Hepatic:	mild transaminitis
	GI:	nausea, vomiting, indigestion
	Hematologic:	thrombocytopenia, platelet dysfunction
	Other:	edema
	Toxic levels	
	CNS:	ataxia, confusion, coma, death
	Cardiac:	cardiac arrest
	Idiosyncratic	
	Hepatic:	fatal hepatotoxicity
	GI:	pancreatitis
	Hematologic:	agranulocytosis
Carbamazepine	Therapeutic levels	
	CNS:	ataxia, sedation, dizziness, diplopia
	Dermatologic:	rash
	Cardiac:	decreased atrioventricular conduction
	Hematologic:	benign leukopenia
	GI:	nausea
	Toxic levels	
	CNS:	somnolence, autonomic instability, coma
	Cardiac:	atrioventricular block
	Respiratory:	respiratory depression
	Idiosyncratic	
	Hematologic:	agranulocytosis, pancytopenia, aplastic anemia

Side Effects

Carbamazepine, at therapeutic levels, produces similar CNS side effects to lithium and valproate. Nausea, rash, and mild leukopenia are also common. At toxic levels, autonomic instability, atrioventricular block, respiratory depression, and coma can occur. Carbamazepine has idiosyncratic side effects of agranulocytosis, pancytopenia, and aplastic anemia.

Therapeutic Monitoring

Carbamazepine levels should be monitored regularly until a stable dosing regimen has been obtained. Patients should be carefully monitored for rash, signs of toxicity, or evidence of severe bone marrow suppression.

Novel Mood Stabilizers

Several new agents are being used as mood stabilizers for bipolar disorder. Like valproic acid, they are all FDA-approved anticonvulsants that are now being studied for use in treatment of acute manic episodes and for maintenance of bipolar disorder (see Table 13-3).

LAMOTRIGINE

Mechanism of Action

The mechanism of action of lamotrigine in bipolar disorder is unknown. Lamotrigine is approved by the FDA for use as an anticonvulsant and is used off-label in mood disorders. Lamotrigine in vitro has been shown to inhibit voltage-sensitive sodium channels. This effect is believed to stabilize neuronal membranes and modulate presynaptic excitatory neurotransmitter release.

Choice of Medication

Although studies are still ongoing regarding the use of lamotrigine in bipolar disorder, it appears to have some antidepressant as well as mood stabilizing properties. Since its efficacy is not yet certain, it is currently only used after a patient has failed or been allergic to more traditional therapies.

Dosages are generally altered for the elderly, those with renal or other organ impairment, or when combined with interacting agents.

Therapeutic Monitoring

The development of serious allergic reactions to lamotrigine appears to be related to rapid dose escalation and/or drug interactions. A clinically useful assay for serum levels of lamotrigine is not available. Generally, this medication should only be prescribed by a qualified psychiatrist, neurologist, or other physician who is aware of the complex drug interactions that exist particularly between valproic acid and lamotrigine.

Side Effects

Lamotrigine can commonly cause ataxia, blurred vision, diplopia, dizziness, nausea, and vomiting. Severe, potentially life-threatening allergic rashes have been reported with the use of lamotrigine. The allergic reaction can begin as a simple rash and lead to Stevens-Johnson syndrome.

GABAPENTIN

Mechanism of Action

The mechanism of action of gabapentin in bipolar disorder and in seizure disorders is unknown. Gabapentin inhibits induced seizures in rats and mice. Although it is structurally related to the neurotransmitter GABA, it has no binding affinity to the GABA receptor or to any other known brain receptors.

TABLE 13-3

Common Mood Stabilizers[1]

Drugs	Starting Dosage	Therapeutic Dosage	Therapeutic Serum Concentration
Lithium carbonate	300 mg bid-tid	900–1500 mg/day	0.6–1.1 mEq/L
Valproic acid	250 mg bid-tid	1000–2500 mg/day	50–120 mg/L
Carbamazepine	100 mg bid-tid	600–1200 mg/day	4–12 mg/L
Lamotrigine	25–50 mg qhs[2]	300–500 mg/day	—
Gabapentin	300 mg qd	900–1800 mg/day	—
Oxcarbazepine	150 mg bid	600–1200 mg/day	—

[1] Dosages are generally lower for geriatric patients, patients taking interacting medications, or patients with other medical problems.
[2] Valproate will increase lamotrigine serum level, necessitating lower dosages.
Adapted from Stoll AL, Psychopharmacology reference card, 1996 and from Physician's desk reference, 56th ed. 2002.

Choice of Medication

Studies indicate that gabapentin may be a useful adjunctive medication for mood stabilization and anxiolysis in bipolar disorder, but it appears to lack sufficient efficacy for use as monotherapy for bipolar disorder episode prophylaxis. It is generally used as an adjunct to more traditional agents such as lithium or valproic acid or to address particular target symptoms such as anxiety.

Therapeutic Monitoring

A similar dosage range is used for seizure and bipolar disorders, although the drug is not FDA approved for this use. The drug is excreted renally and unchanged. There is no clinically available assay for serum gabapentin.

KEY POINTS

1. Mood stabilizers are indicated for the treatment of bipolar disorder.
2. They work by unknown but likely varied mechanisms.
3. Efficacy varies according to the subtype of bipolar illness.
4. Mood stabilizers have serious toxicities, so patients require regular monitoring.

Chapter

14 Anxiolytics

The medications discussed in this chapter have anxiolysis in common. Although benzodiazepines have a wide variety of clinical applications (e.g., as preanesthetics, in the treatment of status epilepticus, as muscle relaxants, and in the treatment of insomnia) and other medications (e.g., antidepressants) are of utility in treating some forms of anxiety, the benzodiazepines are uniquely effective for the rapid relief of a broad spectrum of anxiety symptoms. Buspirone is a novel medication that, at present, is used primarily in the treatment of generalized anxiety disorder; it does not appear to be effective in treating other types of anxiety (e.g., panic).

INDICATIONS

Benzodiazepines

Benzodiazepines are among the most widely used drugs in all of medicine. In psychiatry they are used as the primary treatment of a disorder or as adjunct treatment to other pharmacologic agents. Benzodiazepines are used to treat a variety of anxiety disorders: panic disorder, generalized anxiety disorder (GAD), anxiety associated with stressful life events (as in adjustment disorders with anxiety), and anxiety that complicates depression (see Chapter 3). In addition, benzodiazepines are used for the short-term treatment of insomnia, for the treatment of alcohol withdrawal, for the agitation of mania, dementia, and psychotic disorders, and in the treatment of catatonia (Table 14-1).

Buspirone (Buspar)

Buspirone is used primarily for generalized anxiety disorder.

MECHANISM OF ACTION

Benzodiazepines

Benzodiazepines appear to function as anxiolytics via their agonist action at the central nervous system (CNS) $GABA_A$ (gamma-amino-butyric-acid) receptors (the $GABA_A$ receptor complex regulates a chloride ion channel, $GABA_B$ receptors appear to work by second messenger systems). GABA is a widespread inhibitory neurotransmitter with a complicated receptor structure, having multiple binding sites for GABA, benzodiazepines, and barbiturates. The most likely mode of action of benzodiazepines in treating psychiatric illnesses is via their augmentation of GABA function in the limbic system. Because benzodiazepines are direct agonists at a rapidly responding ion channel, their mechanism of action is virtually instantaneous with their arrival in the CNS (in contrast to buspirone, see below).

Buspirone

Buspirone is a novel medication that appears to act as an anxiolytic via its action as an agonist at the serotonergic $5HT_{1\alpha}$ receptor. In addition, it has some D_2 antagonist effects, although with unclear clinical significance. Unlike the benzodiazepines, it does not work rapidly; a period of several weeks of sustained dosing is required to obtain symptomatic relief. Buspirone has no GABA receptor affinity and is therefore not useful in treating benzodiazepine or alcohol withdrawal. It is not a sedative and is not useful in treating insomnia.

TABLE 14-1

Psychiatric Uses for Benzodiazepines

- Anxiety disorders
  - Generalized anxiety disorder
  - Panic disorder
- Mood disorders
  - Temporary treatment of anxiety associated with depression
  - Temporary treatment of insomnia associated with depression
  - Treatment of agitation in acute mania
  - Possible mood-stabilizing effect in bipolar disorder
- Adjustment disorders
  - Treatment of adjustment disorder with anxiety
- Sleep disorders
  - Short-term treatment of insomnia
- Miscellaneous
  - Treatment of akathisia induced by neuroleptics
  - Agitation from psychosis or other causes
  - Catatonia (especially lorazepam)
  - Alcohol withdrawal

CHOICE OF MEDICATION

Benzodiazepines

The selection of a benzodiazepine should be based on an understanding of potency, rate of onset, route of metabolism, effective half-life, and clinically proven effectiveness. Although all benzodiazepines appear to function by common mechanisms, the particular combination of the above factors (and perhaps as yet unknown variations in affinity for receptor subtypes) produce varied clinical indications for different benzodiazepines. Table 14-2 illustrates the properties of some commonly used benzodiazepines and their common clinical uses.

Potency

The high-potency benzodiazepines alprazolam and clonazepam are used in the treatment of panic disorder.

Rate of Onset

Fast-onset benzodiazepines, such as diazepam, may produce a "high" feeling and are potentially more addictive. The fast-onset benzodiazepines flurazepam and triazolam are commonly used for insomnia, as is diazepam.

Route of Metabolism

All benzodiazepines listed, with the exception of lorazepam, oxazepam, and temazepam, require oxidation as a step in their metabolism. Because the oxidative functions of the liver are impaired with liver disease (e.g., cirrhosis) or with a general decline in liver function (e.g., aging), benzodiazepines that require oxidation are more likely to accumulate to toxic levels in individuals with impaired liver function.

Elimination Half-Life

The elimination half-life depicts the effective duration of action of the metabolized medications. For medications with long elimination half-lives, toxicity can easily occur with repetitive dosing. In addition, toxicology screens may remain positive for several days after the last dose of a long-acting benzodiazepine. Drugs with longer elimination half-lives offer less likelihood of interdose symptom rebound. For example, clonazepam is now favored over alprazolam in the treatment of panic because its longer elimination half-life provides better interdose control of panic symptoms. Medications with shorter elimination half-lives are useful for conditions such as insomnia because they are less likely to produce residual daytime sedation or grogginess.

Active Metabolites

Medications with active metabolites generally have a longer elimination half-life. Among the benzodiazepines, all but three drugs metabolized by conjugation (lorazepam, oxazepam, temazepam) and clonazepam have active metabolites.

Buspirone

Buspirone is indicated for the treatment of GAD. Because of its long lag time to therapeutic effect, patients with severe anxiety symptoms may be unable to sustain a clinical trial. Buspirone is favored as a treatment in individuals with a history of substance or benzodiazepine abuse. In general, buspirone lacks the reliability of benzodiazepines in relieving anxiety but can be effective in some people.

THERAPEUTIC MONITORING

Benzodiazepines

Benzodiazepine dosing is generally titrated to maximize symptom relief while minimizing side effects

TABLE 14-2

Frequently Used Benzodiazepines

Drug	Oral Dosage Equivalency (mg)*	Single Dosage (mg)	Usual Therapeutic Dosage (mg/day)	Onset	Metabolism	Elimination Half-Life**	Active Metab	Common Uses in Psychiatry
Alprazolam (Xanax)	2	0.25–1.0	1.0–4.0	Intermediate	Oxidation	6–20	Yes	Panic, anxiety
Chlordiazepoxide (Librium)	40–100	5.0–25.0	15.0–100.0	Intermediate	Oxidation	30–100	Yes	Alcohol detoxification
Clonazepam (Klonopin)	1	0.5–2.0	1.0–4.0	Intermediate	Oxidation	18–50	No	Panic, anxiety
Diazepam (Valium)	20	2.0–10.0	4.0–40.0	Fast	Oxidation	30–100	Yes	Anxiety, insomnia
Flurazepam (Dalmane)	120	15.0–30.0	15.0–30.0	Fast	Oxidation	50–160	Yes	Insomnia
Lorazepam (Ativan)	4	0.5–2.0	1.0–6.0	Intermediate	Conjugation	10–20	No	Anxiety, catatonia
Oxazepam (Serax)	60	10.0–30.0	30.0–120.0	Slow	Conjugation	8–12	No	Alcohol detoxification
Temazepam (Restoril)	60–120	7.5–15.0	7.5–30.0	Intermediate	Conjugation	8–20	No	Insomnia
Triazolam (Halcion)	1	0.125–0.25	0.125–0.5	Fast	Oxidation	1.5–5	Yes	Insomnia

* Single dose equivalency.
** In hours. Elimination half-life includes all active metabolites.

and the potential for abuse. No routine monitoring is required; although serum drug levels can be obtained, they are not of great clinical use. Care must be taken in prescribing benzodiazepines because of their ability to cause physiologic dependence. They cannot be discontinued abruptly due to the risk of a withdrawal syndrome that may include seizures.

Buspirone

No routine monitoring or drug levels are required when using buspirone.

SIDE EFFECTS AND ADVERSE DRUG REACTIONS

Benzodiazepines

The major side effects of benzodiazepines are related to the CNS. The primary side effect of benzodiazepines is sleepiness or a general groggy feeling. Although benzodiazepines are often used to treat agitation, they may produce disinhibition (and therefore worsen agitation) in some patients (i.e., the elderly). Benzodiazepines are minimally depressive to the respiratory system in healthy individuals but can lead to fatal carbon dioxide retention in patients with chronic obstructive pulmonary disease. In healthy individuals, death after overdose on benzodiazepines alone is rare but does occur when benzodiazepines are taken with alcohol and other CNS depressant medications.

Buspirone

Buspirone does not tend to cause sedation, nor does it produce a significant withdrawal syndrome or dependence. The major side effects are dizziness, nervousness, and nausea.

KEY POINTS

1. Anxiolytics include benzodiazepines and buspirone.
2. Benzodiazepines bind to $GABA_A$ receptors and have a comparatively rapid onset of action; buspirone binds to serotonin receptors and takes effect after weeks of daily usage.
3. Benzodiazepines have a wide variety of uses, including anxiolysis, alcohol detoxification, agitation, and insomnia.
4. Benzodiazepines produce physiologic dependence and may manifest a significant withdrawal syndrome.
5. Buspirone treats only generalized anxiety, but does not cause physiologic dependence.

Chapter

15 Miscellaneous Medications

This chapter includes medications that are commonly used in psychiatric practice but that do not fall into the conventional categories of psychotherapeutic drugs. Many medications used in general medical practice have side effects such as sedation, stimulation, or anxiolysis. These side effects are often exploited in psychiatry to target specific symptoms (e.g., insomnia, anergia). Other drugs, such as psychostimulants, have precise indications for psychiatric usage. Many more medications than are discussed here are included in the psychiatric armamentarium.

PSYCHOSTIMULANTS

Psychostimulants are used in psychiatry to treat attention deficit disorder, narcolepsy, and some forms of depression (Table 15-1). The most commonly used psychostimulants are dextroamphetamine (Dexedrine), methylphenidate (Ritalin), and pemoline (Cylert). The mechanism of action of these medications appears to occur through their alterations of central nervous system (CNS) monoamine function. Their primary mechanism of action is thought to be facilitating endogenous neurotransmitter release (rather than acting as a direct agonist). Psychostimulants have the liabilities of inducing tolerance and psychological dependence, which may lead to abuse. The side effects of these medications are due largely to their sympathomimetic actions and include tachycardia, insomnia, anxiety, hypertension, and diaphoresis. Weight loss may be an unwanted side effect in young children but a desirable one in overweight adults.

ANTICHOLINERGICS

Medications with anticholinergic activity are commonly used in psychiatry to treat or provide prophylaxis for some types of neuroleptic-induced movement disorders (Table 15-1). Anticholinergics are generally used as first-line agents in the treatment of neuroleptic-induced parkinsonism and for acute dystonia; they may also have some utility in treating akathisia but are best tried after beta blockers and lorazepam. The most commonly used anticholinergics are benztropine and trihexyphenidyl. In addition, diphenhydramine, an antihistamine that also possesses anticholinergic properties, is frequently used to treat neuroleptic-induced movement disorders and to provide nonspecific sedation. These medications are CNS muscarinic antagonists. Side effects of anticholinergics, due to peripheral anticholinergic action, include blurry vision (due to cycloplegia), constipation, and urinary retention; their principal central side effects are sedation and delirium. Anticholinergic toxicity is a major cause of delirium, especially in individuals with dementia and HIV encephalopathy.

BETA BLOCKERS

Beta blockers are used widely in general medicine. In psychiatry, they have a few specific indications (see Table 15-1). Beta blockers likely alter behavior and mood states by altering both central and peripheral catecholamine function. For example, in anxiety, they may diminish central arousal; peripherally, they may reduce tachycardia, tremor, sweating, and hyperventilation. Common side effects of beta blockers include bradycardia, hypotension, asthma exacerba-

TABLE 15-1

Psychiatric Uses of Miscellaneous Medications

Medication	Major Psychiatric Uses
Psychostimulants	Treatment of attention deficit disorder Treatment of depression in the elderly or medically ill Treatment of narcolepsy Augmentation of antidepressants in refractory depression
Anticholinergics	Treatment of neuroleptic-induced parkinsonism Treatment of neuroleptic-induced dystonia
Beta blockers	Treatment of impulsivity Treatment of performance anxiety Treatment of akathisia Treatment of lithium-induced tremor
Disulfuram	Prevention of alcohol ingestion
Clonidine	Treatment of impulsivity Treatment of Tourette's syndrome Treatment of opiate withdrawal
Tacrine	Treatment of mild to moderate memory loss in Alzheimer's disease
Thyroid hormones	Augmentation of antidepressants in refractory depression

tion, and masked hypoglycemia in diabetics. Beta blockers may also produce depression-like syndromes characterized by fatigue and depressed mood.

DISULFIRAM (ANTABUSE)

Disulfiram is used to prevent alcohol ingestion through the fear of the consequences of ingesting alcohol while taking disulfiram (Table 15-1). Disulfiram blocks the oxidation of acetaldehyde, a step in the metabolism of alcohol. The buildup of acetaldehyde produces a toxic reaction, making an individual who ingests alcohol while taking disulfiram severely ill within 5 to 10 minutes. Symptoms include flushing, headache, sweating, dry mouth, nausea, vomiting, and dizziness. In more severe reactions, chest pain, dyspnea, hypotension, and confusion occur. Fatal reactions, although rare, can occur. Disulfiram use should be restricted to carefully selected patients who are highly motivated and who fully understand the consequences of drinking alcohol while taking disulfiram. Side effects in the absence of alcohol ingestion include hepatitis, optic neuritis, and impotence.

CLONIDINE

Clonidine is a CNS alpha$_2$ adrenoreceptor agonist. The alpha$_2$ adrenoreceptor is a presynaptic autoreceptor that inhibits the release of CNS norepinephrine. Clonidine's primary use in medicine is as an antihypertensive (Table 15-1). In psychiatry, clonidine has been variously used. It is effective in decreasing autonomic symptoms associated with opiate withdrawal and in the treatment of Tourette's syndrome, and may be useful for impulsiveness and other forms of behavioral dyscontrol. Side effects include sedation, dizziness, and hypotension.

COGNITIVE ENHANCERS

Donepezil (Aricept) and tacrine (Cognex) are reversible inhibitors of the enzyme acetylcholinesterase and are used to enhance cognition in patients with mild to moderate dementia of the Alzheimer's type. Some of the cognitive deficits in Alzheimer's disease are due to loss of cholinergic neurons in the basal forebrain that project to the cerebral cortex and hippocampus, which results in a deficiency of cholinergic neurotransmission. By inhibiting the enzyme that hydrolyzes synaptic acetylcholine, these drugs are thought to raise synaptic concentrations of acetylcholine in the remaining cholinergic neurons. Initially, these drugs reduce cognitive impairment; however, this effect wanes with the progressive loss of cholinergic neurons. Common side effects include gastrointestinal upset and other cholinomimetic effects including bradycardia and increased gastric acid secretion. Tacrine can cause elevations in serum transaminases.

THYROID HORMONES

Thyroid hormones are used primarily in psychiatry to augment the effects of antidepressants (Table 15-

1). They also may be used as adjuncts in treating rapid cycling bipolar disorder. Although clinical hypothyroidism can mimic the symptoms of depression, some individuals without clinical hypothyroidism may respond to thyroid augmentation. The theoretic basis for using thyroid hormones lies in the finding of altered hypothalamic-pituitary-adrenal axis functioning in depressed individuals. Although there is debate as to their relative efficacy, both T_3 (tri-iodo thyronine) and T_4 (tetra-iodo thyronine) cross the blood brain barrier. T_4 has been shown to be of use in conjunction with lithium to improve clinical control of rapid cycling bipolar disorder. Side effects at low doses are minimal; when dosages result in over-replacement, symptoms of hyperthyroidism emerge.

KEY POINTS

1. Miscellaneous medications are widely used for treatment of symptoms and side effects.
2. They overlap with medications used in other medical practice.
3. They have side effects and efficacy specific to each medication and its target symptoms.

Chapter

16 Major Adverse Drug Reactions

This chapter describes a group of major adverse reactions associated with use of psychiatric medications. Minor adverse reactions and side effects are outlined in the chapters on the respective medications. Although the adverse drug reactions discussed below (with the exception of serotonin syndrome) are most commonly produced by antipsychotic medications, they may occur in response to other medications. The major adverse drug reactions to antipsychotics, their risk factors, onset, and treatment are outlined in Table 16-1. Although the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition, classifies dystonia, akathisia, extrapyramidal symptoms (EPS), neuroleptic malignant syndrome (NMS), and tardive dyskinesia as neuroleptic-induced movement disorders, it is clear that akathisia can occur with the use of nonneuroleptic psychiatric medications.

DYSTONIA

Dystonia is a neuroleptic-induced movement disorder characterized by muscle spasms. Dystonia commonly involves the musculature of the head and neck but may also include the extremities and trunk. Symptoms may range from a mild subjective sensation of increased muscle tension to a life-threatening syndrome of severe muscle tetany and laryngeal dystonia (laryngospasm) with airway compromise. The muscle spasms may lead to abnormal posturing of the head and neck with jaw muscle spasm. Spasm of the tongue leads to macroglossia and dysarthria; pharyngeal dystonia may produce impaired swallowing and drooling. Ocular muscle dystonia may produce oculogyric crisis.

Risk factors include use of high-potency antipsychotics; young men are at increased risk. The condition usually develops early in drug therapy (within days).

Treatment of dystonia depends on the severity of the symptoms. In the absence of laryngospasm or severe patient discomfort, intramuscular (IM) anticholinergic medication (benztropine or diphenhydramine) can be used. In more severe cases or if laryngospasm is present, IV anticholinergic medication is used. Some cases may require intubation if respiratory distress is severe. Discontinuation of the precipitating antipsychotic is sometimes necessary; in other cases, the addition of anticholinergic medications on a standing basis prevents the recurrence of dystonia.

AKATHISIA

Akathisia, a common side effect produced by antipsychotic medications, is also caused by serotonin reuptake inhibitors. Akathisia consists of a subjective sensation of inner restlessness or a strong desire to move one's body. Individuals with akathisia may appear anxious or agitated. They may pace or move about, unable to sit still. Akathisia can produce severe dysphoria and anxiety in patients and may drive them to become assaultive or to attempt suicide. It is important to accurately diagnose akathisia because if mistaken for agitation or worsening psychosis, antipsychotic dosage may be increased with resultant worsening of the akathisia.

Risk factors for akathisia include a recent increase in medication dosing or the recent onset of medication use. Most cases occur within the first month of drug therapy but can occur anytime during treatment.

TABLE 16-1

Neuroleptic-Induced Movement Disorders*

Disorder	Risk Factors	Onset	Treatment
Dystonia	High-potency antipsychotics Young men	First few days of therapy	IM/IV benztropine or diphenhydramine Severe laryngospasm may require intubation
Akathisia	Recent increase/onset of medication dosing	First month of therapy	Propanolol, lorazepam (maybe anticholinergics)
EPS	High-potency antipsychotics Elderly Prior episode of EPS	First few weeks of therapy	Anticholinergic Lower antipsychotic dosage or change to lower-potency antipsychotic
NMS	High-dose antipsychotics, rapid dose escalation, or IM injection of antipsychotics Agitation, dehydration Prior episode of NMS	Usually within first few weeks; can occur at any point in antipsychotic therapy	Discontinue antipsychotic medication Supportive symptom management Dantrolene, bromocriptine May require intensive care
Tardive dyskinesia	Elderly Long-term antipsychotic treatment Female African American Mood disorders	Usually after years of treatment	Lower dosage of antipsychotic Change antipsychotics Change to clozaril

* The DSM-IV classification for these disorders defines them as neuroleptic-induced movement disorders. The term *neuroleptic* generally refers to typical antipsychotics (see Chapter 11). Exceptions include risperidone, which is classified as an atypical antipsychotic but can cause all the above disorders; SSRIs, which are not neuroleptic drugs but can clearly produce akathisia; and clozaril, which does not appear to produce dystonia, akathisia, EPS, or tardive dyskinesia but may cause NMS.

Treatment consists of reducing the medication (if possible) or using either beta blockers (propanolol is commonly used) or benzodiazepines (especially lorazepam). Although there is some doubt as to their efficacy, anticholinergics (diphenhydramine or benztropine) are also used frequently.

EXTRAPYRAMIDAL SYMPTOMS (EPS)

EPS, also known as neuroleptic-induced parkinsonism, consist of the development of the classic symptoms of Parkinson's disease but in response to neuroleptic use. The most common symptoms are rigidity and akinesia, which occur in as many as half of all patients receiving long-term neuroleptic therapy. A 3- to 6-Hz tremor may be present in the head and face muscles or the limbs. Akinesia or bradykinesia are manifested by decreased spontaneous movement and may be accompanied by drooling. Rigidity consists of the classic parkinsonian "lead pipe" rigidity (rigidity that is present continuously throughout the passive range of motion of an extremity) or cogwheel rigidity (rigidity with a catch and release character).

Risk factors for the development of EPS include the use of high-potency neuroleptics, increasing age, and a prior episode of EPS. EPS usually develops within the first few weeks of therapy.

Treatment consists of reducing the dosage of antipsychotic (if possible) and adding anticholinergic medications to the regimen.

NEUROLEPTIC MALIGNANT SYNDROME (NMS)

NMS is an idiosyncratic and potentially life-threatening complication of antipsychotic drug use. Symptoms of NMS may develop gradually over a period of hours to days and can often overlap with symptoms of general medical illness. The major clinical findings in patients with NMS are presented in Table 16-2. Many symptoms of NMS are nonspecific and overlap with symptoms common to other psychiatric and medical conditions. The diagnosis of NMS is also complicated by the waxing and waning nature of the clinical picture.

Autonomic instability coupled with motor abnormalities is essential to the diagnosis of NMS. Autonomic alterations can include cardiovascular alterations with cardiac arrhythmias and labile blood pressure. Low-grade fever progressing to severe hyperthermia may also be present. Motor findings may overlap with other motor abnormalities in psychiatric illness, for example, rigidity/dystonia can be confused with simple dystonia or with EPS. Mutism can be a sign of severe psychosis or catatonia alone, although this does occur with NMS. Behavioral features such as agitation can also overlap with other psychiatric syndromes; however, the presence of delirium or seizures is a harbinger of more serious general medical illness (including drug withdrawal) or NMS. Laboratory findings may reveal an increased creatine kinase secondary to myonecrosis from sustained muscular rigidity. Liver enzymes may be elevated, but their relation to NMS is unclear. Leukocytosis is also often present.

Risk factors for the development of NMS (see Table 16-1) include the use of high-dose antipsychotics, rapid dose escalation, IM injection of antipsychotics, dehydration, agitation, or a prior history of NMS. Some factors may be related to severity of illness (e.g., severely ill patients often have poor oral intake and become dehydrated, are more likely to be placed in restraints, and require IM injection of an antipsychotic) rather than causative factors. Although NMS is most common during the first few weeks of antipsychotic drug therapy, it can occur at any time during therapy.

Treatment of this potentially fatal disorder is largely supportive. Specific interventions include discontinuation of antipsychotics (an option that may take a long period of time in individuals treated with depot antipsychotics); dantrolene (a muscle relaxant) is used to treat rigidity and decrease myonecrosis; and bromocriptine (a dopamine agonist) is sometimes used to reverse dopamine blocking effects of antipsychotics. Symptom management including intensive care with cardiac monitoring and intubation may be necessary. Symptoms of NMS overlap with serotonin syndrome (Table 16–3). However, in NMS, muscular rigidity and increased creatine kinase are prominent findings. In addition, serotonin syndrome develops in response to the use of multiple medications that affect serotonin function (especially monoamine oxidase inhibitors [MAOIs]), whereas NMS develops in response to antipsychotic medications. In patients using both MAOIs and antipsychotics (e.g., refractory psychotic depression), the differential diagnosis can be quite difficult.

TABLE 16-2

Neuroleptic Malignant Syndrome

Autonomic
Tachycardia, other cardiac arrhythmias
Hypertension
Hypotension
Diaphoresis
Fever progressing to hyperthermia
Motor
Rigidity/dystonia
Akinesia
Mutism
Dysphagia
Behavioral
Agitation
Incontinence
Delirium
Seizures
Coma
Laboratory
Increased creatine kinase
Abnormal liver function tests
Increased white blood cell count

TARDIVE DYSKINESIA (TD)

TD is a movement disorder that develops with long-term neuroleptic use; rarely, especially in the elderly,

TABLE 16-3

Serotonin Syndrome

Autonomic
Tachycardia
Hypertension
Diaphoresis
Fever progressing to hyperthermia
Motor
Shivering
Myoclonus
Tremor
Hyperreflexia
Oculomotor abnormalities
Behavioral
Restlessness
Agitation
Delirium
Coma

onset may not be as delayed. TD consists of constant, involuntary, stereotyped choreoathetoid movements most frequently confined to the head and neck musculature. At times, the extremities and respiratory and oropharyngeal musculature are also involved.

Risk factors include long-term treatment with neuroleptics, increasing age, female sex, and the presence of a mood disorder. Although TD is reversible in some cases, it tends to be permanent.

Treatment consists of changing antipsychotics, lowering the dosage, or switching to clozapine. Clozapine, which appears to work by a different mechanism from other antipsychotics, may reduce or eliminate the abnormal movements of TD.

SEROTONIN SYNDROME

Serotonin syndrome can occur when multiple medications that alter serotonin metabolism are used. Classically, this syndrome is produced when other serotonin-altering medications are used with MAOIs. This syndrome, which can be life-threatening, consists of symptoms outlined in Table 16-3. These include severe autonomic instability, motor abnormalities, and behavioral changes. The course of the disorder can become malignant and end in coma and death. A similar syndrome occurs when MAOIs are used with meperidine or dextromethorophan, and perhaps other opiates.

Serotonin syndrome has many similarities to NMS but may be distinguished in the following ways. Serotonin syndrome does not produce muscular rigidity or dystonia as does NMS. Further, NMS occurs during the use of antipsychotic medication; while serotonin syndrome occurs through use of MAOIs or other serotonergic agents.

Risk factors for serotonin syndrome, other than combining MAOIs with other serotonin-altering medications, are not known.

Treatment for serotonin syndrome is largely supportive and may require intensive care with cardiac monitoring and mechanical ventilation. The offending medications should be discontinued.

KEY POINTS

1. Major adverse drug reactions occur most commonly in psychiatry with use of antipsychotics and serotonin-altering medications.
2. Antipsychotics can cause dystonia, akathisia, EPS, NMS, and TD.
3. Serotonin-altering medications can cause akathisia and serotonin syndrome.
4. All of the above adverse drug reactions are reversible, except for TD, which may be permanent.

Chapter

17 Psychological Theories

There are a large number of competing theories influencing contemporary psychotherapeutic thinking. Psychotherapies derived from psychoanalytic, cognitive, and behavior theory are the most widely used. Cognitive and behavioral interventions have the strongest empirical verification; little empirical evidence supports the efficacy of analytic/dynamic therapies.

PSYCHOANALYTIC/ PSYCHODYNAMIC THEORY

The principal theorist responsible for launching psychoanalysis as a technique and psychodynamic theory in general is Sigmund Freud. Freud's theories proposed that unconscious motivations and early developmental influences were essential to understanding behavior. Freud's original theories have proven quite controversial and have led to the creation of various alternative or derivative theories.

Twentieth-Century Schools of Psychodynamic Psychology

There are three major twentieth-century psychodynamic schools: drive psychology, ego psychology, and object relations theory.

Drive Psychology

Drive psychology posits that infants have sexual (and other) drives. This theory proposes that sexual and aggressive instincts are present in each individual and that each individual passes sequentially through psychosexual developmental stages (oral, anal, phallic, latency, and genital). Included in drive psychology is conflict theory, which proposes to explain how character and personality development are influenced by the interaction of drives with the conscience and reality.

Ego Psychology

Freud eventually developed a tripartite theory of the mind in which the psychic structure was composed of the id, ego, and superego. Under this theory, the id is the compartment of the mind containing the drives and instincts. The superego contains the sense of right and wrong, largely derived from parental and societal morality. The ego is responsible for adaptation to the environment and for the resolution of conflict. A major function of the ego is the reduction of anxiety. Ego defenses (Table 17-1) are proposed as psychic mechanisms that protect the ego from anxiety. Some ego defenses (e.g., sublimation) are more functional to the individual than others (e.g., denial).

Object Relations Theory

Object relations theory (*objects* refers to important people in one's life) departs from drive theory in that the relationship to an object is motivated by the primacy of the relationship rather than the object being a means of satisfying a drive. Child observation furthered object relations theory, emphasizing concepts of attachment and separation.

The interpersonal school arose as an outgrowth of object relations theory. The interpersonal theorists emphasize that intrapsychic conflicts are less important than one's relationship to one's sense of self and to others. In other words, the relationships in a

TABLE 17-1

Common Ego Defense Mechanisms

Denial	Feelings or ideas that are distressing to the ego are blocked by refusing to recognize evidence for their existence.
Projection	Feelings or ideas that are distressing to the ego are attributed to others.
Regression	Feelings or ideas that are distressing to the ego are reduced by behavioral return to an earlier development phase.
Repression	Feelings or ideas that are distressing to the ego are relegated to the unconscious.
Reaction formation	Feelings or ideas that are distressing to the ego are converted into their opposites.
Displacement	Feelings or ideas that are distressing to the ego are redirected to a substitute that evokes a less intense emotional response.
Rationalization	Feelings or ideas that are distressing to the ego are dealt with by creating an acceptable alternative explanation.
Suppression	Feelings or ideas that are distressing to the ego are not dealt with, but they remain components of conscious awareness.
Sublimation	Feelings or ideas that are distressing to the ego are converted to those that are more acceptable.

Modified from Sadock BJ, Sadock VA. Comprehensive textbook of psychiatry. 7th ed. Philadelphia: Lippincott, Williams & Wilkins, 1999.

person's life are given primary importance in producing happiness or misery.

ERIKSON'S LIFE CYCLE THEORY

Erik Erikson made major contributions to the concept of ego development. Erikson theorized that ego development persists throughout one's life. Erikson conceptualized that psychosocial events drive change, leading to a developmental crisis. According to Erikson's model, individuals pass through a series of life cycle stages (Table 17-2). Each stage presents core conflicts produced by the interaction of developmental possibility with the external world. Individual progress and associated ego development occur with successful resolution of the developmental crisis inherent in each stage. This model allows for continued ego development until death.

COGNITIVE THEORY

Cognitive theory recognizes the importance of the subjective experience of oneself, others, and the world. It posits that irrational beliefs and thoughts about oneself, the world, and one's future can lead to psychopathology.

In cognitive theory, thoughts or cognitions regarding an experience determine the emotions that are evoked by the experience. For example, the perception of danger in a situation naturally leads to anxiety. When danger is truly present, anxiety can be adaptive, leading to hypervigilance and self-protection. When the situation is only perceived as dangerous (such as in fear of public speaking), the resulting anxiety can be psychologically paralyzing. A person may fear public speaking because of an irrational fear that something disastrous will occur in public. A principal type of irrational belief is a cognitive distortion (Table 17-3).

BEHAVIORAL THEORY

Behavioral theory posits that behaviors are fashioned through various forms of learning, including modeling, classical conditioning, and operant conditioning (Table 17-4). A behaviorist might propose that through operant conditioning, depression is caused by a lack of positive reinforcement (as may occur after the death of a spouse), resulting in a general lack of interest in behaviors that were once pleasurable (or reinforced).

TABLE 17-2

Erikson's Life Cycle Stages

Trust versus mistrust	Birth to 18 mos.	The infant has many needs but does not have the power to have those needs met. The child is dependent on caretakers. If caretaking is appropriate, a sense of trust and hope are created. If inappropriate or inadequate, mistrust develops.
Autonomy versus shame	18 mos. to 3 yrs.	The child is learning about the use of language and control of bowel and bladder function and walking. As a result, it begins to choose to influence and explore the world. If caretaking is appropriate, the child will develop a healthy balance between exerting its autonomy and feeling shame over the consequences of exerting autonomy.
Initiative versus guilt	3–5 yrs.	As the child develops increasing control of language and walking, he or she has increased initiative to explore the world. The potential for action carries with it the risk of guilt at indulging forbidden wishes.
Industry versus inferiority	5–13 yrs.	The child begins to develop a sense of self based on the things he or she creates. Caretaker influences are important in helping the child develop a sense of mastery and competence over creating.
Identity versus role confusion	13–21 yrs.	Corresponds to adolescence. How one appears to others is important in this stage. There are conflicts between one's identity and the need to gain acceptance.
Intimacy versus isolation	21–40 yrs.	The anxiety and vulnerability produced by intimacy are balanced against the loneliness produced by isolation.
Generativity versus stagnation	40–60 yrs.	If successful, the individual develops a positive view of his or her role in life and a sense of commitment to society at large. If unsuccessful, individuals move through life without concern for the greater welfare.
Ego integrity versus despair	60 yrs. to death	An individual accepts his or her life course as appropriate and necessary. If this fails, the individual may regret or wish to relive some part of life, leading to despair.

Modified from Sadock BJ, Sadock VA. Comprehensive textbook of psychiatry. 7th ed. Philadelphia: Lippincott, Williams & Wilkins, 1999.

TABLE 17-3

Types of Cognitive Distortions

Arbitrary inference	Drawing a specific conclusion without sufficient evidence
Dichotomous thinking	A tendency to categorize experience as "all or none"
Overgeneralization	Forming and applying a general conclusion based on an isolated event
Magnification/ minimization	Over- or undervaluing the significance of a particular event

Modified from Sadock BJ, Sadock VA. Comprehensive textbook of psychiatry. 7th ed. Philadelphia: Lippincott, Williams & Wilkins, 1999.

TABLE 17-4

Important Concepts in Behavior Theory

Modeling	A form of learning based on observing others and imitating their actions and responses
Classical conditioning	A form of learning in which a neutral stimulus is repetitively paired with a natural stimulus, with the result that the previously neutral stimulus alone becomes capable of eliciting the same response as the natural stimulus
Operant conditioning	A form of learning in which environmental events (contingencies) influence the acquisition of new behaviors or the extinction of existing behaviors

COGNITIVE-BEHAVIORAL THERAPY (CBT)

Cognitive and behavioral theories form part of the bases of CBT. CBT involves the examination of cognitive distortions and the use of behavioral techniques to treat common disorders such as major depression.

KEY POINTS

1. Psychological theories are numerous, but those derived from psychoanalytic, cognitive, and behavioral theories are most widely used.
2. The psychoanalytic school emphasizes unconscious motivations and early influences.
3. The cognitive school emphasizes subjective experience, beliefs, and thoughts.
4. The behavioral school emphasizes the influence of learning.
5. The cognitive and behavioral schools have the greatest empirical support.

18 Legal Issues

Legal issues affect all areas of medicine, including psychiatry. The laws that govern medical practice address physician duty, negligence, and malpractice as well as patient competence, consent, and right to refuse treatment. Previous court decisions, or precedents, are used as the standard by which a given action (or inaction) is judged. Practitioners should be aware of the pertinent laws of the state in which they practice in order to comply adequately with standards of practice while respecting the rights and duties of their role. Reducing adverse events and legal claims in the health care system is known as risk management.

MALPRACTICE

The legal definition of malpractice requires the presence of four elements: negligence, duty, direct causation, and damages. Negligence can be thought of as failure to perform some task with respect to the patient, falling short of the care that would be provided by the average practitioner (the standard of care). Duty reflects the law's recognition of the obligation of the physician to provide proper care to his or her patients. Direct causation requires that the negligence directly caused the alleged damages. Finally, damages (e.g., physical or emotional harm) must in fact be shown to have occurred. In short, malpractice involves the negligence of a duty that directly causes damages. Malpractice claims in psychiatry principally involve suicides of patients in treatment, misdiagnosis, medication complications, false imprisonment (involuntary hospitalization or seclusion), and sexual relations with patients.

INFORMED CONSENT

Informed consent has three components: information, competence, and consent. First, appropriate levels of information regarding a proposed treatment, including side effects, alternative treatments, and outcome without treatment, must be provided. Second, the patient must be competent (i.e., have the capacity to understand, reason, and make reasonable decisions regarding the risks and benefits of treatment). Third, the patient must give consent voluntarily. True emergency situations are an exception to this rule; treatments necessary to stabilize a patient in an emergency can be given without informed consent.

INVOLUNTARY COMMITMENT

Commitments are generally judicially supported actions that require persons to be hospitalized or treated against their will. Although laws vary from state to state, commitment criteria usually require evidence that the patient is a danger to self, a danger to others, or is unable to care for him- or herself. Psychiatrists, in most localities, have the right to temporarily involuntarily commit a patient if any of these criteria are met and a diagnosis of a mental disorder is provided (in other words, both a mental disorder and danger must exist). The duration of temporary commitment and the rights of the patient vary by jurisdiction. Patients who have been committed have a right to be treated, and unless they have been declared incompetent, they have the right to refuse treatment.

THE TARASOFF DECISIONS: DUTY TO WARN (OR PROTECT)

Tarasoff *v* The Board of Regents of the University of California (or simply, Tarasoff) was a landmark case that was heard twice in the California Supreme Court in 1976. Tarasoff I held that therapists have a duty to warn the potential victims of their patients. Tarasoff II held that therapists have a duty to take reasonable steps to protect potential victims of their patients. In most localities, this means that the therapist should take reasonable action to protect a third party if a patient has specifically identified the third party and a risk of serious harm seems imminent.

M'NAGHTEN RULE: THE INSANITY DEFENSE

Named after a mentally ill man (M'Naghten) who attempted to assassinate the prime minister of England in 1843, this rule forms the basis of the insanity defense. According to the M'Naghten rule, a person is not held responsible for a criminal act if at the time of the act he or she suffered from mental illness or mental retardation *and* did not understand the nature of the act *or* realize that it was wrong. In more recent history, the appropriate use of the insanity defense has been called into question. Some legal theorists argue for the designation "guilty but insane" to indicate culpability but at the same time recognize the presence of a mental illness (and presumably the need for treatment).

KEY POINTS

1. Malpractice consists of negligence, duty, direct causation, and damages.
2. The Tarasoff decision led to an expectation that therapists and doctors take reasonable action to protect persons whom their patients have specifically threatened.
3. The M'Naghten rule is the basis of the insanity defense.

Questions

1. A 24-year-old man with a history of schizophrenia presents with a fever of 102°F, shaking, chills, and rigors. He was well until a few days ago when he returned from an unplanned trip across the country by train. He had missed a weekly CBC that he had been receiving since beginning a new antipsychotic medication 6 weeks ago. A stat CBC reveals a WBC count of 0.2. The most likely antipsychotic agent the patient has been taking is:
   a. Risperidone
   b. Olanzapine
   c. Clozapine
   d. Quetiapine
   e. Thioridazine

2. A 19-year-old woman presents for a gynecological examination and reports that she has been feeling sad lately. Further inquiry reveals that she also has lack of energy, lack of enjoyment of usual activities, trouble concentrating, and has been sleeping and eating more than usual. She was well until these symptoms began more than 2 months ago. She notes that her father died 8 months ago. You notice on her clinic record that her primary care physician checked a TSH level a month ago that was normal at 1.4. She reports occasional alcohol use of 1 to 2 drinks per week. The most likely diagnosis is:
   a. Dysthymic disorder
   b. Major depression
   c. Mood disorder due to a general medical condition
   d. Substance-induced mood disorder
   e. Normal bereavement

3. Three weeks after beginning fluoxetine for an episode of depression, a student is brought to the emergency room by her roommate. Approximately 1 week ago, the patient began acting strangely at her dormitory. She began staying up all night working on projects that were not required of her classes and despite only getting 2 to 3 hours of sleep per night, reported feeling rested. She was speaking very rapidly and kept referring to a top-secret study involving the FBI and the CIA. She shifted rapidly from excited, agitated expressions to tearful exasperation. She reported never having felt this way before and said that she had been taking the fluoxetine as instructed. The diagnosis at this time is:
   a. Schizoaffective disorder
   b. Bipolar disorder
   c. Schizophrenia
   d. Substance-induced mood disorder
   e. Borderline personality disorder

4. An 85-year-old retired dean of a local university is brought in by her son for a routine visit to her primary care provider. He reports that she is progressively more forgetful and is now somewhat paranoid, accusing him of stealing her money and planning to put her in a nursing home. She has no prior history of cardiovascular or cerebrovascular illness. She has no known HIV risk factors. There is a family history of dementia in an older sibling. On exam the patient is alert, pleasant, and engaging. She shows evidence of mild anomia on her mental status examination, and doesn't know the names of current national political leaders but can recite details of political events and parties from 20 years prior. She states that she is convinced that her son is out to "take her money and put her away," but cannot describe any evidence that would support this conclusion. Neurological exam is nonfocal. Head CT scan with and without contrast is normal except for mild cortical atrophy. HIV test is negative. Thiamine, folate, vitamin B_{12}, and niacin levels are normal. Serum RPR is negative. The most likely diagnosis is:
   a. Vascular dementia
   b. Tertiary neurosyphilis
   c. Major depression
   d. Alzheimer's dementia
   e. Psychotic disorder

5. A 39-year-old man visits a new primary care physician for a first visit at the urging of his mother. He denies symptoms of depression except for occasional low mood. His father died when he was 14 and he has lived with his mother since that time. He works as a computer programmer and says that he has many acquaintances but few friends and no romantic relationships. His mother had to convince him to go to the appointment because he had trouble deciding to see a physician. His mother accompanies him on the visit and relays that she often decides things for him because he has "never been able to think for himself." He says, "People think I'm too clingy. The truth is I just want people to care about me." When you speak with him alone, he appears to have a normal intellect, no magical or odd thinking, and a stable sense of self but reveals a desperate fear that his mother will abandon him. He denies any self-destructive or impulsive behaviors. The most likely diagnosis is:

a. Borderline personality disorder
b. Dependent personality disorder
c. Schizotypal personality disorder
d. Narcissistic personality disorder
e. Schizoid personality disorder

6. A 77-year-old female with a history of mild Alzheimer's dementia is evaluated in the EW for confusion. Her family reports that she was at her baseline until this morning when she became increasingly disoriented, no longer recognizing family members she had known the day before. In addition, she repeatedly complained of hearing voices down the hall when there was only a television. On examination, she is initially pleasant and alert. She is oriented to person, but is convinced that she is in an army barracks and that you and the other medical staff are military police. She can spell the word *world* forward but not backwards. Later in the interview she seems distracted and then dozes off. Laboratory results reveal normal findings except for a mild anemia, increased WBC with increased PMNs and band forms. Urinalysis reveals no blood, 50–100 WBC per HPF, gross bacturia. The most likely diagnosis is:

a. Worsening dementia
b. Delirium
c. Psychotic disorder
d. Major depressive disorder
e. Meningitis

7. A 23-year-old male who is in psychopharmacological and psychotherapy treatment with you for major depression associated with a recent romantic break-up states that he would like to kill the new boyfriend of his ex. He has no history of violence, but has spoken at length of his rage, humiliation, and powerlessness in regard to the breakup. He tells you the intended victim's name and address, saying that everything will be fine once his rival is out of the picture. You are very concerned and offer the patient an inpatient admission; in addition, you tell him you believe you need to warn the intended victim and the police. The patient refuses an admission and refuses to allow you to contact any outside parties, stating that you are his therapist, and are therefore bound by confidentiality laws. You fear that you will lose your license if you disclose the contents of the session. The patient then storms from your office, yelling that you can't be trusted. You should:

a. Notify the police and the intended victim of your patient's statements.
b. Request a case consultation from the hospital ethicist.
c. Attempt to contact your patient's family so they can convince him to cool down.
d. Respect the confidentiality of the therapy session.
e. Consider raising the patient's medication dosage at the next meeting.

8. A 53-year-old male is brought to the EW by the police after he was found wandering in traffic. He is dishevelled, agitated, and confused. On physical examination he has vital signs of blood pressure 200/110, heart rate of 124, respirations 22, temperature 101°F. He has prominent dilated pupils, symmetrical hyperreflexia, acne rosacea, palmar erythema, and tender hepatomegaly. On mental status examination he is oriented only to person. He has difficulty paying attention to your questions, at times appearing to focus on the interview but at other times commenting on bugs crawling on the walls. He cannot provide any useful history. He has intermittent episodes of out of control behavior, eventually requiring restraints by the EW staff. Laboratory exams reveal mild pancytopenia on CBC with an increased MCV; electrolytes are normal with the exception of reduced magnesium; liver functions show increased liver enzymes with decreased albumin. Urine and serum toxicology screens are negative. Head CT is negative. LP is normal. The most likely diagnosis is:

a. Dementia
b. Schizophrenia
c. Opiate withdrawal
d. Cocaine withdrawal
e. Alcohol withdrawal

9. A 20-year-old female college student comes to the emergency room complaining of brief episodes of "fluttering" in her chest and chronic lightheadedness. She has a benign medical history and takes no medications. On physical exam, her skin is pale, her heart rate is 54, and her blood pressure is 88/50. When she reluctantly partially disrobes for further examination, you note an emaciated body. She weighs 78 pounds and stands

5' 6". When you comment on her low body weight, she blithely states, "I'm a fat cow." She says that she has been exercising for 3 to 4 hours per day since beginning college 2 years ago. She restricts herself to 900 calories per day, which she counts compulsively. She has not had her period in 14 months. She denies depressed mood and, other than her distorted body image, appears to have intact reasoning. She denies binging or purging behavior. The most likely diagnosis is:

a. Bulimia nervosa, nonpurging type
b. Anorexia nervosa, binge eating/purging type
c. Delusional disorder, somatic type
d. Anorexia nervosa, restricting type
e. Body dysmorphic disorder

10. An 85-year-old woman with a history of slowly progressive dementia is brought from a local nursing home to the emergency room due to increasing levels of agitation and confusion. On arrival, her face is flushed with anhidrosis and her pupils are dilated. Her blood pressure is 160/90, heart rate is 99, respiratory rate is 16, and body temperature is 99.6°F. She is disoriented to place and time, complains of blurry vision, constipation, and a "fullness" in her bladder. The nursing home records indicate that the patient has been increasingly agitated and that numerous medications have been tried to decrease the agitation. The accompanying staff member also notes that she has become this confused over the last 24 hours. She has not experienced any falls and her physical examination, including full neurological and fundoscopic examination, is otherwise normal. The most likely diagnosis is:

a. Worsening dementia
b. Pseudodementia
c. Cerebral haemorrhage
d. Anticholinergic delirium
e. Cerebrovascular accident

11. A 64-year-old woman is seen in an outpatient medical clinic 2 months after an admission for a CVA. Her initial symptoms of right facial and arm paresis have now resolved completely. However, she complains of feeling "not her usual self." She says that she used to enjoy going to visit her daughter and grandchildren once a week but that she has not been in 4 weeks. For this, she reports feeling "terribly guilty." She also says that a man that she was dating just prior to the stroke has been calling and she has not returned his calls. She says, "I just don't want anyone to see me like this." When asked if she has been feeling sad, she begins crying and says that she has and that she doesn't understand it because she has never felt this way before. Her history and physical examination are within normal limits though she says, "I feel like I am just wasting away." She has lost 15 pounds from apparent poor appetite. The most likely diagnosis is:

a. Mood disorder due to a general medical condition
b. Avoidant personality disorder
c. Somatization disorder
d. Social phobia
e. Dysthymic disorder

12. A Vietnam veteran seeks psychiatric care. He reports that he was in good psychological health until his combat experiences, when he witnessed the violent death of multiple friends. In addition, he was severely wounded from a land mine, but without a loss of consciousness during the injury. He has minimal physical disability from his injuries, but notes that for several years after his discharge he was alcohol dependent, losing his job and his home. He has remained abstinent from alcohol for more than 10 years with the help of individual CBT and AA. Despite maintained sobriety, he has remained on mental health disability because of an inability to work due to continual reexperiencing of combat trauma, including flashbacks and vivid dreams that awaken him from sleep. He notes that he is always on edge, alert to the slightest sound, and that he makes every attempt to avoid news reports of combat and carefully avoids movies containing war imagery. The most likely diagnosis is:

a. Dissociative identity disorder (DID)
b. Generalized anxiety disorder (GAD)
c. Major depressive disorder
d. Complex partial epilepsy
e. Posttraumatic stress disorder (PTSD)

13. A 27-year-old female is brought to the EW by the local police because she has been harassing her neighbors. Her family arrives shortly afterward and appears very concerned. The police report that the young woman's apartment manager called because she was banging on all her neighbors' doors and screaming in the stairwell. Upon examining the patient's apartment, the police found the place to be filthy and malodorous, with rotting garbage and food in the kitchen. The windows were sealed and covered with dark curtains, and there were several televisions and computers in the living room, all turned on at the same time. The patient's family reports that for the past year or so she has seemed increasingly odd, dissociating herself from family activities and giving up a well-paying job as a computer system administrator. In addition, she had abruptly ended a long-term romantic relationship for no particular reason. On mental status examination the young woman is dishevelled and wearing several layers of dirty clothes. She appears wary and guarded. Her speech is normal in rate, volume, and production. She is conversant, and explains that she has been defending herself against aliens who want to use her as a specimen. She believes that she began picking up on hidden transmissions in her e-mail at work, reveal-

ing an alien conspiracy, perhaps involving the CIA. She states that she left her job to monitor these hidden transmissions full-time and that she has been hiding in her apartment because they know she is on to them. She ended her recent relationships because she felt that the aliens would harm the people she cared about. She denies substance use or any medical symptoms. She reports that she has been eating and sleeping well, and that her mood is good. She denies hearing voices. Diagnostic testing, including drug screen, CBC, chemistry panel, and brain MRI are all normal. The most likely diagnosis is:

a. Schizophrenia
b. Bipolar disorder
c. Major depression
d. Alcohol abuse
e. Paranoid personality disorder

14. An 18-year-old man is seen in a prison clinic for a routine physical exam. He tells you that he is in jail for murdering two people in a botched robbery attempt. When you ask him about his other criminal history, he relays numerous arrests and some convictions for theft, assault, drunk driving, and rape. Penal records indicate a persistent pattern of lying and theft while incarcerated and that he exhibits no remorse when caught by authorities. He has never been psychiatrically hospitalized and denies any episodes of elevated, expansive, or fluctuating mood. He exhibits no grandiosity, pressured speech, or psychotic symptoms. Family history is significant for alcohol dependence in his father and mother and for no history of bipolar disorder. He calmly reports that his father and subsequent foster parents often beat him with straps or boards when he was a child. He was knocked unconscious on several occasions but has no history of seizures and denies nightmares or flashbacks from the abuse. He has had suicidal ideation but has not made suicide attempts or been self-destructive. The most likely diagnosis is:

a. Conduct disorder
b. Posttraumatic stress disorder (PTSD)
c. Cyclothymic disorder
d. Bipolar disorder
e. Antisocial personality disorder (ASP)

15. A 33-year-old male is referred for a substance abuse evaluation after he was arrested for driving while intoxicated. He is reluctant, but realizes he must cooperate with the evaluation to avoid more serious legal consequences. He states that alcohol is not really a problem for him, and that he can control his drinking saying, "I can quit whenever I want to." He does admit to two prior drunken driving arrests. He reports that he was fired from two different jobs for "smelling like alcohol," but believes he was really fired because his bosses were "jerks." His most recent romantic relationship also ended because he drank too much. He reports that he doesn't drink daily, only after work a few times a week and more heavily on weekends. He reports no symptoms of alcohol withdrawal, and no prior attempts to reduce his level of alcohol intake. The most likely diagnosis is:

a. Narcissistic personality disorder
b. Alcohol dependence
c. Antisocial personality disorder (ASP)
d. Alcohol abuse
e. Alcohol intoxication

16. A 23-year-old woman comes to an emergency room after being found unconscious with an empty bottle of 1-mg tablets of lorazepam by her body. She is awake upon arrival in the ER but appears sedated and has a respiratory rate of 10, heart rate of 64, and blood pressure of 90/60. She is immediately given IV naloxone with no effect. Several minutes later a dose of a different IV medication is given, which results in increased alertness. Her boyfriend relays that the two have been together for a few weeks and that she was getting "really serious, really soon." A few hours prior to the overdose, he reports that he told her that he didn't want to move in with her. She became visibly upset and he left. After the patient is more awake, you begin to interview her. She relays that she has been in this situation before and "can't believe" that it is happening again. She reports a series of volatile relationships with both men and women. Each relationship involves rapidly "falling in love" with a "perfect" person followed by a period in which the person disappoints her. Two years ago, she threatened to jump out of a window after her boyfriend broke up with her. She has been in psychotherapy since that time and has recently been prescribed lorazepam for "intolerable anxiety." During the interview, she is intermittently tearful, but her speech is normal in rate and prosody. She exhibits no delusional beliefs and denies auditory hallucinations. The IV medication that aroused the above patient is likely:

a. Naloxone
b. Thiamine
c. Methohexital
d. Flumazenil
e. Diazepam

17. A 41-year-old man is seen in an outpatient psychiatrist's office complaining of long-standing feelings of sadness, low self-esteem, and low motivation that he has experienced most of the time for many years. He has never been hospitalized, never made a suicide attempt, and never been affected enough by the symptoms to stop working or to limit his active social life. He recently got divorced. While he thinks that his symptoms have contributed to

his difficulties in significant relationships, he is not particularly distressed about the divorce. The most likely diagnosis is:

a. Major depression
b. Schizoid personality disorder
c. Dysthymic disorder
d. Avoidant personality disorder
e. Adjustment disorder with depressed mood

18. A 3-year-old boy is evaluated in the pediatric general practice clinic because his mother thinks he's "too quiet." She notes specifically that her pregnancy and delivery were uncomplicated. A few months after birth she noted that her child did not seem to respond to her attempts to communicate with him. He failed to respond when smiled at, had almost no facial expressions, and didn't maintain eye contact. He didn't babble or begin talking like other children his age, but used only occasional odd-sounding phrases in a repetitive fashion. In addition, she worries that he sits for hours rocking back and forth. He doesn't play with other children, and overall shows little interest in his external world. The most likely diagnosis is:

a. Autism
b. Childhood onset schizophrenia
c. Conduct disorder
d. Attention deficit disorder
e. Separation anxiety disorder

19. A 47-year-old male is evaluated in the EW of a teaching hospital because he is a little confused and has run out of his medications. His wife reports that he filled a prescription for amitryptiline a few days ago, and now reports that he is out of them. He seems somewhat sedated and displays some lip smacking, as if he has a dry mouth. He says he may have taken a few extra pills to sleep better, but that he is not suicidal, nor has he made any overdose attempt. His wife concurs that he has not made suicidal statements. Vital signs show pulse of 110, blood pressure 140/80, respirations 14/min, temp 99°F. Because the history given by the patient and his wife seems reliable and because he does not have a declining level of consciousness in the EW, he is discharged to follow up with his psychiatrist the next day for evaluation of his need for a refill. Two hours later he is brought in by ACLS ambulance in cardiac arrest after he collapsed at home and turned blue. His wife could not detect a pulse, and administered CPR until the ambulance came. The patient cannot be resuscitated and is pronounced dead shortly after arriving in the EW for the second time. The most likely cause of death in this patient is:

a. Pulmonary embolism
b. Myocardial infarction
c. Cardiac arrhythmia
d. Opiate overdose
e. Intracranial bleeding

20. A 46-year-old female corporate executive comes to her primary care physician reporting that her partner says that she sits up in bed and screams or cries out and appears extremely frightened. She does not awaken during the episodes and does not recall their occurrence. Her partner also notes that she snores loudly. She reports feeling rested despite the episodes. The most likely diagnosis is:

a. Sleep terror disorder
b. Narcolepsy
c. Breathing-related sleep disorder
d. Nightmare disorder
e. Sleepwalking disorder

21. A 19-year-old man is seen on rounds on a psychiatric unit after being admitted two nights before. He was diagnosed with a psychotic disorder not otherwise specified and begun on haloperidol 2.5 mg BID. The nurse asked you to see him first because he had been "agitated all morning." When you enter his room, you find him pacing the floor. He tells you that he feels restless and that he has to keep moving to feel better. Other than pacing, he is not exhibiting any tremors or other movements of the extremities. Although he appears agitated, his psychotic symptoms, which include hallucinations and delusions, do not appear to have worsened since admission. Vital signs are within normal limits and he is afebrile. A serum CPK is drawn and is normal. The most likely diagnosis is:

a. Dystonia
b. Parkinsonism
c. Akathisia
d. Neuroleptic malignant syndrome
e. Tardive dyskinesia

22. A 15-year-old male is brought to his pediatrician's office because his parents are embarrassed by his behavior. They report that he has been intermittently barking and yelping repetitively during conversations. This behavior persists in public and at school. In addition, he has periods of bizarre body posturing with jerky upper extremity movements and snorts like a horse. His parents believe he is punishing them for setting limits on television and social time with his friends. The young man denies any retaliation against his parents. He states that he develops a powerful urge to bark and yelp, and that other times he finds himself moving uncontrollably. He is upset about his behaviors and feels like a "freak." The most likely reason for the young man's behavior is:

a. Adolescent rebellion
b. Schizophrenia
c. Substance abuse
d. Tourette's disorder
e. Conduct disorder

23. A 28-year-old woman tells her gynecologist that she has never had an orgasm during sexual intercourse. She reports that she has an active libido and masturbates to orgasm frequently. She reports that she and her partner are committed to each other and that they are affectionate. She says that when they are together she becomes aroused with engorgement of the mons pubis and vaginal lubrication, but whenever her partner attempts to enter her vagina she feels "disgusted" and "loses all interest." Her vagina does not prevent entry and entry is not painful. She denies other psychiatric or medical problems. However, she acknowledges a history of childhood sexual abuse. The most likely diagnosis is:

a. Female orgasmic disorder
b. Vaginismus
c. Hypoactive sexual desire disorder
d. Sexual aversion disorder
e. Female sexual arousal disorder

24. A 24-year-old female with no prior psychiatric or medical history is brought to the EW by a friend because she felt that she was having a heart attack. The patient has no known risk factors for coronary artery disease. She reports a sense of difficulty breathing, chest tightness, and nausea. She is agitated, pacing, and very concerned that she might be dying. On physical exam she is tachypnic, tachycardic, and flushed but is otherwise normal. Diagnostic testing, including an EKG, pulse oximetry, erythrocyte sedimentation rate, cardiac enzymes, drug screen, and TSH, shows no abnormalities. The most likely diagnosis for this patient is:

a. Unstable angina
b. Generalized anxiety disorder (GAD)
c. Panic disorder
d. Major depressive disorder
e. Alcohol intoxication

25. A 29-year-old male creative writing professor is evaluated by his primary care provider in late December because he has been feeling increasingly tired, apathetic, and depressed for the past 2 months. He reports that he has felt like this from December through February of the past 5 years after moving to Seattle, Washington, from Miami, Florida. He reports that he feels as if he can't get out of bed in the mornings, has stopped participating in sports or social activities, and is feeling increasingly depressed with poor concentration and an inability to work. He also notes an increased appetite, particularly for carbohydrates, and has gained 10 pounds in the past month. He is quite concerned that he is now "too lazy" to perform any creative work. His physical examination is normal with the exception that he is about 15 pounds over ideal body weight. Diagnostic testing including CBC, electrolyte panel, and TSH is normal. The most likely diagnosis at this point is:

a. Adjustment disorder with depressed mood
b. Mood disorder due to a general medical condition
c. Major depressive disorder
d. Dysthymic disorder
e. Schizophrenia

26. A 43-year-old male who is a former heroin addict and is now maintained on methadone at 80 mg/day is admitted to the surgery service following an automobile accident in which he sustained a tibia/fibula fracture requiring open reduction with internal fixation. Because of his history of heroin dependence, the intern orders ketorolac (Toradol) instead of the routine patient-controlled analgesia (PCA) pump, but does allow an order of oxycodone/acetaminophen (Percocet) at 1 tablet p.o. every 6 hours as needed for breakthrough pain. The patient is also given his 80 mg maintenance dosage of methadone. Despite the maximum dose of Toradol and use of the prescribed Percocet, the patient complains of severe pain, stating that the Percocet "isn't touching the pain." On examination, the patient is diaphoretic, pulse is 120, blood pressure 180/100, respirations 30/minute. The best intervention at present is to:

a. Tell the patient you will add meperidine
b. Tell the patient he is drug seeking and needs to stick with the prescribed regimen
c. Decrease the Percocet because the patient is likely to abuse it
d. Switch the patient to a PCA pump and titrate the dose to pain relief
e. Tell the patient he shouldn't be in pain because he is receiving "a lot" of opiates

27. A 47-year-old female with a 30-year history of heavy alcohol dependence is evaluated in the EW after "tripping" on the stairs and hitting her head. She is disheveled, has a bloody nose, and reeks of alcohol. On mental status examination, she is initially intoxicated and uncooperative. She is admitted to the hospital for detoxification. After a successful detoxification you conduct a more thorough mental status examination. The patient reports that she is in a good mood and has no complaints. She says she thinks she will stay away from alcohol this time. She is jovial, but does not appear to remember your name or having met you before. This has happened twice today. The most likely diagnosis is:

a. Anxiety disorder
b. Epidural hematoma
c. Alcohol-induced persisting amnestic disorder
d. Alzheimer's dementia
e. Alcohol withdrawal delirium

28. A 41-year-old man with a 21-year history of bipolar disorder comes to the EW with ataxia, coarse tremor of his upper extremities, confusion, and diarrhea. He reports

that he has been sweating a great deal because it is "so hot outside" and that he "gets no relief" at home because his halfway house is not air-conditioned. His medications are fluoxetine 40 mg per day, lithium carbonate 300 mg each morning and 600 mg each evening, carbamazepine 300 mg each morning and 400 mg each evening, olanzapine 2.5 mg each evening, and lorazepam 1 mg TID. He denies having used any illicit drugs. On examination, his heart rate is 100 and regular, blood pressure is 98/60, respiratory rate is 14 and temperature is 98.7°F. The most likely diagnosis is:

a. Lithium toxicity
b. Lorazepam toxicity
c. Fluoxetine toxicity
d. Carbamazepine toxicity
e. Haloperidol toxicity

29. A 59-year-old female is interviewed in the EW after her sister brought her in because she was afraid she might "do something crazy." The sister reports that the patient has been depressed for a very long time, and that she has been undergoing serious stress. She states that the patient has been saying she would be better off dead, and that life isn't worth living. The patient reports that she has been progressively more depressed during the past month. She recounts stressors of losing her job one month ago due to cutbacks, but she believes they let her go because she wasn't performing well. In addition, 3 weeks ago she ended a long-term romantic relationship of 5 years after a great deal of anger and turmoil. One week prior to admission she learned that her previously rent-controlled apartment where she had lived for many years was being sold and that she would have to move. She loves her neighborhood, but won't be able to afford living there any longer. She reports that she has felt seriously depressed for at least one month, and has had marked neurovegetative symptoms of depression. In addition, she reports a profound sense of anhedonia, and spends her days lying in bed without eating or showering. She notes that she is hopeless about the future, and that she doesn't believe she will ever feel better. She is vague when directly questioned about suicidality. You make the diagnosis of major depression, but you are concerned about the patient's suicide risk. The most important risk factor for suicide in this patient is:

a. Major depression
b. Neurovegetative symptoms
c. Loss of a romantic relationship
d. Loss of her job
e. Hopelessness

30. A 16-year-old boy with no known alcohol or drug history presents to the emergency room appearing confused. As you are examining him he tells you that your face looks like it is "melting." His breathalyzer is negative for alcohol. The boy most likely ingested:

a. Rohypnol
b. GHB
c. LSD
d. Alcohol
e. Heroin

31. A male who appears to be in his mid 30s is brought to the EW by a friend because the patient was turning blue. On brief observation the patient is pale, with perioral cyanosis, periungual cyanosis, and shallow respirations of 3–5/minute. He is unresponsive except that he moans to deep sternal rub. The friend reports that the patient "may have taken something to relax," but he denies any idea of what it might have been or the mode of ingestion. No track marks are visible on the patient's body. The most likely drug the patient ingested was:

a. A benzodiazepine
b. Alcohol
c. Cocaine
d. Ketamine
e. An opiate

32. A 17-year-old male visits his primary care provider because his parents are worried about his behavior. They complain that he is always in the bathroom, taking hours during showering in the morning and frequently spending long periods of time in hand washing throughout the day. He explains that he has to make sure to get rid of all the germs on his body, and that he has to make sure they are all gone before he leaves. His hands are somewhat dry and chapped. The most likely diagnosis for this patient is:

a. Obsessive-compulsive disorder (OCD)
b. Panic disorder
c. Generalized anxiety disorder (GAD)
d. Body dysmorphic disorder
e. Narcissistic personality disorder

33. A 23-year-old medical student is seen in a first psychotherapy visit for stress and feelings of sadness associated with the break up of a significant same-sex romantic relationship. The therapist evaluates the patient thoroughly, and finds no evidence of major depression or anxiety disorder. The student expresses a long-standing same-sex sexual and romantic orientation, but notes that life seems simpler for heterosexuals. The psychotherapist suggests the student should enter reparative, or change psychotherapy, to develop a heterosexual orientation. The therapist:

a. Should find out whether the student wants to change
b. Is proposing an evidence-based treatment modality
c. Should send the student to reparative therapy group therapy

d. Should send the student to reparative therapy individual therapy
e. Is proposing an unproven and ethically questionable treatment

34. An 18-year-old college student is brought to the emergency room by the campus police due to creating a disturbance on campus. On examination he has auditory hallucinations, agitation, and rapid, incoherent speech. The length of time that he has had the symptoms is unknown. Substance abuse history is unknown. The most likely diagnosis is:
a. Schizophreniform disorder
b. Winter depression
c. Generalized anxiety disorder (GAD)
d. Atypical depression
e. Obsessive-compulsive disorder (OCD)

35. A 29-year-old investment banker is brought to the emergency room by his business partners because of increasing paranoia. The partners note that the patient has been working almost around the clock for the past week to meet a critical deadline. On exam the patient is tense, tremulous, paranoid, hypervigilant, tachycardic, and hypertensive. His urine drug screen is positive for amphetamines. The patient reluctantly admits to taking about a bottle of an unknown type of uppers during the past week. The most effective agent for treating this condition would be:
a. Buspirone
b. Sertraline
c. Flumazenil
d. Dextroamphetamine
e. Olanzapine

36. The mother of a 27-year-old psychotic female is contacted (after obtaining permission from the patient) prior to initiating treatment. You explain to the mother that a psychosis is present and that initiation of an antipsychotic medication is the appropriate treatment. The mother says that she is worried about long-term side effects because her uncle was treated with stelazine for many years and now has strange, slow movements of his upper body and mouth. Of the following antipsychotics, which has the highest risk of causing tardive dyskinesia?
a. Haloperidol
b. Risperidone
c. Clozapine
d. Quetiapine
e. Olanzapine

37. A 53-year-old male patient suffers sudden death due to a presumed cardiac arrhythmia associated with TCA toxicity. The patient had been (not too carefuily) evaluated at an EW for symptoms of confusion only a few hours beforehand, but had been discharged. Although the clinical scenario was suggestive of anticholinergic intoxication, often associated with anticholinergic properties of some antidepressants, the EW physician failed to check a tricyclic antidepressant level. The patient's wife decides to sue the EW physician and the hospital for medical malpractice in the care of her husband. A legal element that constitutes a component of medical malpractice is:
a. The Hippocratic Oath
b. Apathy
c. Beneficence
d. Direct causation
e. Intention

38. You prescribe an SSRI-class antidepressant for a patient suffering from major depression requiring an inpatient psychiatric admission. Two days later the patient appears markedly agitated and is pacing the corridors. The nursing staff is very concerned, suggesting the patient may need additional sedation. On your exam the patient is agitated and pacing, bouncing her legs up and down during your interview, and she can't focus on the conversation. She notes that she doesn't know what is happening, but that she just can't sit still, feeling like she has to move her legs around. The most likely diagnosis is:
a. Neuroleptic malignant syndrome
b. Medication-induced akathisia
c. Generalized anxiety disorder (GAD)
d. Serotonin syndrome
e. Caffeine intoxication

39. A 19-year-old is diagnosed with OCD after several years of symptoms consisting of intrusive thoughts that he had harmed someone and chronic obsessions about cleanliness. He is nearly disabled by anxiety. During a family interview, his parents ask you if they did something wrong while raising their child. You explain that, contrary to older ideas, contemporary theories for the origins of OCD include genetic, neurochemical, and subtle brain injury (such as following mechanical brain injury and some forms of encephalitis). You explain that this condition is quite treatable with medications from the SSRI class of antidepressants and psychotherapy. The most useful form of psychotherapy for OCD is:
a. Interpersonal psychotherapy
b. Psychodynamic psychotherapy
c. Psychoanalysis
d. Cognitive-behavioral psychotherapy
e. Supportive psychotherapy

40. A 38-year-old man has a history of bipolar disorder that began at age 19 with a manic episode. Since then he has had several manic episodes and several depressive

episodes, but not more than four episodes per year. He made one unsuccessful suicide attempt by overdose at age 24 during a particularly severe depressive episode. The most appropriate maintenance therapy for this patient is:

a. Fluoxetine
b. Lamotrigine
c. Phenytoin
d. Lithium carbonate
e. Clonazepam

41. A 53-year-old male admitted to the inpatient psychiatric unit for treatment of bipolar disorder is found lying in the hallway. On exam, he is conscious, but is confused and dysarthric. The staff note that he has been progressively sedated and confused for the past 24 hours. He states: "I think the doctors are trying to kill me." Upon reviewing his chart, you note that he was admitted one week prior and restarted on his usual dose of carbamazepine due to a low level. Three days ago, he developed an upper respiratory infection and was treated with an antibiotic. His last carbamazepine level was 3 days ago. You conduct a thorough physical and review of systems to assure yourself that the patient did not suffer a syncopal episode and wasn't injured when he fell. The most likely diagnosis is:

a. Malingering
b. Dementia
c. Drug toxicity
d. Progressive upper respiratory infection
e. Normal pressure hydrocephalus

42. A 24-year-old woman who lives with her parents sees you for consultation because her psychotherapist believes that she may need a medication. She reports that since graduating from college and the death of her father 3 years ago she has became progressively despondent with low energy, poor appetite, poor concentration, and feelings of worthlessness. She has never had racing thoughts or other symptoms of mania and has a family history of unipolar depression. You diagnose her with major depression. Of the following, the best first-line treatment would be:

a. Buspirone therapy
b. Citalopram therapy
c. Phelezine therapy
d. Reserpine therapy
e. Lithium therapy

43. An 80-year-old man presents in your primary care clinic with his partner. His partner tells you that the man has become forgetful recently. He has been in excellent health all of his life but he has a family history of Alzheimer's dementia. His father developed Alzheimer's in his 70s and an aunt developed it in her 60s. After testing for other causes of dementia, you suspect Alzheimer's dementia. The best medication intervention would be:

a. Dopamine antagonist medications
b. Acetylcholinesterase inhibitor medications
c. Serotonin reuptake inhibitor medications
d. GABA antagonist medications
e. GABA agonist medications

44. A 22-year-old man who recently was fired from his job as an auto mechanic presents in the emergency room complaining of a conspiracy against him at his former workplace. When he first begins to explain the situation it sounds like possible work place harassment. You think about referring him to a lawyer when he informs you that his boss is an alien emperor from Jupiter and that his coworkers are the emperor's loyal subjects. When you ask him to describe the situation more, he coherently elaborates a richly detailed story about the aliens at the garage and how they conspired to eliminate him. Other than these unusual beliefs, he appears normal in terms of dress and body posture. The most likely subtype of schizophrenia in this patient is:

a. Paranoid
b. Catatonic
c. Disorganized
d. Undifferentiated
e. Residual

45. A 20-year-old male patient of yours with schizophrenia, paranoid type, stabs his friend in the abdomen with a knife because he believed that the man was Adolf Hitler reincarnated. His friend is severely injured but not killed. Your patient has been recurrently noncompliant with medications and upon examination shortly after the incident is found to be out of touch with reality. He tells you "I was trying to save the world from a tyrannical monster." At the trial, your patient pleads not guilty to charges of attempted murder by reason of insanity. The prosecutor agrees that the patient was mentally ill at the time of the event, but argues that the presence of mental illness alone is not sufficient for the insanity defense. The prosecutor is correct. The elements of the insanity defense that must exist in addition to the presence of a mental illness are:

a. Having taken medications for the mental illness
b. Committing the crime with a high likelihood of being caught
c. An inability to comprehend that the act was wrong
d. The presence of hallucinations or delusions
e. Being provoked by the victim

46. A 56-year-old man with a longstanding history of alcohol dependence is brought into the emergency room by

ambulance after having a series of seizures while at home. His wife said that her husband had been drinking daily but had recently thrown out all of the alcohol in the house and told her that he was stopping "cold turkey." You believe that the seizures may have been due to aicohol withdrawal. If the patient did have a withdrawal seizure, he most likely quit drinking how many hours prior to his admission?

a. 2–6
b. 6–12
c. 12–24
d. 24–72
e. 72–144

47. A 48-year-old woman with alcohol dependence presents in the emergency room after being found down outside her home in a semi-obtunded state. You learn from the emergency record that she has frequently presented in the past with alcohol intoxication. While evaluating her she exhibits waxing and waning disorientation and then suddenly becomes hypotensive. She is transferred to the ICU after being initially medically stabilized with a preliminary diagnosis of alcohol withdrawal delirium. After several days in the ICU, her delirium and autonomic instability resolve, but the nursing staff report that she is still "acting funny." On exam the patient is noted to have nystagmus, ataxia, and anterograde amnesia. She covers this memory deficit fairly well in casual conversation, but it is clear that she is confabulating during a great deal of the interview. The most likely diagnosis for this patient is:

a. Wernicke's encephalopathy
b. Korsakoff's psychosis
c. Wernicke-Korsakoff syndrome
d. Subdural hematoma
e. Epidural hematoma

48. A 23-year-old woman is brought to the EW by the police who say she was creating a disturbance in a local shopping mall. The woman is agitated upon arrival and speaking rapidly. She tells you that all the young men in the mall were laughing at her and that they all wanted to hurt her. After several minutes of discussion, she begins to act strangely towards you and asks you to leave her alone. A nurse tells you a few minutes after you leave her side that she thinks you are laughing at her too and that you might hurt her. She appears to have paranoia and you begin to try to determine if she has a primary mental disorder or if the condition is due to the effects of a substance or a general medical condition. If the condition is due to a substance, which of the following is most likely to produce these symptoms?

a. Amphetamines
b. Benzodiazepines
c. Opiates
d. Nicotine
e. Caffeine

49. A 57-year-old woman had a cerebrovascular accident two months ago that resulted in left upper extremity partial hemiparesis. Her son brought her to your clinic because he was concerned that she seemed lethargic for the past month. The patient reveals that she feels hopeless about her life situation and is having trouble with early morning awakening. Since the condition may have been precipitated by the CVA, the best course of action would be to:

a. Begin treatment with buspirone
b. Begin treatment with lorazepam
c. Begin treatment with sertraline
d. Begin treatment with lithium
e. Begin treatment with valproic acid

50. You are asked to see a 27-seven-year-old man who was admitted to the psychiatric unit in the hospital 2 days ago. The nurse reports to you that the patient has developed a hand tremor that looks like he is rolling a pill. He also tells you that the patient is walking very stiffly and appears to be having trouble initiating his movements. Which of the following medications would be most likely to have caused the condition above?

a. Lorazepam
b. Benztropine
c. Thioridazine
d. Clozapine
e. Haloperidol

A Answers

1. c

The patient has severe granulocytopenia and, likely, an opportunistic infection. Although many medications can cause granulocytopenia or agranulocytosis, clozapine carries the highest risk of all the antipsychotics for this major adverse drug reaction. It is also the only antipsychotic agent that requires frequent WBC monitoring.

2. b

This patient presents with a constellation of symptoms characteristic of depression: sad mood, anergia, anhedonia, poor concentration, and disturbances of sleep and appetite. The hypersomnia and increased appetite are consistent with the atypical subtype of major depression. The symptoms have been present for the minimum required 2 weeks. This constellation of symptoms is too severe and too sudden in onset to meet criteria for dysthymic disorder, which requires 2 years of low-level depressive symptoms. While a general medical condition could theoretically be the cause of any apparent depression, the presence of a precipitating stressor and the lack of evidence for hypothyroidism, a common cause of secondary depression, make this diagnosis unlikely. Substance-induced mood disorder should always be considered in first-episode depression, but the patient's pattern of moderate alcohol consumption is unlikely to be the primary etiology of her mood disorder. The fact that her father died 6 months prior to the onset of symptoms suggests that the loss may have been a precipitant, but this constellation of symptoms is more severe than normal bereavement.

3. d

While this patient may later be diagnosed with bipolar disorder, currently the presentation is of an apparent fluoxetine-induced manic episode. In order to meet the criteria for schizoaffective disorder, the patient would have to have had an episode of psychotic symptoms in the absence of a depressive or manic episode. While the psychotic symptoms that are present could be the onset of schizophrenia, the manic symptoms would generally not be present. Symptoms must also have been present for at least 6 months to meet criteria for schizophrenia. The episode described appears to be a sudden change in behavior, not a pervasive pattern of impulsive and self-destructive behavior that would characterize a person with borderline personality disorder.

4. d

The patient displays symptoms consistent with early dementia, including recent memory loss, anomia, and paranoia. Risk factors for Alzheimer's dementia (the most common cause of dementia) include advanced age, family history of dementia, Down syndrome, and prior head trauma. The patient has risk factors of advanced age and family history of dementia. HIV dementia is unlikely in the setting of no known risk factors and a negative HIV test. Potentially treatable causes of dementia such as vitamin deficiency, neurosyphilis, and normal pressure hydrocephalus are ruled out using the appropriate tests. A nonfocal neurological examination, no prior history of cardiovascular or cerebrovascular illness, and no CT evidence of infarction rule out vascular dementia (the second most common cause of dementia). Major depression can present with memory impairment; however, this patient has no other symptoms to support a diagnosis of depression. The patient's worries about her son appear to be paranoia, but psychosis, including paranoia, hallucinations, and delusions, is common in dementia.

5. b

People with this disorder yearn to be cared for and often require excessive support for everyday decision making. They tend to be described as "clingy" by others and often live in fear of abandonment by those on whom they depend. Compared to those with borderline personality disorder, they have a more stable sense of self and are less impulsive and self-destructive. This patient does not have the oddities of speech, thought, affect, and belief that characterize schizotypal personality disorder; nor is he an aloof, detached loner with schizoid personality disorder.

6. b

Dementia and delirium show a great deal of symptom overlap, and dementia is a risk factor for delirium. This patient has a newly developed delirium. The time course of delirium is abrupt in onset (hours to days), and is characterized by altered cognition, including disturbances in perception (hearing voices, misperceiving medical personnel as military personnel), altered attention (inability to spell *world* backwards), and altered level of arousal (alert to dozing in a brief time period). Hallucinations (such as hearing voices) are components of a psychotic disorder, but this patient's sensory misperceptions are not typical of psychosis. Major depression alone would not account for the patient's disturbed sensorium and cognitive clouding, particularly with such an acute time course. The clinical picture—absence of headache, nuchal rigidity, photophobia or other localizing signs—does not support the diagnosis of meningitis in this individual.

7. a

The Tarasoff case, decided by the California Supreme Court, established that the confidentiality of a psychotherapeutic session must be violated when an identified third party is at risk of imminent harm. In addition, this ruling established that the therapist must take reasonable steps to protect the potential victim from a patient's actions. Further, notification of the police in this case would also include an application for involuntary commitment of the patient to a psychiatric hospital. While the standards for involuntary commitment vary from state to state, they generally include that the patient must have a mental illness, and that the patient is at risk of self-harm or of harming others due to this mental illness.

8. e

The clinical evidence points to a history of chronic heavy alcohol use in this patient. The physical exam findings of palmar erythema and acne rosacea suggest long-standing alcohol ingestion; the enlarged tender liver suggests an alcohol-induced fatty or cirrhotic liver. The elevated vital signs, hyperreflexia, and dilated pupils all point to autonomic arousal produced by alcohol withdrawal. The CBC reveals evidence of alcohol-induced bone marrow suppression with a megaloblastic anemia due to folate deficiency. Additional metabolic findings common in alcohol-dependent individuals include hypomagnesemia and alcohol-induced transaminitis. This individual is delirious and has visual hallucinosis, indicating that he has developed the most severe complication of alcohol withdrawal, major withdrawal with alcohol withdrawal delirium (delirium tremens).

9. d

The failure to maintain more than 85% of ideal body weight combined with the distorted self-image makes the diagnosis anorexia nervosa. The lack of binge eating and/or purging further refines the diagnosis to the restricting type. Although patients with anorexia have a distorted body image, patients with body dysmorphic disorder do not have severe weight loss and anorexia.

10. d

The patient presents with classic symptoms of an anticholinergic delirium: dry mouth, mild fever, constipation, urinary retention, blurred vision, confusion, and agitation. The acute change in the patient's mental status is not consistent with a prior course of slowly progressive dementia. Pseudodementia is a term used to describe patients who develop apparent cognitive deficits in the setting of depression. Although a cerebral hemorrhage would likely cause an acute mental status change in an elderly person, the patient would likely have neurologic abnormalities and/or a history of head trauma.

11. a

There is a high rate of depression post-CVA; this patient exhibits low mood, anhedonia, feelings of guilt, poor appetite, and weight loss for more than the minimum required 2 weeks. The symptoms are severe enough and are not the long-standing low-level symptoms of dysthymic disorder. While she hasn't been visiting her grandchildren or returning her friend's phone calls, this avoidance is part of the depression, not an indication of an avoidant personality disorder or social phobia. She has realistic concerns about her apparent weight loss, unlike a person with somatization disorder who would generally have concerns about her body that have no basis in fact.

12. e

PTSD is characterized by witnessing or experiencing trauma with resultant symptoms of: (1) reexperiencing the trauma, (2) attempts to avoid recollections of the traumatic experience, and (3) persistent autonomic hyperarousal. Use of alcohol or other drugs to block the symptoms of PTSD is common and alcohol dependence is frequently diagnosed in these individuals. GAD is not associated with the experience of trauma. An individual with GAD might appear to suffer from autonomic hyperarousal, but does not have the experience of trauma, flashbacks, or avoidance present in PTSD. Major depression frequently co-occurs with PTSD, but the symptoms requisite to each diagnosis can be separated with a careful history. Complex partial epilepsy can present with seizure events that may mimic flashbacks, but the additional symptoms and history required to diagnose PTSD are not present.

13. a

A diagnosis of schizophrenia requires a greater than 6-month period of positive and negative symptoms of psychosis com-

bined with social and occupational deterioration. Bipolar disorder requires the presence of at least one episode of mania and usually consists of cycles of mania and depression. Psychosis is common in the manic phase of bipolar illness, but this patient does not display symptoms of mania (i.e., pressured speech, decreased sleep, increased energy, etc.). Major depression may be associated with psychosis, but this patient does not provide evidence of having major depression. Alcohol abuse can lead to psychosis, especially auditory hallucinosis, but other symptoms of alcohol abuse should be present. Paranoid personality disorder does not lead to the level of severe social and occupational dysfunction experienced by this patient.

14. e

This patient exhibits a pervasive pattern of flagrant disregard for the rules and laws of society. A childhood history of severe neglect or abuse is not uncommon. Despite the report of a childhood trauma, a diagnosis of PTSD is not made unless there is hyperarousal, intrusive recollection of the trauma, and efforts to avoid recollection of the trauma. His denial of mood disturbance and lack of prior hospitalizations makes both bipolar disorder and cyclothymic disorder unlikely. This patient may have been diagnosed with conduct disorder in childhood. When conduct disorder behavior persists into adulthood, the diagnosis becomes ASP.

15. d

A psychoactive substance abuse disorder is diagnosed when an individual has a substance-produced impaired ability to fulfill major roles, social or interpersonal problems, use of substances in physically hazardous circumstances, and recurrent legal problems related to substance use. Additional history from a friend or family member would be critical in this patient's case to verify that the history is accurate and to rule out alcohol dependence. Denial is a common defense mechanism in substance users; they frequently minimize or underreport the degree of substance intake. The history as obtained is insufficient to support an additional diagnosis of personality disorder. There is no evidence during the interview to support a diagnosis of alcohol intoxication (i.e., odor of alcohol, slurred speech, incoordination, impaired judgment, and so on).

16. e

Flumazenil acts as an antagonist at benzodiazepine receptors and can immediately reverse the effects of benzodiazepine intoxication. Naloxone is an opiate receptor antagonist and thiamine is a B vitamin; neither has any effect on benzodiazepine intoxication. Methohexital, a short-acting barbiturate, and diazepam, a benzodiazepine, would both worsen benzodiazepine intoxication.

17. c

This patient presents with chronic, low-level depressive symptoms of greater than 2 years duration. He does not meet criteria for a major depression. His active social life makes the diagnoses of avoidant personality disorder and schizoid personality disorder unlikely. While adjustment disorder with depressed mood is a possibility, the low-level depressive symptoms predate the apparent stressor (the divorce) and the patient denies that the divorce has significantly affected him.

18. a

This child displays the classic diagnostic criterion for autism, namely: (1) impaired social interactions; (2) impaired communication; and (3) stereotyped behavior or interests. The child does not display evidence of a formal thought disorder or ongoing psychosis, so would not meet criteria for schizophrenia. Conduct disorder is a behavioral disturbance where a child violates the basic rights of others and does not adhere to rules and societal norms. It is the childhood equivalent of ASP in adults. Attention deficit disorder is characterized by a persistent attention disturbance in at least two different settings (such as school and home). It may be accompanied by hyperactivity, in which circumstance attention-deficit/hyperactivity disorder (ADHD) would be diagnosed. Separation anxiety disorder is characterized by anxiety as evidenced by behavioral disturbance (such as crying) when separated from a primary caregiver.

19. c

The patient displays clinical evidence of having ingested a toxic level of a TCA. He has taken much more medication than prescribed and shows evidence of anticholinergic toxicity—dry mouth and slight confusion, tachycardia, and low-grade fever. TCAs cause QT prolongation and can produce ventricular tachycardia. Seizure, hypotension, and delirium are also common.

20. a

The distinguishing feature of sleep terror disorder as compared to nightmare disorder is that in the former, the person generally remains asleep and in nightmare disorder, the person generally awakens. Sleep terror disorder episodes occur during delta wave sleep; nightmare disorder episodes occur during REM sleep. Patients with narcolepsy experience daytime sleepiness that results in sleep attacks and cataplexy (sudden, reversible, bilateral loss of skeletal muscle tone). In breathing-related sleep disorder, abnormal breathing during sleep (e.g., apneic episodes from airway obstruction) leads to sleep disruption and daytime sleepiness. Sleepwalking disorder is a condition in which a person rises from the sleeping position and walks about or performs other complex motor activities while asleep.

21. c

This patient exhibits the classic symptoms of akathisia, a common side effect of antipsychotic medications. Patients with this side effect exhibit agitation, restlessness, and pacing. They usually endorse an inner feeling of restlessness (often localized to the legs). It is important to distinguish akathisia from restlessness and agitation due to worsening psychosis or anxiety. Other movement disorders, such as dystonia (muscle spasm), tardive dyskinesia (slow, involuntary muscle movements) or Parkinsonism (pill-rolling tremor, festinating gait, masked facies) should always be ruled out in patients taking neuroleptics. Patients with neuroleptic malignant syndrome can present with agitation or motor excitability, but the absence of fever and a normal serum CPK make this diagnosis unlikely.

22. d

Tourette's disorder is characterized by involuntary vocal and motor tics. Sometimes the vocal tics are grunts or barks, but they can develop into word tics. Tourette's disorder is more common in young males (3:1 male-female ratio) and onset is usually in late adolescence. Adolescent rebellion is an anecdotal term for teenage behavior but does not explain vocal and motor tics. Schizophrenia is not likely because this young man does not have psychotic symptoms. Substance abuse can produce a behavioral change in an individual, but vocal and motor tics are not classic. Conduct disorder is diagnosed when a child consistently violates the basic rights of others and ignores societal rules and norms. A child suspected of having Tourette's disorder should have a careful medical and neurological evaluation. Wilson's disease and Huntington's disease need to be ruled out, and an EEG should be performed to assess for seizure disorder.

23. d

The patient has sexual desire and is physiologically capable of arousal and orgasm. Although she is averse to genital contact, the contact is neither painful nor prevented by vaginal contraction as it would be in vaginismus. Sexual aversion is of unclear etiology. Many, though not all, patients report a childhood history of sexual abuse. Treatment is through couple's sex therapy.

24. c

Panic disorder typically occurs more often in females and has an onset in the early 20s. A person with myocardial ischemia might present with similar symptoms, but this disease would be unlikely in a patient with no cardiac risk factors and normal laboratory tests. GAD presents with chronic worry that pervades every aspect of life and psychomotor symptoms of restlessness, easy fatigue, impaired concentration, irritability, muscle tension, and sleep difficulty. Major depression may have associated symptoms of anxiety, but requires the presence of depressed mood or anhedonia in conjunction with neurovegetative symptoms. Alcohol intoxication may produce symptoms of agitation or behavioral dyscontrol, but alcohol withdrawal is most likely to produce anxiety. In addition, other physical signs of alcohol intoxication are usually present, and a urine drug screen would detect alcohol.

25. c

This young man has had 2 months' duration of depressed mood with neurovegetative symptoms of decreased energy, decreased interest in life, increased appetite with weight gain, disturbed sleep, and impaired concentration. An adjustment disorder is unlikely in an individual who reports no stressors, and would not be diagnosed if symptoms meet criteria for major depression even in the presence of a precipitating event. There is no evidence to suggest a general medical condition as causative of this patient's problems; however, this should always be considered. In this patient, thyroid testing and anemia and electrolyte screen rule out common endocrine, hematologic, and metabolic causes of fatigue. Dysthymic disorder requires that mood and neurovegetative symptoms are present for 2 years and that they be less severe than those required for major depression. The patient does not display psychotic symptoms consistent with schizophrenia.

26. d

Opiate-dependent patients, whether on methadone maintenance or other chronic opiates, will have developed tolerance to the effects of opiates added for pain relief. As a result, they will require a higher dose of pain-relieving opiate than a non-opiate-dependent patient. While drug-seeking behavior is common among individuals with substance abuse disorders, this patient displays objectively verifiable signs of pain-related sympathetic activation including diaphoresis and elevated vital signs. He also has a severe injury, likely to produce a great deal of pain. The patient may attempt to overuse the PCA pump, but will display signs of oversedation, euphoria, and vital sign depression, all of which can be objectively monitored.

27. c

This patient has a long history of alcoholism and displays amnesia for recent events. Although anxiety can interfere with memory, the patient does not appear anxious. Epidural hemorrhage might cause a declining level of consciousness or focal neurological defect in this patient but would not produce focal memory defects. Alzheimer's dementia is associated with memory loss, but requires additional cognitive impairments and is more common in an older age group. This patient is at risk for alcohol withdrawal delirium but does not show evidence of clouded sensorium or attention disturbance.

28. a

Since lithium is renally cleared, its serum level can be affected by states of dehydration and/or renal insufficiency. Signs of lithium toxicity, including coarse tremor, confusion, ataxia, and diarrhea, are evident in this patient. Although carbamazepine toxicity can similarly result in ataxia and confusion, it is not associated with coarse tremor or diarrhea. Carbamazepine levels are also not as sensitive to levels of dehydration. Fluoxetine can cause nausea, headache, restlessness, insomnia, and anorgasmia. It is generally nontoxic even in overdose. Haloperidol toxicity can cause stiffness, dystonia, akathisia, and at high serum levels, QT prolongation and possible torsade de pointes.

29. e

Among all the factors listed, hopelessness has been shown to be one of the most reliable indicators of long-term suicide risk. Major depression and other mood disorders and substance abuse disorders are also risk factors for suicide.

30. c

This patient presents with unusual visual hallucinations involving the distortion of objects in the visual field. This is a common finding in LSD intoxication. Rohypnol is a potent benzodiazepine that is not known to cause hallucinations. Alcohol can produce hallucinosis (usually auditory) during complicated alcohol withdrawal. This is unlikely in this case as the young boy has no known alcohol abuse history. Finally, heroin does not cause hallucinations.

31. e

Overdoses of opiates commonly produce profound respiratory depression. Benzodiazepine alone or alcohol alone are unlikely to produce this magnitude of respiratory depression but might do so if ingested in combination with other drugs. Cocaine does not produce respiratory depression. Ketamine is a dissociative anesthetic that produces little respiratory depression.

32. a

The patient displays obsessions with germs and cleanliness and compulsive washing behaviors to remove the germs. While the patient likely has anxiety relating to his obsessions, he does not display symptoms consistent with panic disorder. GAD can be ruled out due to the fact that his anxiety is not pervasive and that the patient engages in compulsive behaviors. Body dysmorphic disorder can be ruled out because the patient does not have a distorted body image. Narcissistic personality disorder can be ruled out as the patient does not exhibit arrogance coupled with low self-esteem.

33. e

Sexual orientation is a complex concept requiring much further study. The neural origins of sexual desire for members of a particular sex, race, age, body-type, etc., remain to be determined. Historically, many minority or unpopular behaviors, such as homosexuality, were pathologized by the medical community. With increasing scientific evidence that homosexual orientations were as normal as heterosexual orientations, homosexuality was removed from the DSM (Diagnostic and Statistical Manual of Mental Disorders) in 1973. Some psychotherapists believe "reparative" therapy can potentially convert an individual's homosexual orientation to heterosexual orientation. Because there is no scientific evidence to support this claim, and because reparative therapy is potentially damaging, the American Psychiatric Association recommends that ethical therapists refrain from attempts to change an individual's sexual orientation.

34. a

This patient presents with psychotic symptoms and agitation. Until further history is obtained or future episodes are observed, the diagnosis is uncertain. This could be a so-called "first break" of schizophreniform disorder that may persist for the required 6 months to meet the criteria for schizophrenia. It might be a manic episode with psychotic features in a patient with underlying bipolar disorder or schizoaffective disorder. The psychosis may also be substance-induced (e.g., amphetamine-induced psychotic disorder). Although patients with GAD can present with restlessness or irritability, they are usually not agitated and do not have auditory hallucinations or thought disorder. Patients with winter depression and atypical depression usually present with symptoms of decreased energy, increased appetite, and increased sleep. They also complain of psychomotor slowing. Individuals with OCD have unreasonable obsessions and compulsions.

35. e

Olanzapine is an atypical antipsychotic that is used in the treatment of psychosis of any etiology. Buspirone is indicated for GAD but is ineffective in psychosis. Sertraline is an SSRI that could be used in conjunction with an antipsychotic to treat a major depression with psychotic features but is ineffective alone in psychosis and might worsen a manic episode. Flumazenil is a benzodiazepine receptor antagonist that is used in benzodiazepine toxicity. Dextroamphetamine is a psychostimulant that would exacerbate psychotic symptoms, and may have been the causative agent in this patient's psychosis.

36. a

Haloperidol is the only neuroleptic or typical antipsychotic on this list. Neuroleptics carry a much higher risk of causing

tardive dyskinesia than do the newer, atypical antipsychotics such as risperidone, olanzapine, clozapine, and quetiapine.

37. d

The components of medical malpractice include duty, negligence, direct causation, and damages. All the other options are not part of the legal basis for medical malpractice. Namely, that there must have been a duty on the physician's part to provide proper care to the patient. There must have been negligence on the physician's part, in failing to meet the standard of care in such circumstances. Direct causation means that the physician's negligence must have been the direct cause of the alleged damages. Damages means that the patient must have suffered some bad outcome because of the physician's behavior. Beneficence refers to a component of ethical decision making.

38. b

Akathisia is most commonly caused by antipsychotic medications, particularly typical antipsychotics (neuroleptics). Akathisia produced by drugs in the SSRI class is less likely, but these medications are so widely used and are prescribed by so many non-psychiatric physicians that this important side effect may be misdiagnosed. Akathisia is profoundly distressing to some patients, and in some cases leads to attempted or successful suicide. It is easily treatable by removing the offending agent in most cases. Adding betablockers and benzodiazepines is also helpful.

39. d

Interpersonal psychotherapy is useful in treating depression, particularly when relationship issues are a key component of the patient's depression. Psychodynamic, psychoanalytic, and supportive psychotherapies have not been demonstrated to be as effective as cognitive-behavioral psychotherapy for the treatment of OCD, nor for many other conditions.

40. d

Despite the emergence of a variety of new mood stabilizers, lithium remains the most empirically grounded treatment for bipolar disorder without rapid cycling features. Fluoxetine, an antidepressant of the SSRI class, is generally contraindicated in individuals with bipolar disorder, as most antidepressants can increase the likelihood of a manic episode. Lamotrigine is an anti-seizure medication with mood stabilizing properties also. Lamotrigine requires further study, but early reports indicate that it may treat both the depressive and the manic phases of bipolar disorder. Phenytoin is an anti-seizure medication that has not been shown to be effective in treating bipolar disorder. Clonazepam is a long-acting benzodiazepine that may have useful properties in adjunctive treatment of acute mania, but is not an established treatment for maintenance therapy in bipolar disorder.

41. c

The best explanation for the patient's condition at the moment is drug toxicity due to elevated carbamazepine levels. The patient displays known symptoms consistent with carbamazepine toxicity. The clue to making the diagnosis rests with the sequence of events. The patient likely was noncompliant with his medications as an outpatient leading to reduced levels of mood stabilizer and a manic break. Upon admission, he was restarted at his usual outpatient doses, which presumably provide a therapeutic drug level when taken as scheduled. Then, an antibiotic is added. We are not provided with the name of the drug, but it is important to remember that some antibiotics, such as macrolide antibiotics, may inhibit the metabolism of medications metabolized by the liver's cytochrome p450 enzyme system.

42. b

Many medications can be used to treat depression. Neither buspirone nor reserpine would be used to treat depression. Buspirone is an anxiolytic that has little effect on depression. Reserpine depletes central adrenergic neurons of norepinephrine and can, in fact, induce depression. Lithium is often used in conjunction with antidepressants as an augmentation strategy in severe depression, but would not generally be a first-line treatment unless a bipolar depression is suspected. Citalopram and phenelzine are both effective antidepressants. However, phenelzine, a monoamine oxidase inhibitor with potentially dangerous side effects, is not a first-line treatment. Citalopram is safe, well-tolerated, and considered along with other SSRIs to be a first-line treatment for depression.

43. b

Memory impairment in Alzheimer's dementia is thought to be secondary to loss of basal forebrain cholinergic neurons. Acetylcholinesterase inhibitor medications including tacrine (Cognex) and donepezil (Aricept) increase synaptic acetylcholine concentration. Anticholinergic medications (such as low-potency antipsychotics and diphenhydramine [Benadryl]) should be avoided in individuals with dementia. Dopamine antagonist medications are frequently used for treating psychotic symptoms (such as paranoia, hallucinations, and agitation) in dementia. Serotonin reuptake inhibitors are used to treat depression and are frequently used in depression co-occurring with dementia. GABA antagonist medications (e.g., flumazenil) are used in the treatment of acute benzodiazepine overdose; GABA agonist medications (e.g., benzodiazepines, barbiturates) may be used for agitation in dementia but may worsen memory difficulties.

44. a

Catatonic schizophrenia requires the presence of catatonic symptoms (motor and vocal changes). Disorganized schizophrenia requires the presence of disorganized speech and behavior and inappropriate affect. Undifferentiated schizophrenia is diagnosed when criteria are not met for another subtype. Residual schizophrenia is diagnosed when a patient who formerly had prominent positive symptoms of schizophrenia now has only residual negative symptoms or minor positive symptoms.

45. c

Taking medications for a mental illness, taking a risk of being caught, and having hallucinations or delusions are factors associated with the patient's condition but are not part of the insanity defense. Being provoked by the victim is not a component of the insanity defense. The insanity defense is also known as the M'Naghten rule (named for a mentally ill man who attempted to assassinate a British prime minister).

46. d

Minor alcohol withdrawal begins as early as a few hours after the last drink. Major alcohol withdrawal begins somewhat later. Alcohol withdrawal seizures occur between 6 and 48 hours after cessation of alcohol intake. Alcoholic hallucinosis (consisting primarily of auditory hallucinations with a clear sensorium) occur within 48 hours after cessation of alcohol intake. Alcohol withdrawal delirium is characterized by confusion and hallucinations, autonomic hyperarousal, and mild fever, and has onset 48 to 72 hours after the last alcohol intake.

47. c

Wernicke-Korsakoff syndrome is a serious and usually irreversible complication of heavy alcohol use caused by thiamine deficiency with associated hemorrhagic infarction in the mammillary bodies. Wernicke's encephalopathy is the early stage of this condition and consists of confusion, nystagmus, and ataxia. Prompt treatment with thiamine repletion (intramuscular or intravenous) may prevent a progression to Korsakoff's psychosis (anterograde amnesia and confabulation). Once the full Wernicke-Korsakoff syndrome is established, it is irreversible in about two thirds of patients.

48. a

Paranoia and other schizophrenic symptomology are thought to be strongly related to dopamine function in the brain. In addition to other effects, amphetamines have potent effects at dopaminergic synapses. Opiates and benzodiazepines rarely produce paranoid symptoms during use, but may produce psychotic symptoms during withdrawal. Nicotine and caffeine are psychomotor stimulants but rarely produce paranoia.

49. c

An antidepressant is the most appropriate treatment for major depression even if it may have been caused by a CVA. Lithium would only be used in this case for augmentation of an antidepressant in the case of a partial response. Valproic acid would be used if bipolar disorder or a seizure disorder were suspected. Buspirone and lorazepam might be used adjunctively with sertraline for anxiety but would not generally be first-line treatment.

50. e

The patient has developed pseudo-parkinsonism, a drug-induced condition resembling Parkinson's disease. The "pill rolling" tremor, festinating gait, and difficulty initiating movements are all symptoms of Parkinson's. Since the disease is due to low cerebral dopamine levels, drugs that block dopamine receptors are most likely to mimic the disease. Neither lorazepam or benztropine block dopamine receptors. Although thioridazine and clozapine both block dopamine receptors, they are low potency antipsychotics that only weakly block dopamine receptors and are less likely to cause pseudo-parkinsonism. Haloperidol, a potent dopamine blocker, is most likely to cause the syndrome.

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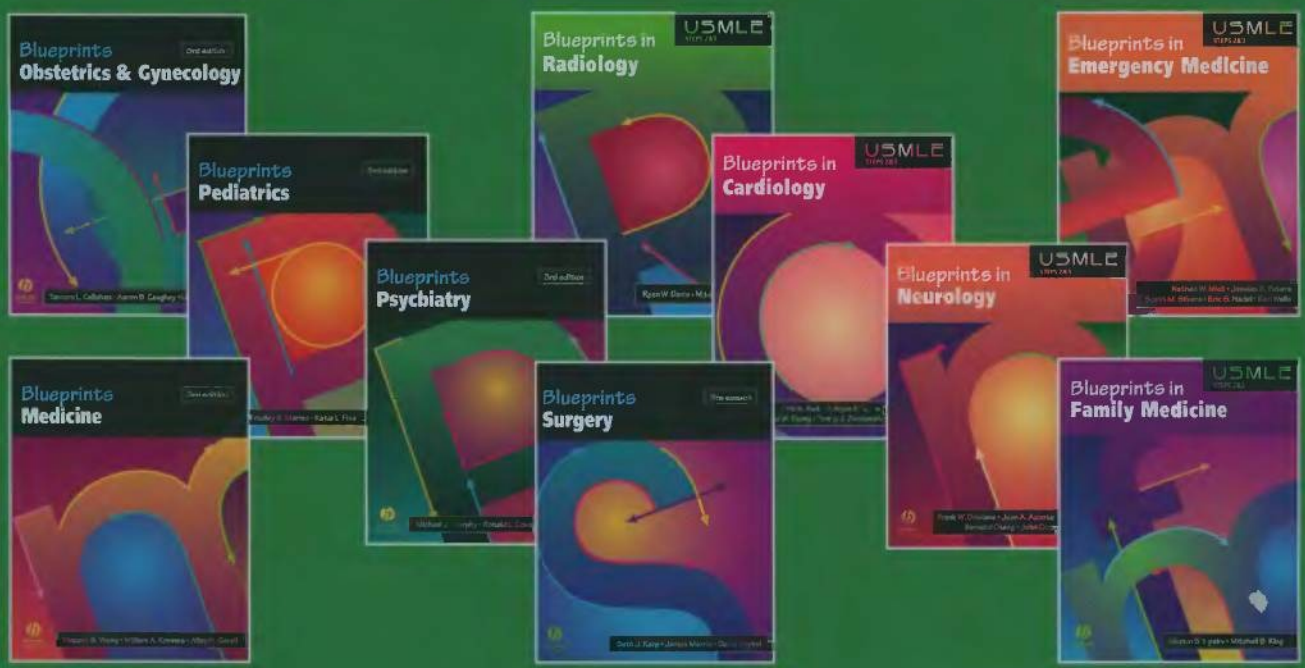

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