CHAPTER 12

Mood Disorders: Depression
Elizabeth M. Varcarolis

Key Terms and Concepts

- anergia, p. 000
- anhedonia, p. 000
- dysthymic disorder (DD), p. 000
- hypersomnia, p. 000
- light therapy, p. 000
- major depressive disorder (MDD), p. 000
- mood, p. 000
- novel antidepressants, p. 000
- psychomotor agitation, p. 000
- psychomotor retardation, p. 000
- selective serotonin reuptake inhibitors (SSRIs), p. 000
- St. John’s wort, p. 000
- transcranial magnetic stimulation (TMS), p. 000
- tricyclic antidepressants (TCAs), p. 000
- vegetative signs of depression, p. 000

Objectives

1. Compare and contrast major depressive disorder (MDD) with dysthymia.
2. Discuss the links between the stress model of depression and the biological model of depression.
3. Be able to assess behaviors in a depressed individual with regard to each of the following areas: (a) affect, (b) thought processes, (c) feelings, (d) physical characteristics, and (e) communication.
4. Discuss some communication strategies that are useful for depressed patients.
5. Evaluate the advantages of the selective serotonin reuptake inhibitors (SSRIs) over the tricyclic antidepressants.
6. Explain the unique attributes of two of the newer atypical antidepressants for use in specific circumstances.
7. Explain why special dietary/medication restrictions have to be maintained with the use of a monoamine oxidase inhibitor (MAOI).
8. Discuss why the selegiline transdermal system (STS) is such a breakthrough MAOI.
9. Identify potential adverse reactions to the SSRIs.
10. Describe the types of depression for which electroconvulsive therapy is most helpful.

For additional resources related to the content of this chapter, visit the Evolve website at http://evolve.elsevier.com/Varcarolis/essentials.

- Chapter Outline
- Rationales and Page References for Chapter Review Questions
- Additional Chapter Review Questions
- Case Study and Nursing Care Plans
- Concept Map Creator
- Nurse, Patient, and Family Resources
No amount of information can adequately convey the personal pain and suffering experienced by the individual with depression (Young et al., 2001). All races, all ages, and males as well as females are susceptible to depressive episodes, although some individuals are more susceptible than others.

PREVALENCE AND COMORBIDITY

Depression is the fourth leading cause of disability in the United States, and it is projected to be the second leading cause of disability by 2020 (Montano, 2003). The lifetime prevalence of a major depressive episode is 16.6%, with a projected lifetime risk at age 75 being 23.2% (Kessler et al., 2005). Lifetime prevalence for females is 21% (Oquendo & Liebowitz, 2005).

Most studies find that major depressive disorder (MDD) is twice as common in women (12.0%) than in men (6.6%). Dysthymic disorder (DD) (chronic mild depression) occurs in about 2.5% of the population over a lifetime (Kessler et al., 2005). About 40% of people with DD also meet the criteria for MDD or bipolar disorder (BD) in any given year (NIMH, 2001b).

A depressive syndrome frequently accompanies other psychiatric disorders such as anxiety disorders, schizophrenia, substance abuse, eating disorders, and schizoaffective disorder. People with anxiety disorders (e.g., panic disorder, generalized anxiety disorder, obsessive-compulsive disorder) commonly present with depression, as do people with personality disorders (particularly borderline personality disorder), adjustment disorder, and brief depressive reactions.

Mixed anxiety–depression is perhaps one of the most common psychiatric presentations. Symptoms of anxiety occur in an average of 70% of cases of major depression. The presence of comorbid anxiety disorder and depression has a negative effect on the disease course. Comorbidity has been shown to result in a higher rate of suicide, greater severity of depression, greater impairment in social and occupational functioning, and poorer response to treatment (Simon & Rosenbaum, 2003).

The incidence of major depression greatly increases among people with a medical disorder. People with chronic medical problems are at a higher risk for depression than those in the general population. Depression is often secondary to a medical condition. Depression also may be secondary to use of substances such as alcohol, cocaine, marijuana, heroin, and even anxiolytics and other prescription medications. Depression also can be a sequela of bereavement and grief (Chapter 22).

Children and Adolescents

Children as young as 3 years of age have been diagnosed with depression. MDD is said to occur in as many as 18% of preadolescents, which is perhaps a low estimate because depression in this age-group is often underdiagnosed. Children at 10 years have a lifetime prevalence of 14%, and by age 25, a person has a 19% chance of having an MDD (Kessler et al., 2005). Girls 15 years and older are twice as likely to experience a major depressive episode as boys (NIMH, 2000a). Major depression among adolescents is often associated with substance abuse and antisocial behavior, both of which can obscure accurate diagnosis (Dubovsky et al., 2004).

MDD in children has a high recurrence rate of up to 70% within 5 years. The appearance of MDD in adolescence heralds a severe disorder with a recurrent course (Gruenberg & Goldstein, 2003). Children in families with other depressed members seem to become depressed earlier (ages 12 to 13 years) than children in families with no other depressed members (16 to 17 years). Even before adolescence, girls are more vulnerable to depression than boys.

Older Adults

Depression among older adults (65 or older) is approximately 6% to 9% for major depression and 17% to 18% for minor depression. In fact, a disproportionate number of depressed older Americans are likely to die by suicide, accounting for 25% of all suicides, and the incidence appears to increase with age (Rosenbaum & Covino, 2006). The symptoms of geriatric depression often go unrecognized because many masquerade as medical symptoms. Depression in older adults is often associated with chronic illnesses and depression can go undiagnosed 50% of the time. The good news is that efforts to improve recognition of depression and education have led to positive treatment response among older adults.

THEORY

Although many theories attempt to explain the cause of depression, the many psychological, biological, and cultural variables make identification of any one cause difficult. It is unlikely that there is a single cause for depression. It is becoming evident that depression is a heterogeneous, systemic illness involving an array of different neurotransmitters, neurohormones, and neuronal pathways. The idea that depression is the result of a simple hereditary process or traumatic life event that ultimately leads to a single neurotransmitter deficiency is simply unsubstantiated by the evidence (Dubovsky et al., 2004).
EXAMINING THE EVIDENCE  Depression

If depression is a real illness with chemical causation (like diabetes), how does talking help a person get better? After all, I don’t think it would be very helpful to try to talk a person’s blood sugar down.

Depression is caused by a combination of neural vulnerability and environmental factors. Some people may possess highly vulnerable chemistry and spontaneously or with little provocation develop depression, whereas others who are not genetically predisposed to depression may develop it only when exposed to sufficient stress. Considering the role of a hostile environment in the development of depression in many people, it stands to reason that a therapeutic environment would be helpful in alleviating depression.

What do we know? Let’s start with sea slugs, creatures whose brains have been well mapped out. Dr. Eric Kandel, a neurobiologist and psychiatrist, is famous for studying the slugs’ brains and demonstrating that learning actually remodels neurons (Friedman, 2002). The implication is that if psychotherapy is a form of learning, then it causes changes to brain structure and function.

Researchers have been hard at work to objectively measure, mainly through neuroimaging, the effects of psychotherapy. Roffman and colleagues (2005) critically reviewed investigations of the effect of psychotherapy on brain function. They found that cognitive behavioral therapy and interpersonal therapy resulted in cortical-subcortical changes; these changes also occur with antidepressant medications. Another group of researchers, Etkin and colleagues (2005), also conducted an integrated literature review with similar results.

What is interesting is that the imaging changes associated with psychotherapy occur mainly in different areas (with some overlap) than changes associated with psychopharmacological treatments. In fact, researchers have found that people respond best when they receive a combination of talking and medication treatment (Schram et al., 2007).


The high variability in symptoms, response to treatment, and course of the illness supports the supposition that depression may result from a complex interaction of causes. For example, genetic predisposition to the illness combined with childhood stress may lead to significant changes in the central nervous system (CNS) that result in depression. However, there are common risk factors for depression that may signal the presence of this common and serious psychiatric illness (Gruenberg & Goldstein, 2003) (Box 12-1).

BOX 12-1

Primary Risk Factors for Depression

- History of prior episodes of depression
- Family history of depressive disorder, especially in first-degree relatives
- History of suicide attempts or family history of suicide
- Female gender
- Age 40 years or younger
- Postpartum period
- Medical illness
- Absence of social support
- Negative, stressful life events
- Active alcohol or substance abuse
- History of sexual abuse


Biological Theories

Genetic Factors

Twin studies consistently show that genetic factors play a role in the development of depressive disorders. Various studies reveal that the average concordance rate for unipolar depression mood disorders among monozygotic twins (twins sharing the same genetic constitution) is 50%. That is, if one twin is affected, the second has a 50% chance of being affected. The percentage for dizygotic twins (different genetic complement) is 20%. Thus identical twins (mono-
Adoptive studies also have pointed to genetic contribution for the development of depression. For example, children born to a parent or parents with a depressive illness have the same risk of depression if adopted to a nondepressive family as those children who are not adopted away (Sadock & Sadock, 2007). Therefore mood disorders are heritable for some people. Increased heritability is associated with an earlier age of onset, greater rate of comorbidity, and increased risk of recurrent illness. However, any genetic factors that are present must interact with environmental factors for depression to develop.

Biochemical Factors
The brain is a highly complex organ that contains billions of neurons. There is much evidence to support the concept that depression is a biologically heterogeneous disorder; that is, many CNS neurotransmitter abnormalities can probably cause clinical depression. These neurotransmitter abnormalities may be the result of inherited or environmental factors, or even of other medical conditions, such as cerebral infarction, hypothyroidism, acquired immunodeficiency syndrome, or drug use. Whatever the etiologic contributions of depression for an individual might be, depression is ultimately mediated through changes in the brain’s neurochemistry and the circuitry involved in emotional regulations (Gelder et al., 2006).

Neurobiological investigations in depression have focused on the monoamine neurotransmitters (serotonin, noradrenaline, and dopamine). Specific neurotransmitters in the brain are believed to be related to altered mood states. 

Serotonin (5-hydroxytryptamine or 5-HT) and norepinephrine are two major neurotransmitters involved in depression. Serotonin is an important regulator of sleep, appetite, and libido. A serotonin circuit dysfunction can result in poor impulse control, low sex drive, decreased appetite, and irritability (Sadek & Nemeroff, 2000). Decreased levels of norepinephrine in the medial forebrain bundle may account for anergia (reduction in or lack of energy), anhedonia (an inability to find meaning or pleasure in existence), decreased concentration, and diminished libido in depression.

Serotonin and norepinephrine are also involved in the perception of pain by modifying the effects of substance P, glutamate, γ-aminobutyric acid (GABA), and other pain mediators (Montano, 2003). In fact, one study demonstrated that 43% of people with major depression had at least one chronic painful condition. This was four times the rate in those without MDD (Montano, 2003). There is considerable evidence of overlap in the physiology of pain and mood disorders (Kramer, 2004).

Current research suggests that depression results from the dysregulation of a number of other neurotransmitter systems in addition to serotonin and norepinephrine. The dopamine, acetylcholine, and GABA systems are believed to be involved in the pathophysiology of a major depressive episode (APA, 2000). Dopamine neurons in the mesolimbic system are thought to play a role in the reward and incentive behavior processes that are disrupted in depression. This is particularly true in melancholic states (severe MDD) (Gelder et al., 2006). It is now considered unlikely that a catecholamine deficiency alone is the actual cause of depression (Gruenberg & Goldstein, 2003).

It is important to keep in mind that the neurotransmitters specific to depression (norepinephrine, serotonin, and dopamine) each have many subtypes, thus the complexity in treatment and varied patient responses to attempts to increase these neurotransmitters through medications.

Stressful life events, especially losses, seem to be a significant factor in the development of depression. According to Gelder and colleagues (2006), research has shown the following:

- There is a sixfold excess of adverse life events in the months before the onset of a depressive disorder.
- “Loss” events are associated more with depression, whereas “threat” events seem to be associated more with anxiety.
- The importance of stressful adverse life events in the onset of depression decreases once depressive disorder is clearly established. Then depressive episodes continue in the absence of adverse stressful life events.

Norepinephrine, serotonin, and acetylcholine play a role in stress regulation. When these neurotransmitters become overtaxed through stressful events, neurotransmitter depletion may occur. There is evidence that people who possess a “short” gene, or stress-sensitive version of the serotonin transporter gene, are at a higher risk of depression if they have been abused as children or if they have been exposed to multiple stressful life events. However, people with the “long” or protected version of the gene who underwent multiple life stressors experienced no more depression than people who were totally spared life stressors (NIMH, 2003a).

No unitary mechanism of depressant action has been found. The relationships among the serotonin, norepinephrine, dopamine, acetylcholine, and GABA systems are complex and need further assessment and study. However, medication that helps regulate these neurotransmitters has proved empirically successful in the treatment of many patients. Figure 12-1 shows a positron emission tomogra-
Neuroendocrine Factors
The neuroendocrine characteristic most widely studied in relation to depression has been hyperactivity of the hypothalamic-pituitary-adrenal cortical axis. Evidence of increased cortisol secretion is apparent in 20% to 40% of depressed outpatients and 40% to 60% of depressed inpatients. This increase was once thought to be the result of depression, but research indicates that excess cortisol may in fact be causative (Ardayfio & Kim, 2006).

The dexamethasone suppression test (DST) is used to determine if cortisol is being inhibited properly by adrenocorticotropic hormone (ACTH) feedback. Dexamethasone, a synthetic steroid, is administered, and in about 50% of people with depression, it fails to suppress serum cortisol. Significantly, patients with severe and psychotic unipolar MDD have rates of nonsuppression of 80% to 90% (Dubovsky et al., 2004).

Image Findings
Computed axial tomography (CAT) and magnetic resonance imaging (MRI) scans show ventricular enlargement, cortical atrophy, and sulcal widening in many studies (Sadock & Sadock, 2007). Other areas that have shown consistent changes are decreased size of the caudate, putamen, and possibly the cerebellum (Dubovsky et al., 2004). PET scans have repeatedly revealed reduced metabolic activity in the frontal lobes (Sadock & Sadock, 2007).

Psychodynamic Influences and Life Events
The stress-diathesis model of depression is a psychological theory that explains depression from an environmental and life-events perspective (stress) combined with biological vulnerability or predisposition (diathesis). What is certain is that psychosocial stressors and interpersonal events trigger certain neurophysical and neurochemical changes in the brain (NIMH, 2002). Early life trauma may result in long-term hyperactivity of the CNS corticotropin-releasing factor (CRF) and norepinephrine systems with a consequent neurotoxic effect on the hippocampus that leads to neuronal loss. These changes could cause sensitization of the CRF circuits to even mild stress in adulthood, leading to an exaggerated stress response (Heim & Nemeroff, 1999). With exposure to repeated stress in adulthood, these already stress-sensitive pathways become “markedly hyperactive leading to a persistent increase in CRF and cortisol secretion, which causes alterations in the glucocorticoid receptors and thus forms the basis for the development of mood and anxiety disorders” (Sadek & Nemeroff, 2000).
These stressors and life events may lead to a depressive syndrome in some individuals, particularly those who are biologically vulnerable to depression, such as those with the short or stress-sensitive version of the serotonin transporter gene mentioned earlier. Therefore life events (psychosocial stressors and interpersonal events) may influence the development and recurrence of depression through the psychological and biological experience of stress in some people, which results in changes in the connections among nerve cells in the brain.

Cognitive Theory

Aaron T. Beck, one of the early proponents of cognitive therapy, applied cognitive-behavioral theory to depression. Beck proposed that people acquire a psychological predisposition to depression through early life experiences. These experiences contribute to negative, illogical, and irrational thought processes that may remain dormant until they are activated during times of stress (Beck & Rush, 1995).

Beck found that depressed people process information in negative ways, even in the midst of positive factors that affect the person’s life. Beck believed that three automatic negative thoughts—called Beck’s cognitive triad—are responsible for the development of depression:
1. A negative, self-deprecating view of self: “I really never do anything well; everyone else seems smarter.”
2. A pessimistic view of the world: “Once you’re down, you can’t get up. Look around, poverty, homelessness, sickness, war, and despair are every place you look.”
3. The belief that negative reinforcement (or no validation for the self) will continue: “It doesn’t matter what you do; nothing ever gets better. I’ll be in this stupid job the rest of my life.”

The phrase automatic negative thoughts refers to thoughts that are repetitive, unintended, and not readily controllable. This cognitive triad seems to be consistent in all types of depression, regardless of clinical subtype.

The goal of cognitive-behavioral therapy (CBT) is to change the way a patient thinks, which will in turn help relieve the depressive syndrome. This is accomplished by assisting the patient in the following:
1. Identifying and testing negative cognition
2. Developing alternative thinking patterns
3. Rehearsing new cognitive and behavioral responses

Learned Helplessness

One of the most popular theories of the cause of depression is Martin Seligman’s theory of learned helplessness. Seligman (1973) stated that although anxiety is the initial response to a stressful situation, anxiety is replaced by depression if the person feels no control over the outcome of a situation. A person who believes that an undesired event is his or her fault and that nothing can be done to change it is prone to depression. The theory of learned helplessness has been used to explain the development of depression in certain social groups, such as older adults, people living in impoverished areas, and women.

CULTURAL CONSIDERATIONS

According to the Cross-National Collaborative Group, the prevalence rate of depressive disorders in Asian Americans was the lowest in comparison to whites, African Americans, and Hispanics (Rihmer & Angst, 2005). Prevalence rates for MDD in whites are significantly higher than in African Americans and Mexican Americans, although the opposite is true for DD (Riolo et al., 2005).

CLINICAL PICTURE

Figure 12-2 presents diagnostic criteria for MDD and DD, the two depressive disorders defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (APA, 2000a). Depression can be manifested in a variety of other symptoms that are called specifiers. Other subgroups are being researched as well (Table 12-1).

Major Depressive Disorder

Patients with a major depressive disorder (MDD) experience substantial pain and suffering, as well as psychological, social, and occupational disability. A patient with MDD presents with a history of one or more major depressive episodes and no history of manic or hypomanic episodes. In MDD, the symptoms often interfere with the person’s social or occupational functioning and in some cases may include psychotic features. Psychotic major depression is a severe form of mood disorder that is characterized by delusions or hallucinations. For example, patients might have delusional thoughts that interfere with their nutritional status (e.g., “God put snakes in my stomach and told me not to eat.”).

The emotional, cognitive, physical, and behavioral symptoms an individual exhibits during a major depressive episode represent a change in the person’s usual functioning.

The course of MDD is variable. An average episode may last about 9 months, although it has been shown that 20% of individuals will not have recovered by that time. Long-term studies indicate that after 5 years there is a 70% recurrence rate, and an 80% recurrence rate after 8 years (Psychdirect.com, 2006).
**DSM-IV-TR CRITERIA FOR DEPRESSIVE DISORDERS**

**DEPRESSIVE DISORDERS**

**Major Depressive Disorder**

1. Represents a change in previous functions.
2. Symptoms cause clinically significant distress or impair social, occupational, or other important areas of functioning.
3. **Five or more** of the following occur nearly every day for most waking hours over the same 2-week period:
   - Depressed mood most of day, nearly every day
   - Anhedonia
   - Significant weight loss or gain (more than 5% of body weight in 1 month)
   - Insomnia or hypersomnia
   - Increased or decreased motor activity
   - Anergia (fatigue or loss of energy)
   - Feelings of worthlessness or inappropriate guilt (may be delusional)
   - Decreased concentration or indecisiveness
   - Recurrent thoughts of death or suicidal ideation (with or without plan)

**Dysthymia**

1. Occurs over a 2-year period (1 year for children and adolescents), depressed mood.
2. Symptoms cause clinically significant distress in social, occupational, and other important areas of functioning.
3. **Two or more** of the following are present:
   - Decreased or increased appetite
   - Insomnia or hypersomnia
   - Low energy or chronic fatigue
   - Decreased self-esteem
   - Poor concentration or difficulty making decisions
   - Feelings of hopelessness or despair

**Specifiers Describing Most Recent Episode**

1. Chronic
2. Atypical features
3. Catatonic features
4. Melancholic features
5. Postpartum onset

**Specify If**

1. Early onset (before 21 years of age)
2. Late onset (21 years of age or older)
3. Atypical features

---

**VIGNETTE**

Sally, a bright, successful, 24-year-old businessperson, finds her world is changing. Over the past few weeks she has become more and more withdrawn. Her life has become empty of meaning. She has great difficulty getting out of bed in the mornings, but finds it hard to sleep more than 3 to 4 hours a night, waking at 2 or 3 AM. She is constantly exhausted.

Sally finds it impossible to concentrate at work and has called in sick the past 2 days, unable to find the energy to dress, bathe, groom, or even eat. She hasn’t eaten for 3 days except for some water and a few glasses of milk and a few crackers she found in a neglected box tucked away in the pantry. She has lost considerable weight.

continued
### TABLE 12-1 Depressive Disorders: Specifiers and Clinical Phenomena

<table>
<thead>
<tr>
<th>Disorder</th>
<th>DSM-IV-TR Status</th>
<th>Symptoms and Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major depressive disorder (MDD)</td>
<td>Disorder</td>
<td>Specific DSM-IV-TR criteria are outlined in Figure 12-2. Symptoms represent a change from usual functioning. Associated with high mortality rate. Impairment in physical, social, and role functioning, as well as increased potential for pain and physical illness.</td>
</tr>
<tr>
<td>With psychotic features</td>
<td>Specifier</td>
<td>Indicates the presence of delusions (e.g., delusions of guilt or being punished for sins, somatic delusions of horrible disease or body rotting, delusions of poverty or going bankrupt), or hallucinations (usually auditory, voices berating person for sins or shortcoming).</td>
</tr>
<tr>
<td>With postpartum onset</td>
<td>Specifier</td>
<td>Indicates onset within 4 weeks after childbirth. Can present with or without psychotic features. <strong>Severe ruminations or delusional thoughts about infant signify increased risk of harm to infant.</strong> Indicates that episodes mostly begin in fall or winter and remit in spring. Characterized by anergia, hypervigilance, weight gain, and a craving for carbohydrates. Responds to light therapy. Indicates MDD lasting 2 years or longer.</td>
</tr>
<tr>
<td>With seasonal characteristics; seasonal affective disorder (SAD)</td>
<td>Specifier</td>
<td>Specific DSM-IV-TR criteria are presented in Figure 12-2. Has an early and insidious onset (childhood to early adulthood). Shows a chronic course. Some 75% of people with DD go on to develop MDD. When dysthymia is superimposed on a major depression, it is called double depression.</td>
</tr>
<tr>
<td>With chronic features</td>
<td>Specifier</td>
<td>Indicates mood reactivity (can be cheered with positive events) and rejection sensitivity (pathological sensitivity to perceived interpersonal rejection) that are present through life and result in functional impairment. Other symptoms include hypervigilance, difficulty concentrating, fatigue, low self-esteem, irritability, and more, all causing significant distress or impairment in functioning.</td>
</tr>
<tr>
<td>Dysthymic disorder (DD)</td>
<td>Disorder</td>
<td>Prevalence of 5%. Characterized by significant functional disability. Criteria include at least 1 month of persistent dysphoric mood, with possible hypervigilance, difficulty concentrating, fatigue, low self-esteem, irritability, and more, all causing significant distress or impairment in functioning.</td>
</tr>
<tr>
<td>With atypical features</td>
<td>Specifier</td>
<td>Meets criteria for depressive episode, but episodes last 1 day to 1 week. Depressive episode must recur at least once per month over 12 months or more. <strong>Carries a high risk for suicide.</strong> Characterized by more severe symptoms than premenstrual syndrome. Symptoms begin toward last week of luteal phase and are absent in the week following menses. Symptoms include anergia, fatigue, irritability, and more. <strong>Other symptoms include anergia, oversleeping, difficulty concentrating, feeling of being out of control or overwhelmed, and more.</strong></td>
</tr>
<tr>
<td>Mixed anxiety–depression</td>
<td>RDC</td>
<td>Characterized by sustained depressed mood without the full depressive syndrome. Pessimistic attitude and self-pity are required for the diagnosis (Dubovsky &amp; Buzan, 1999). May be chronic and may be complicated by a superimposed major depressive episode.</td>
</tr>
<tr>
<td>Recurrent brief depression</td>
<td>RDC</td>
<td>Characterized by more severe symptoms than premenstrual syndrome. Symptoms begin toward last week of luteal phase and are absent in the week following menses. Symptoms include anergia, fatigue, irritability, and more. <strong>Other symptoms include anergia, oversleeping, difficulty concentrating, feeling of being out of control or overwhelmed, and more.</strong></td>
</tr>
</tbody>
</table>

*RDC, Research diagnostic category.*
UNIT THREE  ■  ■  ■ Caring for Patients with Psychobiological Disorders

Subtypes Seen in MDD
The diagnosis for MDD may include a specifier in patients with specific symptoms. Specifiers include the following:

- **Psychotic feature**: breaks with reality (e.g., hallucinations, delusions)
- **Catatonic features**: such as peculiar voluntary movement, echopraxia or echolalia, and negativism
- **Melancholic features**: such as anorexia or weight loss, diurnal variations with symptoms worse in the morning, early morning awakening
- **Postpartum onset**: within 4 weeks postpartum (e.g., severe anxiety, possible psychotic features)
- **Seasonal features (seasonal affective disorder, or SAD)**: for example, generally occurring in fall or winter and remitting in the spring
- **Atypical features**: such as appetite changes or weight gain, hypersomnia, extreme sensitivity to perceived interpersonal rejection, high levels of anxiety

Dysthymia
Dysthymic disorder (DD) often has an early and insidious onset and is characterized by a chronic depressive syndrome that is usually present for most of the day, more days than not, for at least 2 years (APA, 2000b). The depressive mood disturbance, because of its chronic nature, cannot be distinguished from the person’s usual pattern of functioning (“I’ve always been this way”) (APA, 2000b). Although people with dysthymia suffer from social and occupational distress, it is not usually severe enough to warrant hospitalization unless the person becomes suicidal. The age of onset is usually from early childhood and teenage years to early adulthood. Patients with DD are at risk for developing MDD as well as other psychiatric disorders. This may be referred to as double depression.

Differentiating MDD from DD can be difficult because the disorders have similar symptoms. The main differences are in the severity of the symptoms; a DD is much less severe than an episode of MDD.

VIGNETTE
Sam has had another bad week at work. He just can’t seem to perform the way he thinks he should—he never gets things right. Although his work seems acceptable to others, he constantly puts himself down. He wanted to take a class to improve his computer skills, but can’t seem to find the energy or the time. His weekends are filled with hanging around his apartment, nothing much going on . . . there is never much going on. Life is dull; has it ever been otherwise? His brother is always telling him that he has a face as long as a football field. “What’s the matter with you, bro? You’re good looking, smart. Go find a girl and have some fun in life. Why can’t you just enjoy anything?” Sam just sighs. Who could be interested in him? He gets a cold beer from the fridge and watches reruns on TV.

Application of the Nursing Process
ASSESSMENT
Undiagnosed and untreated depression is often associated with more severe presentation of depression, greater suicidality, somatic problems, and severe anxiety or anxiety disorders. Depression in older adults is often missed, especially if there are coexisting medical problems. Depression in children and adolescents may go undiagnosed when attention is focused on behavioral problems (“just a stage”). Racial disparities in health care, among other things, allow for under diagnosing and undertreating African Americans, Hispanics, and other minorities (AHRQ, 2004).

A study by Bijl and associates (2004) concluded that depressed individuals who sought treatment manifesting psychological symptoms were recognized as depressed 90% of the time, in contrast with those who showed somatic symptoms (e.g., chronic pain, insomnia), who were recognized as depressed 50% of the time. In those who had a medical disorder, depression was identified 20% of the time.

Assessment Tools
Numerous standardized screening tools can help the clinician assess the type of depression a person may be
experiencing. For example, the Beck Depression Inventory, the Hamilton Depression Scale, and the Geriatric Depression Scale all are valuable tools. The Zung Self-Rating Depression Scale is a short inventory that highlights predominant symptoms seen in depressed individuals; it is presented here because of its ease of use and summation of depressive symptoms (Figure 12-3).

The National Mental Health Association (NMHA) has a website (www.depression-screening.org) that enables people to take a confidential screening test for depression online and find reliable information on the illness.

**Assessment of Suicide Potential**

The patient should be evaluated for suicidal or homicidal ideation. Between 10% and 15% of depressed people eventually commit suicide (Fuller & Sajatovic, 2000). Initial suicide evaluation might include the following statements or questions:

- “You have said you are depressed. Tell me what that is like for you.”
- “When you feel depressed, what thoughts go through your mind?”
- “Have you ever thought about taking your own life in the past? Now? Do you have a plan? Do you have the means to carry out your plan? Is there anything that would prevent you from carrying out your plan?”

Refer to Chapter 20 for more on suicide prevention and intervention.

**Areas to Assess**

**Mood**

A depressed mood and anhedonia (lack of enjoyment in life) are the key symptoms in depression. Nearly 97% of people with depression have anergia (lack of energy). Anxiety, a common symptom in depression, is seen in about 60% to 90% of depressed patients. Some feelings that may be inherent in a depressed mood are as follows:

- Feelings of worthless range from feeling inadequate to having an unrealistic evaluation of self-worth.
These feelings reflect the low self-esteem that is a painful partner to depression. Statements such as “I am no good, I’ll never amount to anything” are common. Themes of one’s inadequacy and incompetence are repeated relentlessly.

- **Guilt** is a common accompaniment to depression. A person may ruminate over present or past failings. Extreme guilt can assume psychotic proportions: “I have committed terrible sins,” “I have caused terrible pain and destruction to everyone I have ever known and now I’m paying for it.”

- **Helplessness** is evidenced by everything believed too difficult to accomplish (e.g., grooming, housework, working, caring for children). With feelings of helplessness come feelings of hopelessness. Even though most depressive states are usually time limited, during a depressed period, people believe that things will never change, which leads some to look at suicide as a way out of constant mental pain. Hopelessness is one of the core characteristics of depression and suicide, as well as a characteristic of schizophrenia, alcoholism, and physical illness. Hopelessness results in negative expectations for the future and loss of control over future outcomes.

- **Anger** and irritability are natural outcomes of profound feelings of helplessness. Anger in depression is often expressed inappropriately. For example, anger may be expressed in destruction of property, hurtful verbal attacks, or physical aggression toward others. Anger may also be directed toward the self in the form of suicidal or self-destructive behaviors (alcohol abuse, substance abuse, overeating, smoking, etc.). These behaviors often result in feelings of low self-esteem and worthlessness.

### Physical Changes

A person who is depressed sees the world through gray-colored glasses. Posture is poor, and the patient may look older than the stated age. Facial expressions convey sadness and dejection, and the patient may have frequent bouts of weeping. Conversely, the patient may say that he or she is unable to cry. Feelings of hopelessness and despair are readily reflected in the person’s affect. For example, the patient may not make eye contact, may speak in a monotone, may show little or no facial expression (flat affect), and may make only yes or no responses. Frequent sighing is common.

People who are depressed often complain of lack of energy (anergia). Lethargy and fatigue can result in psychomotor retardation. Movements are slow, facial expressions are decreased, and gaze is fixed. The continuum in psychomotor retardation may range from slowed and difficult movements to complete inactivity and incontinence. At other times the nurse may note psychomotor agitation. For example, patients may constantly pace, bite their nails, smoke, tap their fingers, or engage in some other tension-relieving activity. At these times, patients feel fidgety and unable to relax.

Grooming, dress, and personal hygiene are markedly neglected. People who usually take pride in their appearance and dress may be poorly groomed and allow themselves to look shabby and unkempt.

- **Vegetative signs of depression** are universal. Vegetative signs refer to alterations in those activities necessary to support physical life and growth (eating, sleeping, elimination, sex). For example, changes in eating patterns are common. About 60% to 70% of people who are depressed report having anorexia; overeating occurs more often in dysthymia.

- **Changes in sleep patterns** are a cardinal sign of depression. Often, people have insomnia, waking at 3 or 4 AM and staying awake, or sleeping only for short periods. The light sleep of a depressed person tends to prolong the agony of depression over a 24-hour period. For some, sleep is increased (hypersonnia) and provides an escape from painful feelings. This is more common in young depressed individuals or those with bipolar tendencies. In any event, sleep is rarely restful or refreshing.

- **Changes in bowel habits** are common. Constipation is seen most frequently in patients with psychomotor retardation. Diarrhea occurs less frequently, often in conjunction with psychomotor agitation. **Interest in sex declines** (loss of libido) during depression. Some men experience impotence, and a declining interest in sex often occurs among both men and women, which can further complicate marital and social relationships.

Stuart (2003) reports that more than two thirds of people suffering from depression complain of pain with or without reporting psychological symptoms. People who suffer from chronic pain need careful assessment for possible depression.

### Cognition

When people are depressed, their thinking is slow and their memory and concentration are usually affected. Depressed people dwell on and exaggerate their perceived faults and failures and are unable to focus on their strengths and successes. As mentioned, identifying the presence of suicidal thoughts and suicide potential has the highest priority in the initial assessment. Approximately two thirds of depressed people contemplate suicide, and up to 15% of untreated or inadequately treated patients give up hope and actually follow through with the suicide (see Chapter 20).
When depressed, a person’s ability to solve problems and think clearly is negatively affected. Judgment is poor, and indecisiveness is common. The individual may claim that the mind is slowing down. Evidence of delusional thinking may be seen in a person with major depression. Common statements of delusional thinking are “I have committed unpardonable sins,” and “I am wicked and should die.”

**Assessment Guidelines**

**Depression**

1. Always evaluate the patient’s risk of harm to self or others. Overt hostility is highly correlated with suicide (see Chapter 20).

2. A thorough medical and neurological examination helps determine if the depression is primary or secondary to another disorder. Depression can be secondary to a host of medical or other psychiatric disorders, as well as medications. Essentially, evaluate whether:
   - The patient is psychotic
   - The patient has taken drugs or alcohol
   - Medical conditions are present
   - The patient has a history of a comorbid psychiatric syndrome (eating disorder, borderline or anxiety disorder)

3. Assess history of depression and determine what happened as well as what worked and did not work.
   - “Have you ever gone through or felt anything like this before?”
   - “What seemed to help you at that time?”

4. Assess support systems, family, and significant others and the need for information and referrals.
   - “With whom do you live?”
   - “Whom do you trust?”
   - “To whom do you talk when you are upset?”

5. Assess for any events that might have “triggered” a depressive episode.
   - “Has anything happened recently to upset you?”
   - “Have you had any major changes in your life?”
   - “Have you had any recent losses: job, divorce, loss of partner, child moving away, deaths?”

6. Include a psychosocial assessment that includes cultural beliefs related to mental health and treatment, and spiritual practices and how the depression is affecting the patient’s beliefs and practice.
   - “How do you view depression?”
   - “Have you tried taking any over-the-counter remedies [e.g., herbs] to help with your depression?”
   - “Do you find solace in spiritual activities or find in a place of worship [e.g., church, temple, mosque]?”

**DIAGNOSIS**

Depression is complex, and depressed individuals have a variety of needs, and nursing diagnoses are many. However, during the initial assessment, a high priority for the nurse is identification of the presence of suicide potential. Therefore the nursing diagnosis of Risk for suicide is always considered. Other key targets for nursing interventions are represented by the diagnoses of Hopelessness, Ineffective coping, Social isolation, Spiritual distress, and one or more of the Self-care deficits (bathing/hygiene, dressing/grooming, feeding, toileting). Table 12-2 identifies signs and symptoms commonly experienced in depression and offers possible nursing diagnoses.

**OUTCOMES IDENTIFICATION**

Outcomes should include goals for safety. Even if the patient is not having self-destructive thoughts, one goal should be to name a person that the patient will contact if such thoughts arise. Goals for the outcomes of the vegetative or physical signs of depression (e.g., reports adequate sleep) are formulated to show, for example, evidence of weight gain, return to normal bowel activity, sleep of 6 to 8 hours per night, or return of sexual desire.

**PLANNING**

The planning of care for patients with depression is geared toward the phase of depression the person is in and the particular symptoms the person is exhibiting. At all times the nurse and members of the health care team are cognizant of the potential for suicide, and assessment of risk for self-harm (or harm to others) is ongoing during the care of the depressed person. There is evidence that a combination of therapy (cognitive, behavioral, interpersonal) and psychopharmacology can be an effective approach in treating depression.

Nurses and clinicians need to assess and plan for any vegetative signs of depression, as well as changes in concentration, activity level, social interaction, personal appearance, and so on. Therefore the planning of care for a patient who is depressed is based on the individual’s symptoms and attempts to encompass a variety of areas in the person’s life. Safety is always the highest priority.

**IMPLEMENTATION**

**Communication Guidelines**

A person who is depressed may speak and comprehend very slowly. The lack of an immediate response by the patient
to a remark does not mean that the patient has not heard or chooses not to reply; rather, the patient just needs a little more time to compose a reply. In extreme depression, however, a person may be mute.

Some depressed patients are so withdrawn that they are unwilling or unable to speak. Nurses may feel uncomfortable with silence and not be able to “do anything” to effect immediate change. However, just sitting with a patient in silence may be a valuable intervention. It is important to be aware that this time spent together can be meaningful to the depressed person, especially if the nurse has a genuine interest in learning about the depressed individual.

**TABLE 12-2 Potential Nursing Diagnoses for Depression**

<table>
<thead>
<tr>
<th>Signs and Symptoms</th>
<th>Nursing Diagnoses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous suicidal attempts, putting affairs in order, giving away prized possessions, suicidal ideation (has plan, ability to carry it out), overt or covert statements regarding killing self, feelings of worthlessness, hopelessness, helplessness</td>
<td>Risk for suicide</td>
</tr>
<tr>
<td></td>
<td>Risk for self-mutilation</td>
</tr>
<tr>
<td>Lack of judgment, memory difficulty, poor concentration, inaccurate interpretation of environment, negative ruminations, cognitive distortions</td>
<td>Disturbed thought processes</td>
</tr>
<tr>
<td>Difficulty with simple tasks, inability to function at previous level, poor problem solving, poor cognitive functioning, verbalizations of inability to cope</td>
<td>Ineffective coping</td>
</tr>
<tr>
<td></td>
<td>Interrupted family processes</td>
</tr>
<tr>
<td></td>
<td>Risk for impaired parent/infant/child attachment</td>
</tr>
<tr>
<td></td>
<td>Ineffective role performance</td>
</tr>
<tr>
<td>Difficulty making decisions, poor concentration, inability to take action</td>
<td>Decisional conflict</td>
</tr>
<tr>
<td>Feelings of helplessness, hopelessness, powerlessness</td>
<td>Hopelessness</td>
</tr>
<tr>
<td>Feelings of inability to make positive change in one’s life or have a sense of control over one’s destiny.</td>
<td>Powerlessness</td>
</tr>
<tr>
<td>Questioning meaning of life and own existence, inability to participate in usual religious practices, conflict over spiritual beliefs, anger toward spiritual deity or religious representatives</td>
<td>Spiritual distress</td>
</tr>
<tr>
<td></td>
<td>Impaired religiosity</td>
</tr>
<tr>
<td></td>
<td>Risk for impaired religiosity</td>
</tr>
<tr>
<td>Feelings of worthlessness, poor self-image, negative sense of self, self-negating verbalizations, feeling of being a failure, expressions of shame or guilt, hypersensitivity to slights or criticism</td>
<td>Chronic low self-esteem</td>
</tr>
<tr>
<td></td>
<td>Situational low self-esteem</td>
</tr>
<tr>
<td>Withdrawal, noncommunicativeness, speech that is only in monosyllables, avoidance of contact with others</td>
<td>Impaired social interaction</td>
</tr>
<tr>
<td></td>
<td>Social isolation</td>
</tr>
<tr>
<td></td>
<td>Risk for loneliness</td>
</tr>
<tr>
<td>Vegetative signs of depression: changes in sleeping, eating, grooming and hygiene, elimination, sexual patterns</td>
<td>Self-care deficit (bathing/hygiene, dressing/grooming)</td>
</tr>
<tr>
<td></td>
<td>Imbalanced nutrition: less than body requirements</td>
</tr>
<tr>
<td></td>
<td>Disturbed sleep pattern</td>
</tr>
<tr>
<td></td>
<td>Constipation</td>
</tr>
<tr>
<td></td>
<td>Sexual dysfunction</td>
</tr>
</tbody>
</table>

**VIGNETTE**

Doris, a senior nursing student, is working with a depressed, suicidal, withdrawn woman. The instructor notices in the second week that Doris spends a lot of time talking with other students and their patients and little time with her own patient. In postconference, Doris acknowledges feeling threatened and useless and says that she wants a patient who will interact with her. After reviewing the symptoms of depression, its behavioral manifestations, and the needs of depressed individuals, Doris turns her attention back to her patient and spends time rethinking her plan of care. After 4 weeks of sharing her feelings in postconference...
Early in hospitalization, predischarge counseling should be carried out with the patient and the patient’s significant others. One purpose of this counseling is to clarify the interpersonal stresses and discuss steps that can alleviate tension in the family system. Including significant others in discharge planning facilitates progress in the following ways:

- Increases the understanding and acceptance of the depressed family member during the aftercare period
- Increases the patient’s use of aftercare facilities in the community
- Contributes to higher overall adjustment in the patient after discharge
- Increases understanding of symptoms that signal the need for relapse prevention.

Health teaching also may include teaching and interventions for self-care deficits. In addition to experiencing intense feelings of hopelessness, despair, low self-worth, and fatigue, the depressed person also may have physical deficits related to the depression. Some effective interventions targeting the physical needs of the depressed patient are listed in Table 12-5.

**Health Teaching and Health Promotion**

It is important for patients and their families to understand that depression is a legitimate medical illness over which the patient has no voluntary control. Depressed patients and their families need to learn about the biological symptoms of depression, as well as the psychosocial and cognitive changes. Families need to know about the overt and covert signs of suicidal ideation, and know what to do and who to contact should warning signs of suicidal thinking or planning should occur (see Chapter 20). Review of the medications and their adverse reactions helps families evaluate clinical changes and stay alert for reactions that might affect patient compliance. The section on psychopharmacology provides information on adverse effects to antidepressants and specific areas to be covered in patient and family teaching.

Early in hospitalization, predischarge counseling should be carried out with the patient and the patient’s significant others. One purpose of this counseling is to clarify the interpersonal stresses and discuss steps that can alleviate tension in the family system. Including significant others in discharge planning facilitates progress in the following ways:

- Increases the understanding and acceptance of the depressed family member during the aftercare period
- Increases the patient’s use of aftercare facilities in the community
- Contributes to higher overall adjustment in the patient after discharge
- Increases understanding of symptoms that signal the need for relapse prevention.

Health teaching also may include teaching and interventions for self-care deficits. In addition to experiencing intense feelings of hopelessness, despair, low self-worth, and fatigue, the depressed person also may have physical deficits related to the depression. Some effective interventions targeting the physical needs of the depressed patient are listed in Table 12-5.

**Milieu Therapy**

When a person is acutely and severely depressed, the structure of the hospital setting may be necessary. The depressed person needs protection from suicidal acts in a supervised environment for regulating antidepressant medications. If a patient is thought to be suicidal, finding a safe environ-
**APPLYING THE ART  A Patient with Depression**

**SCENARIO:** After a medical workup revealed no physical problems, Nadia, a 39-year-old mother of three, admitted herself voluntarily to the inpatient psychiatric unit, stating she no longer had the energy to care for her children, her marriage, saying, “I am not fit to be a mother or wife.” I saw Nadia 3 days later and set up a contract with her for after breakfast, expecting that later we would attend group therapy, then meet for one-to-one. Instead Nadia, after missing group, reluctantly met with me in the day room.

**THERAPEUTIC GOAL:** By the conclusion of this interaction, the patient will state she understands that depression is a treatable disorder and that her symptoms were the cause of her despondent behavior.

<table>
<thead>
<tr>
<th>Student-Patient Interaction</th>
<th>Thoughts, Listening Techniques, and Mental Health Nursing Concepts</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nadia: Speaking slowly, eyes downcast. “I couldn’t face all those people.”</td>
<td>Depression slows everything: thoughts, feelings, and responses to others. I make an observation and attempt to translate into feelings, then shift to an indirect question. Because depression hinders Nadia’s processing of information, I need to slow my pace. Allow more silence.</td>
</tr>
<tr>
<td>Student: “You’re looking down like you are sad.” No response from Nadia. “I wonder what facing the group means to you.” Student’s feelings: I should have stayed with “sad.” I aimed for her feelings, then did not wait for her to share any feelings.</td>
<td>Student’s feelings: I should have stayed with “sad.” I aimed for her feelings, then did not wait for her to share any feelings.</td>
</tr>
<tr>
<td>Nadia: Slowly shakes her head back and forth. Silent for 3 minutes. No eye contact.</td>
<td>Student’s feelings: I should have stayed with “sad.” I aimed for her feelings, then did not wait for her to share any feelings.</td>
</tr>
<tr>
<td>Student: With a concerned look. “You shake your head as if you are saying no.” Student’s feelings: I know it’s the right thing to do, but waiting during the silence makes me so anxious. I need to stay mindfully alert and attentive. I can endure the silence for Nadia’s sake.</td>
<td>Student’s feelings: I should have stayed with “sad.” I aimed for her feelings, then did not wait for her to share any feelings.</td>
</tr>
<tr>
<td>Nadia: “Everybody in group makes progress. I just keep sinking deeper.”</td>
<td>Student’s feelings: I should have stayed with “sad.” I aimed for her feelings, then did not wait for her to share any feelings.</td>
</tr>
<tr>
<td>Student: “Sinking deeper?”</td>
<td>Nadia: Speaking slowly, eyes downcast. “I couldn’t face all those people.”</td>
</tr>
<tr>
<td>Nadia: “Into depression. I can’t pull it together even though I know my kids need me.” Makes eye contact. Student’s feelings: I know from experience that it’s hard to pull anything together when you feel depressed.</td>
<td>Student’s feelings: I should have stayed with “sad.” I aimed for her feelings, then did not wait for her to share any feelings.</td>
</tr>
<tr>
<td>Student: “You care about your children.” She nods. “Sounds like you find it difficult at this time to care about yourself very much.” Student’s feelings: I’ve noticed that sometimes, like Nadia, nurses find it easier to care for others than take care of self, even basic self-care or prevention measures.</td>
<td>Student’s feelings: I should have stayed with “sad.” I aimed for her feelings, then did not wait for her to share any feelings.</td>
</tr>
<tr>
<td>Nadia: Sustaining eye contact. “I can’t do anything right. I have nothing to show for my life.”</td>
<td>Student’s feelings: I should have stayed with “sad.” I aimed for her feelings, then did not wait for her to share any feelings.</td>
</tr>
</tbody>
</table>
ment may be the first action taken. Hospitals have protocols for suicidal observation and protection. If a patient is highly suicidal, not eating, becoming debilitated, or has a psychotic depression, then electroconvulsive therapy (ECT) may be administered.

**Psychotherapy**

CBT, interpersonal therapy (IPT), and behavioral therapy have been proven effective in the treatment of depression. However, only CBT and IPT demonstrate superiority in the maintenance phase. CBT helps people change their negative styles of thinking and behaving, whereas IPT focuses on working through personal relationships that may contribute to depression. Outcome research has consistently found that CBT and medication are largely comparable, but CBT is more effective in protecting against relapse than medications (Feldman, 2007).

Some studies indicate that psychotherapy alone (CBT or IPT), especially in individuals with early life traumas (child abuse), is more effective than pharmacology alone (Nemeroff et al., 2007). CBT combined with medications is proven effective in people with chronic depressions (Feldman, 2007).

---

**APPLYING THE ART  A Patient with Depression—cont’d**

<table>
<thead>
<tr>
<th><strong>Student-Patient Interaction</strong></th>
<th><strong>Thoughts, Listening Techniques, and Mental Health Nursing Concepts</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Student:</strong> “Think about what you’ve accomplished! You have your children, your marriage, your teaching career.” Nadia shrugs, eyes downcast. <strong>Student’s feelings:</strong> She has so much going for her. Why can’t she see that? My response causes Nadia to pull away by withdrawing eye contact. When I deliver positives about Nadia before she feels more positive about herself, I discount her experience, which interferes with trust. I need to remember that support and nonjudgmental acceptance provide the foundation for the nurse-patient relationship.</td>
<td>I inadvertently minimized her feelings by giving approval and advice, which is nontherapeutic. Even though all the things I pointed out may be valid, none of it rings true for Nadia right now. One step that helps with depression would be for Nadia to problem-solve and work through any cognitive distortions, e.g., “I can’t do anything right.” Cognitive behavioral therapy, like the antidepressant medication takes time, but depression is a treatable disorder.</td>
</tr>
<tr>
<td><strong>Student:</strong> <em>After waiting for 2 minutes.</em> “Nadia, I am here to be with you right where you are at this moment. No pressure.”</td>
<td>I offer self and acceptance.</td>
</tr>
<tr>
<td><strong>Nadia:</strong> <em>Looking up.</em> “Thank you. You don’t know how much that means. I do want to get better and not feel like depression consumes who I am.”</td>
<td></td>
</tr>
<tr>
<td><strong>Student:</strong> <em>Nods.</em> “You want to get better. You were able to take the first courageous step. In deciding to get admitted, you acknowledge that your symptoms are a problem, and they are the symptoms of depression, a disorder.” <strong>Student’s feelings:</strong> Nadia feels swallowed up (consumed) by the depression. I want her to know that depression need not be her life.</td>
<td>I give support. Separating out oneself as distinct from the disorder of depression restores some sense of control to Nadia.</td>
</tr>
<tr>
<td><strong>Nadia:</strong> “Oh. I never thought of it that way . . . as a first step, not a sign of failure. My symptoms are from the depression.” <strong>Student’s feelings:</strong> As a nurse, my belief in Nadia’s ability to battle the depression offers hope.</td>
<td></td>
</tr>
<tr>
<td><strong>Student:</strong> <em>Nods.</em> “A treatable disorder.” I continue to sit with Nadia in silence for a short while.</td>
<td>At some level, Nadia acknowledges a self not fully consumed by depression.</td>
</tr>
<tr>
<td><strong>Nadia:</strong> “Yes, depression is a disorder, not all that I am.”</td>
<td>Hope will grow as Nadia begins to take charge of her disorder through active investment in treatment.</td>
</tr>
</tbody>
</table>
Group Therapy

Group therapy is a widespread modality for the treatment of depression; it increases the number of people who can receive treatment at a decreased cost per individual. Another advantage is that groups offer patients an opportunity to socialize and to share common feelings and concerns as well as provide patients with the opportunity to reach out to others and support others. Belonging to a group can help decrease feelings of isolation, hopelessness, helplessness, and alienation. Medication groups for patients and families can increase understanding of medications, how to handle various side effects, and compliance.

Pharmacological, Biological, and Complementary Interventions

Antidepressant Medication Therapy

Antidepressant therapy benefits about 65% to 80% of people with nondelusional unipolar depression. ECT has a 75% to 85% efficacy rate for those patients who are delusional or melancholic (Maxmen & Ward, 2002). It should be noted, however, that the combination of specific psychotherapies (e.g., CBT, IPT, behavioral) and antidepressant therapy is superior to either psychotherapy or psychopharmacological treatment alone (Sutherland et al., 2003). In fact, it is believed that the combination and continuation of at least two of these therapies may reduce the risk of recurrence or relapse of MDD and DD (Dubovsky et al., 2004). Essentially, the core symptoms of depression improve with antidepressant therapy, and quality-of-life measures improve with certain psychotherapies (Culpepper et al., 2003).

Antidepressant drugs can positively alter poor self-concept, degree of withdrawal, vegetative signs of depression, and activity level. Target symptoms include the following:

- Sleep disturbance
- Appetite disturbance (decreased or increased)
- Fatigue
- Decreased sex drive
- Psychomotor retardation or agitation
- Diurnal variations in mood (often worse in the morning)

TABLE 12-4  Interventions for Depression: Communication

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Help the patient question underlying assumptions and beliefs and consider alternate explanations to problems.</td>
<td>1. Reconstructing a healthier and more hopeful attitude about the future can alter depressed mood.</td>
</tr>
<tr>
<td>2. Work with the patient to identify cognitive distortions that encourage negative self-appraisal. For example:</td>
<td>2. Cognitive distortions reinforce a negative, inaccurate perception of self and world.</td>
</tr>
<tr>
<td>a. Overgeneralizations</td>
<td>a. The patient takes one fact or event and makes a general rule out of it (“He always . . .”; “I never . . .”).</td>
</tr>
<tr>
<td>b. Self-blame</td>
<td>b. The patient consistently blames self for everything perceived as negative.</td>
</tr>
<tr>
<td>c. Mind reading</td>
<td>c. The patient assumes others don’t like him or her, and so forth, without any real evidence that assumptions are correct.</td>
</tr>
<tr>
<td>d. Discounting of positive attributes</td>
<td>d. The patient focuses on the negative.</td>
</tr>
<tr>
<td>3. Encourage activities that can raise self-esteem. Identify need for (a) problem-solving skills, (b) coping skills, and (c) assertiveness skills.</td>
<td>3. Many depressed people, especially women, are not taught a range of problem-solving and coping skills. Increasing social, family, and job skills can change negative self-assessment.</td>
</tr>
<tr>
<td>4. Encourage exercise, such as running and/or weightlifting. Initially walking 10 to 15 minutes a day 3 or 4 times a week has short-term benefits.</td>
<td>4. Exercise can help alleviate depression and anxiety, improve self-concept, and shift neurochemical balance.</td>
</tr>
<tr>
<td>5. Encourage formation of supportive relationships, such as through support groups, therapy, and peer support.</td>
<td>5. Such relationships reduce social isolation and enable the patient to work on personal goals and relationship needs.</td>
</tr>
<tr>
<td>6. Provide information referrals, when needed, for spiritual/religious information (e.g., readings, programs, tapes, community resources).</td>
<td>6. Spiritual and existential issues may be heightened during depressive episodes—many people find strength and comfort in spirituality or religion.</td>
</tr>
</tbody>
</table>
Impaired concentration or forgetfulness
Anhedonia (loss of ability to experience joy or pleasure in living)

One drawback to the use of antidepressant medication is that improvement in mood may take 1 to 3 weeks or longer. If a patient is acutely suicidal, this may be too long to wait. At these times, ECT may be a consideration in some facilities.

TABLE 12-5 Interventions Targeting the Physical Needs of the Depressed Patient

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nutrition—Anorexia</strong></td>
<td>1. Low weight and poor nutrition render the patient susceptible to illness. Small, frequent snacks are more easily tolerated than large plates of food when the patient is anorexic.</td>
</tr>
<tr>
<td>1. Offer small high-calorie and high-protein snacks frequently throughout the day and evening.</td>
<td>1. The patient is more likely to eat the foods provided.</td>
</tr>
<tr>
<td>2. Offer high-protein and high-calorie fluids frequently throughout the day and evening.</td>
<td>2. These fluids prevent dehydration and can minimize constipation.</td>
</tr>
<tr>
<td>3. When possible, encourage family or friends to remain with the patient during meals.</td>
<td>3. This strategy reinforces the idea that someone cares, can raise the patient’s self-esteem, and can serve as an incentive to eat.</td>
</tr>
<tr>
<td>4. Ask the patient which foods or drinks he or she likes. Offer choices. Involve the dietitian.</td>
<td>4. Monitoring the patient’s status gives the information needed for revision of the intervention.</td>
</tr>
<tr>
<td>5. Weigh the patient weekly and observe the patient’s eating patterns.</td>
<td></td>
</tr>
<tr>
<td><strong>Sleep—Insomnia</strong></td>
<td>1. Fatigue can intensify feelings of depression.</td>
</tr>
<tr>
<td>1. Provide periods of rest after activities.</td>
<td>2. Minimizing sleep during the day increases the likelihood of sleep at night.</td>
</tr>
<tr>
<td>2. Encourage the patient to get up and dress and to stay out of bed during the day.</td>
<td>3. These measures induce relaxation and sleep.</td>
</tr>
<tr>
<td>3. Encourage the use of relaxation measures in the evening (e.g., tepid bath, warm milk).</td>
<td>4. Decreasing caffeine and epinephrine levels increases the possibility of sleep.</td>
</tr>
<tr>
<td>4. Reduce environmental and physical stimulants in the evening—provide decaffeinated coffee, soft lights, soft music, quiet activities.</td>
<td></td>
</tr>
<tr>
<td><strong>Self-Care Deficits</strong></td>
<td>1. Being clean and well groomed can temporarily increase self-esteem.</td>
</tr>
<tr>
<td>1. Encourage the use of toothbrush, washcloth, soap, makeup, shaving equipment, and so forth.</td>
<td>2. Slowed thinking and difficulty concentrating make organizing simple tasks difficult.</td>
</tr>
<tr>
<td>2. When appropriate, give step-by-step reminders such as, “Wash the right side of your face, now the left.”</td>
<td></td>
</tr>
<tr>
<td><strong>Elimination—Constipation</strong></td>
<td>1. Many depressed patients are constipated. If the condition is not checked, fecal impaction can occur.</td>
</tr>
<tr>
<td>1. Monitor intake and output, especially bowel movements.</td>
<td>2. Roughage and exercise stimulate peristalsis and help evacuation of fecal material.</td>
</tr>
<tr>
<td>2. Offer foods high in fiber and provide periods of exercise.</td>
<td>3. Fluids help prevent constipation.</td>
</tr>
<tr>
<td>3. Encourage the intake of fluids.</td>
<td>4. These measures prevent fecal impaction.</td>
</tr>
<tr>
<td>4. Evaluate the need for laxatives and enemas.</td>
<td></td>
</tr>
</tbody>
</table>

A Note About Safety

The possibility that antidepressant medication might contribute to suicidal behavior has been well covered in the media and caused grave concerns among the professional community and general public. However, there has not been any conclusive evidence to support this concern. To the contrary, the review of the use of selective serotonin reuptake inhibitors (SSRIs) in 27 countries over time saw a strong
association with increased antidepressant prescribing and a reduction in suicide (Ludwig & Marcotte, 2005).

All treatments have potential risks. At present, there is no conclusive evidence that either the newer or the older antidepressants precipitate suicide (Goldberg et al., 2006). The U.S. Food and Drug Administration (FDA) is still evaluating data from numerous studies and recommends that all consumers of antidepressants be observed carefully for worsening of depression and suicidal thought. This is especially true for children, adolescents, and older adults.

**Choosing an Antidepressant**

All antidepressants work equally well, although they certainly do not all work well for all individuals. Because the complex interplay of neurotransmitters responsible for depression is unique for different individuals, a variety of antidepressants or a combination of antidepressants may need to be tried before the most effective regimen is found. Each antidepressant has adverse effects as well as cost, safety, and maintenance considerations. The following are some of the primary and secondary considerations when choosing a specific antidepressant:

**Primary considerations**
- Side effect profile (e.g., sexual dysfunction, weight gain)
- Ease of administration
- Past response
- Safety and medical considerations
- Specific depressive symptoms (e.g., anxiety, irritability, hyposomnia, insomnia)
- Medical considerations (diabetes, high cholesterol, cardiac disease)

**Secondary considerations**
- Neurotransmitter specificity
- Family history of response
- Cost

The neurotransmitters and receptor sites in the brain are the targets of pharmacological intervention (Table 12-6). While reading the following section, see if you can identify potential side effects caused by the blockage of the given neurotransmitter.

Studies that compare the newer SSRIs to the older tricyclic antidepressants (TCAs) fail to find support for one group over the other (U.S. Dept. of Veterans Affairs, 2006). The difference lies in the quality and quantity of adverse effects and complications. Basic antidepressant classes include the following:

**First-Line Agents**
- Cyclic antidepressants (e.g., TCAs)
- SSRIs
- The newer atypical antidepressants

**Second-Line Agents**
- Monoamine oxidase inhibitors (MAOIs)

**Tricyclic Antidepressants**

The tricyclic antidepressants (TCAs) inhibit the reuptake of norepinephrine and serotonin by the presynaptic neurons in the CNS. Therefore the amount of time that norepinephrine and serotonin are available to the postsynaptic receptors is increased. This increase in norepinephrine and serotonin in the brain is believed to be responsible for mood elevations when TCAs are given to depressed people.
The sedative effects of the TCAs are attributed to antihistamine (H₁ receptor) actions and somewhat to anticholinergic actions (Maxmen & Ward, 2002). Patients must take therapeutic doses of TCAs for 10 to 14 days or longer before they begin to work. The full effects may not be seen for 4 to 8 weeks. An effect on some symptoms of depression, such as insomnia and anorexia, may be noted sooner. A person who has had a positive response to TCA therapy would probably be maintained on that medication for 6 to 12 months to prevent an early relapse. Choice of TCA is based on the following:

- What has worked for the patient or a family member in the past
- The drug’s adverse effects

For example, a patient who is lethargic and fatigued may have the best results with a more stimulating TCA, such as desipramine (Norpramin) or protriptyline (Vivactil). If a more sedating effect is needed for agitation or restlessness, drugs such as amitriptyline (Elavil) and doxepin (Sinequan) may be more appropriate choices. Regardless of which TCA is given, the dosage should always be low initially and should be increased gradually. Caution should be used, especially in older adults because slow drug metabolism may be a problem. Trimipramine (Surmontil) is a good choice for older adults because of its low side effects and its rapid effects in promoting sleep. The rule of thumb for older adults is always, “Start low, go slow.”

Common Adverse Reactions. The chemical structure of the TCAs is similar to that of the antipsychotic medications. Therefore the anticholinergic actions (e.g., dry mouth, blurred vision, tachycardia, constipation, urinary retention, and esophageal reflux) are similar. These side effects are more common and more pronounced in patients taking antidepressants. These adverse effects are usually not serious and are often transitory, but urinary retention and severe constipation warrant immediate medical attention.

The α-adrenergic blockade of the TCAs can produce postural orthostatic hypotension and tachycardia. Postural hypotension can lead to dizziness and increase the risk of falls.

Administering the total daily dose of TCA at night is beneficial for two reasons. First, most TCAs have sedative effects and thereby aid sleep. Second, the minor side effects occur during sleep, which increases compliance with drug therapy. Table 12-7 reviews TCAs in common use, their common side effects, and dosage ranges.

Potential Toxic Effects. The most serious effects of the TCAs are cardiovascular: dysrhythmias, tachycardia, myocardial infarction, and heart block. Because the cardiac side effects are so serious, TCA use is considered a risk in patients with cardiac disease and in older adults. Patients should have a thorough cardiac workup before beginning TCA therapy.

Drug Interactions. Individuals taking TCAs can have adverse reactions to numerous other medications. A few of the more common medications usually not given while TCAs are being used are listed in Box 12-2. A patient who is taking any of these medications along with a TCA should have a medical clearance beforehand because some of the reactions can be fatal.

Use of antidepressants may precipitate a psychotic episode in a person with schizophrenia. An antidepressant can precipitate a manic episode in a patient with bipolar disorder (BD). Depressed patients with BD often receive lithium along with the antidepressant.

Contraindications. People who have recently had a myocardial infarction (or other cardiovascular problems), those with narrow-angle glaucoma or a history of seizures, and pregnant women should not be treated with TCAs, except with extreme caution and careful monitoring.

Patient Teaching. Teaching patients and one or more of their significant others about medications is an expected nursing responsibility. Medication teaching is begun in the hospital. The nurse or another qualified health care provider must review the medications, possible side effects, and necessary patient precautions. Areas for the nurse to discuss when teaching patients and their families about TCA therapy are presented in Box 12-3. Patients and significant others need to have written information for all medications that will be taken at home.

---

**Box 12-2**

Drugs to Be Used with Caution in Patients Taking a Tricyclic Antidepressant

- Phenothiazines
- Barbiturates
- Monoamine oxidase inhibitors
- Disulfiram (Antabuse)
- Oral contraceptives (or other estrogen preparations)
- Anticoagulants
- Some antihypertensives (clonidine, guanethidine, reserpine)
- Benzodiazepines
- Alcohol
- Nicotine
Selective Serotonin Reuptake Inhibitors

The introduction of Prozac, the first selective serotonin reuptake inhibitor (SSRI) in 1988 heralded an important advance in pharmacotherapy. Essentially, the SSRIs selectively block the neuronal uptake of serotonin (e.g., 5-HT, 5-HT1 receptors) thereby leaving more serotonin available at the synaptic site. (See Chapter 4 for detailed information on how the SSRIs work.)

SSRI antidepressant drugs have a lower incidence of anticholinergic side effects (e.g., dry mouth, blurred vision, urinary retention), less cardiotoxicity, and faster onset of action than the TCAs. Patients are more likely to comply with a regimen of SSRIs than of TCAs because of the more favorable side effect profile, and compliance is a crucial step toward recovery or remission. The SSRIs seem to be effective in depression with anxiety features as well as in depression with psychomotor agitation.

Because the SSRIs cause fewer adverse effects and have low cardiotoxicity, they are less dangerous when they are taken in overdose. The SSRIs, serotonin-norepinephrine reuptake inhibitors (SNRIs), and newer atypical antidepressants have a low lethality risk in suicide attempts compared with the TCAs, which have a very high potential for lethality with overdose.

Indications. The SSRIs have a broad base of clinical use. In addition to their use in treating depressive disorders, the SSRIs have been prescribed with success to treat some of the anxiety disorders, in particular, obsessive-compulsive disorder and panic disorder (see Chapter 8). Fluoxetine has been found to be effective in treating some women who suffer from late luteal phase dysphoric disorder and bulimia nervosa.

Common Adverse Reactions. Agents that selectively enhance synaptic serotonin within the CNS may induce agitation, anxiety, sleep disturbance, tremor, sexual dysfunction (primarily anorgasmia), or tension headache. The effect of the SSRIs on sexual performance may be the most significant undesirable outcome reported by patients.

Autonomic reactions (e.g., dry mouth, sweating, weight change, mild nausea, and loose bowel movements) also may be experienced with the SSRIs. See Table 12-8 for a general side effect profile of the SSRIs, specific SSRIs, and dosage.

Potential Toxic Effects. One rare and life-threatening event associated with the SSRIs is serotonin syndrome. This is thought to be related to overactivation of the central nervous system, and has been reported in patients who have ingested SSRIs, TCAs, and MAO inhibitors concomitantly. The serotonin syndrome is characterized by restlessness, agitation, fever, tachycardia, rigidity, diaphoresis, and hyperreflexia; it can be fatal if untreated.
The risk of this syndrome seems to be the greatest when an SSRI is administered in combination with a second serotonin-enhancing agent, such as an MAOI. For example, a person taking fluoxetine would have to be off this medication for a full 5 weeks before being switched to an MAOI (5 weeks is the half-life for fluoxetine). If a patient is already taking an MAOI, the person should wait at least 2 weeks before starting fluoxetine therapy. Other SSRIs have shorter half-lives, but the guidelines for switching between SSRIs and MAOIs are similar. It is important for patients to understand the potential for serotonin syndrome and to be monitored closely by healthcare providers during the transition period between medications.

TABLE 12-8  Selective Serotonin Reuptake Inhibitors (SSRIs): Overview of Adverse Reactions and Dosage Range

<table>
<thead>
<tr>
<th></th>
<th>Sedation</th>
<th>Weight Gain</th>
<th>Sexual Dysfunction</th>
<th>Other Key Adverse Reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>SSRIs</td>
<td>Minimal</td>
<td>Rare</td>
<td>Yes</td>
<td>• Initial: nausea, loose bowel movements, headache, insomnia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Toxic effects (rare): serotonin syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Concern regarding increased suicidal potential as yet unproven through long-term studies</td>
</tr>
</tbody>
</table>

**Specific Medications**

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Initial Dosage (mg/Day)</th>
<th>Dosage After 4 to 8 Weeks (mg/Day)</th>
<th>Maximum Dosage (mg/Day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Citalopram</td>
<td>Celexa</td>
<td>10-20</td>
<td>20-60</td>
<td>60</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>Prozac</td>
<td>10-20</td>
<td>20-80</td>
<td>80</td>
</tr>
<tr>
<td>Fluvoxamine*</td>
<td>Luvox</td>
<td>50-100</td>
<td>100-200</td>
<td>300</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>Paxil</td>
<td>10-20</td>
<td>20-50</td>
<td>50</td>
</tr>
<tr>
<td>Sertraline</td>
<td>Zoloft</td>
<td>50</td>
<td>50-150</td>
<td>200</td>
</tr>
<tr>
<td>Escitalopram</td>
<td>Lexapro</td>
<td>10</td>
<td>10</td>
<td>10</td>
</tr>
</tbody>
</table>

*Older adult patients and those with hepatic disease should start at 50% less than the standard dosages listed in the table. This applies to patients with coexisting panic or anxiety symptoms.

*Escitalopram is the single active isomer of citalopram, which gives it some advantages in the treatment of depression.

Serotonin receptors, caused by either too high a dose or interaction with other drugs. Symptoms include abdominal pain, diarrhea, sweating, fever, tachycardia, elevated blood pressure, altered mental state (delirium), myoclonus (muscle spasms), increased motor activity, irritability, hostility, and mood change. Severe manifestation can induce hyperpyrexia (excessively high fever), cardiovascular shock, or death.
periods of activity; for example, sertraline and paroxetine have half-lives of 2 weeks, so there would need to be a 2-week gap between different medications.

Box 12-4 lists the symptoms of serotonin syndrome and gives emergency treatment guidelines. Box 12-5 is a useful tool for patient and family teaching about the SSRIs.

**New Atypical (Novel) Antidepressants**

Most newly released antidepressants affect a variety of neurotransmitters. These novel antidepressants are all effective agents. Table 12-9 introduces these newer atypical antidepressants and identifies the main neurotransmitters involved. Each of these agents blocks different neurotransmitters and transmitter subtypes, which accounts for their strengths in targeting unique populations of depressed individuals as well as for their efficacy in treating other conditions. Table 12-10 lists the usual maintenance daily dose and presents some of the advantages and disadvantages of each of these atypical agents.

**Monoamine Oxidase Inhibitors**

MAOIs demonstrate proven benefits for patients who have not responded to other medication or ECT treatment. They also have been found useful in refractory anxiety states. In particular, MAOIs have established efficacy in depression in people with atypical depression (Table 12-1).

In addition to being effective with atypical depression and MDD, MAOIs can be useful in treating other disorders such as panic disorder, social phobia, generalized anxiety...
disorder, obsessive-compulsive disorder, posttraumatic stress disorder, and bulimia. Essentially, MAOIs prevent the breakdown of norepinephrine, serotonin, and dopamine in the brain, thereby increasing the levels of these brain amines and resulting in increased mood. (See Chapter 4 for detailed information on how the MAOIs work.) Common adverse reactions and potential toxic effects of MAOIs are outlined in Table 12-11.

Unfortunately, the MAOIs inhibit the enzyme tyramine from breaking down certain foods and drugs in the liver. Increased levels of tyramine can lead to high blood pressure, hypertensive crisis, and eventually cerebrovascular accident and death. Therefore people taking MAOIs must restrict their intake of tyramine so that their blood pressure does not rise to dangerous levels. See Table 12-12 for a list of foods that are high on tyramine and other vasopressors.

Until 2006, the MAOIs commonly used in the United States were phenelzine (Nardil) and tranylcypromine sulfate (Parnate). In 2006 the FDA approved an MAOI that is delivered transcutaneously by way of a patch called the

### Table 12-9 Newer Atypical (Novel) Antidepressants

<table>
<thead>
<tr>
<th>Agent</th>
<th>Neurotransmitters Affected</th>
<th>May Help People With:</th>
</tr>
</thead>
</table>
| Bupropion (Wellbutrin, Zyban) | Blocks norepinephrine (NE) and dopamine (DA) reuptake (NDRI) | • ADHD  
• Chronic fatigue syndrome  
• Rapid cycling bipolar II disorder  
• Sexual side effects from use of other antidepressants  
• Anxiety disorders (GAD, OCD, phobic disorders, PTSD, panic disorders)  
• Nicotine addiction (Zyban)  
• Older adult patients  
• SSRI-induced insomnia |
| Trazodone (Desyrel)  | Shows selective but moderate blockage of serotonin (5-HT	extsubscript{2} receptor) (only used in conjunction with other drugs) | |
| **Dual-Action Reuptake Inhibitors—SNRIs (Serotonin and Norepinephrine)** | | |
| Venlafaxine (Effexor) | Inhibits reuptake of serotonin (5-HT) and NE  
Inhibits DA to a lesser extent | • Treatment-resistant depression  
• Chronic depression  
• Bipolar depression  
• Depression with ADHD  
• Medical illness and depression  
• Anxiety  
• Geriatric depression  
• Sleep disturbances  
• Poor appetite  
• Pain  
• Medical illness with depression  
• Anxiety  
• SSRI-induced sexual dysfunction  
• Major depression  
• Geriatric depression |
| Mirtazapine (Remeron) | Blocks serotonin (5-HT, 5-HT	extsubscript{2a}, 5-HT	extsubscript{3}, 5-HT	extsubscript{4} receptors), is an α	extsubscript{2}-adrenergic receptor antagonist (ACh), and blocks histamine (H	extsubscript{1}) (enhances both nonadrenergic and serotonergic transmitters) | |
| Duloxetine (Cymbalta) | Inhibits reuptake of serotonin (5-HT) and NE (SNRI)  
Inhibits DA to a lesser extent | |
| **Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs)** | | |
| Roboxetine (Vestra, Edronax)* | A selective norepinephrine reuptake inhibitor (NE, ACh) | • SSRI-related sexual dysfunction  
• Lethargy secondary to depression  
• Cognitive difficulties secondary to depression  
• Impaired social functioning  
• Anxiety disorders (panic attacks) |

*Roboxetine is not yet approved for the U.S. market. It is available in Europe.

ACh, Acetylcholine; ADHD, attention deficit hyperactivity disorder; GAD, generalized anxiety disorder; OCD, obsessive-compulsive disorder; PTSD, posttraumatic stress disorder; SSRI, selective serotonin reuptake inhibitor.
selegiline transdermal system (STS). STS is able to inhibit monamine oxidase in the central nervous system, increasing the availability of norepinephrine, serotonin, and dopamine, while at the same time avoiding the breakdown of tyramine in the liver and digestive tract. When STS is applied in doses of 6 mg over 24 hours by way of a skin patch, it does not require a tyramine-restricted diet (Nemeroff et al., 2007). At higher doses (9 or 12 mg), dietary restrictions must be observed.

See Table 12-13 for an overview of MAOIs in current use, their adverse effects, and dosage ranges. Patients who do not improve with initial therapy often show improvement when switched to another class of antidepressants or when a drug from another class is added to the therapy.
## TABLE 12-11  Common Adverse Reactions to and Toxic Effects of Monoamine Oxidase Inhibitors

<table>
<thead>
<tr>
<th>Adverse Reactions</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypotension</td>
<td>Hypotension is the most critical side effect (10%); older adults, especially, may sustain injuries from it.</td>
</tr>
<tr>
<td>Sedation, weakness, fatigue</td>
<td></td>
</tr>
<tr>
<td>Insomnia</td>
<td></td>
</tr>
<tr>
<td>Changes in cardiac rhythm</td>
<td></td>
</tr>
<tr>
<td>Muscle cramps</td>
<td></td>
</tr>
<tr>
<td>Anorgasemia or sexual impotence</td>
<td></td>
</tr>
<tr>
<td>Urinary hesitancy or constipation</td>
<td></td>
</tr>
<tr>
<td>Weight gain</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Toxic Effects</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertensive crisis*</td>
<td>1. Patient should go to local emergency department immediately—blood pressure should be checked.</td>
</tr>
<tr>
<td>Severe headache</td>
<td>2. One of the following may be given to lower blood pressure:</td>
</tr>
<tr>
<td>Stiff, sore neck</td>
<td>• 5 mg intravenous phentolamine (Regitine) or</td>
</tr>
<tr>
<td>Flushing, cold, clammy skin</td>
<td>• Oral chlorpromazine or</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>• Nifedipine (Procardia) (calcium channel blocker), 10 mg sublingually</td>
</tr>
<tr>
<td>Severe nosebleeds, dilated pupils</td>
<td></td>
</tr>
<tr>
<td>Chest pains, stroke, coma, death</td>
<td></td>
</tr>
<tr>
<td>Nausea and vomiting</td>
<td></td>
</tr>
</tbody>
</table>

*Related to interaction with foodstuffs and cold medication.

---

## TABLE 12-12  Foods That Can Interact with Monoamine Oxidase Inhibitors

<table>
<thead>
<tr>
<th>Foods That Contain Tyramine</th>
<th>Unsafes High Tyramine Content</th>
<th>Safe Foods Little or No Tyramine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetables</td>
<td>Avocados, especially if overripe; fermented bean curd; fermented soybean; soybean paste</td>
<td>Most vegetables</td>
</tr>
<tr>
<td>Fruits</td>
<td>Figs, especially if overripe; bananas, in large amounts</td>
<td>Most fruits</td>
</tr>
<tr>
<td>Meats</td>
<td>Meats that are fermented, smoked, or otherwise aged; spoiled meats; liver, unless very fresh</td>
<td>Meats that are known to be fresh (exercise caution in restaurants; meats may not be fresh)</td>
</tr>
<tr>
<td>Sausages</td>
<td>Fermented varieties; bologna, pepperoni, salami, others</td>
<td>Nonfermented varieties</td>
</tr>
<tr>
<td>Fish</td>
<td>Dried, pickled, or cured fish; fish that is fermented, smoked, or otherwise aged; spoiled fish</td>
<td>Fish that is known to be fresh; vacuum-packed fish, if eaten promptly or refrigerated only briefly after opening</td>
</tr>
<tr>
<td>Milk, milk products</td>
<td>Practically all cheeses</td>
<td>Milk, yogurt, cottage cheese, cream cheese</td>
</tr>
<tr>
<td>Foods with yeast</td>
<td>Yeast extract (e.g., Marmite, Bovril)</td>
<td>Baked goods that contain yeast</td>
</tr>
<tr>
<td>Beer, wine</td>
<td>Some imported beers, Chianti</td>
<td>Major domestic brands of beer; most wines</td>
</tr>
<tr>
<td>Other foods</td>
<td>Protein dietary supplements; soups (may contain protein extract); shrimp paste; soy sauce</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Foods That Contain Other Vasopressors</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chocolate</td>
<td>Contains phenylethylamine, a pressor agent; large amounts can cause a reaction.</td>
</tr>
<tr>
<td>Fava beans</td>
<td>Contain dopamine, a pressor agent; reactions are most likely with overripe beans.</td>
</tr>
<tr>
<td>Ginseng</td>
<td>Headache, tremulousness, and mania-like reactions have occurred.</td>
</tr>
<tr>
<td>Caffeinated beverages</td>
<td>Caffeine is a weak pressor agent; large amounts may cause a reaction.</td>
</tr>
</tbody>
</table>

Partial text of the document:

**TABLE 12-13**  
**Monoamine Oxidase Inhibitors (MAOIs): Overview of Adverse Reactions and Dosage Range**

<table>
<thead>
<tr>
<th>MAOIs</th>
<th>Sedation</th>
<th>Weight Gain</th>
<th>Sexual Dysfunction</th>
<th>Other Key Adverse Reactions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rare</td>
<td>Yes</td>
<td>Yes</td>
<td></td>
<td>• Orthostatic hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Insomnia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Peripheral edema (avoid use in patients with CHF)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Avoid phenelzine in patients with hepatitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Potential life-threatening drug interactions</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>• Strict dietary and medication restrictions (see Table 12-12 and Box 12-7)</td>
</tr>
</tbody>
</table>

**Specific Medications**

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Initial Dosage (mg/Day)</th>
<th>Dosage After 4 to 8 Weeks (mg/Day)</th>
<th>Maximum Dosage (mg/Day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenelzine</td>
<td>Nardil</td>
<td>15</td>
<td>45-60</td>
<td>90</td>
</tr>
<tr>
<td>Tranylcypromine</td>
<td>Parnate</td>
<td>10</td>
<td>30-40</td>
<td>60</td>
</tr>
<tr>
<td>Selegiline transdermal system (STS)</td>
<td>EMSAM</td>
<td>6</td>
<td>6-9</td>
<td>12</td>
</tr>
</tbody>
</table>

**Reversible Inhibitors of MAO Not Yet Available in the United States**

| Moclobemide | Manerix, Aurorix | 300 | 300-600 | 900 |

**BOX 12-6**

**Patient and Family Teaching About Monoamine Oxidase Inhibitors**

- Tell the patient and the patient’s family to avoid certain foods and all medications (especially cold remedies) unless prescribed by and discussed with the patient’s physician (see Table 12-12 and Box 12-7 for specific food and drug restrictions).
- Give the patient a wallet card describing the monoamine oxidase inhibitor (MAOI) regimen.
- Instruct the patient to avoid Chinese restaurants (where sherry, brewer’s yeast, and other contraindicated products may be used).
- Tell the patient to go to the emergency department immediately if he or she has a severe headache.
- Ideally, monitor the patient’s blood pressure during the first 6 weeks of treatment (for both hypotensive and hypertensive effects).
- Instruct the patient that after the MAOI is stopped, dietary and drug restrictions should be maintained for 14 days.

**Contraindications.** Use of MAOIs may be contraindicated when one of the following is present:

- Cerebrovascular disease
- Hypertension and congestive heart failure
- Liver disease
- Consumption of foods containing tyramine, tryptophan, and dopamine (see Table 12-12)
- Use of certain medications (see Box 12-7)
- Recurrent or severe headaches
- Surgery in the previous 10 to 14 days
- Age younger than 16 years

Box 12-6 can be used as a teaching guide for patients and their families regarding MAOIs.
Somatic Treatments

Electroconvulsive Therapy

Electroconvulsive therapy (ECT) remains one of the most effective treatments for major depression and life-threatening psychiatric conditions (e.g., self-harm). Unfortunately, it is also one of the most stigmatized treatments for depression (NIMH, 2000a). During the early years of ECT, methods were primitive and unrefined, but today ECT is considered safe and effective.

ECT can achieve a higher than 90% remission rate in depressed patients within 1 to 2 weeks. Because 20% to 30% of depressed individuals do not respond to antidepressants, ECT remains an effective treatment particularly for depressions with psychotic features or those refractory to other treatments. ECT is indicated when:

- There is a need for a rapid, definitive response when a patient is suicidal or homicidal.
- A patient is in extreme agitation or stupor.
- A patient develops a life-threatening illness because of refusal of foods and fluids.
- The patient has a history of poor drug response, a history of good ECT response, or both.

ECT is useful in treating patients with major depressive and bipolar depressive disorders, especially when psychotic symptoms are present (e.g., delusions of guilt, somatic delusions, or delusions of infidelity). Patients who have depression with marked psychomotor retardation and stupor also respond well. However, ECT is not necessarily effective in patients with DD, atypical depression, personality disorders, drug dependence, or depression secondary to situational or social difficulties. The usual course of ECT for a depressed patient is two or three treatments per week to a total of 6 to 12 treatments.

Procedure. The procedure is explained to the patient, and informed consent is obtained if the patient is being treated voluntarily. When informed consent cannot be obtained from a patient treated involuntarily, permission may be obtained from the next of kin, although in some states treatment must be court ordered. Use of a general anesthetic and muscle-paralyzing agents has revolutionized the comfort and safety of ECT.

Potential Adverse Reactions. On awakening from ECT, the patient may be confused and disoriented. The nurse and significant others may need to orient the patient frequently during the course of treatment. Many patients state that they have memory deficits for the first few weeks after treatment. Memory usually, although not always, recovers. ECT is not a permanent cure for depression, and maintenance treatment with TCAs or lithium decreases the relapse rate. Maintenance ECT (once a week to once a month) may also help to decrease relapse rates for patients with recurrent depression.

Vagus Nerve Stimulation

Vagus nerve stimulation (VNS) is an FDA-approved, adjunctive, long-term treatment for patients with treatment resistant depression (TRD) (those with chronic or recurrent MDD who have failed a minimum of four antidepressant medication trials) (Sadock & Sadock, 2007). ECT is considered by many the most effective acute intervention for TRD, but TRD patients often relapse during the first year following ECT.

The exact method of therapeutic action of VNS is not totally understood. VNS does affect blood flow to different parts of the brain and affects neurotransmitters including serotonin and norepinephrine, which are implicated in depression. A 2-year study by Sackeim and associates (2007) of the efficacy of VNS demonstrated a 50% improvement for people with severe chronic depression that was resistant to other therapies. Between 61% and 79% sustained this response for 24 months (Grohol, 2007).

VNS involves a surgically implanted device (upper left chest) that sends electric impulses to the left vagus nerve in the neck at regular intervals. “Wearable” devices are being developed and tested. Because the vagus nerve affects many functions of the brain, VNS is being studied for other conditions as well (e.g., anxiety disorder, Alzheimer’s disease, migraines, and fibromyalgia) (Grohol, 2007).

Integrative Therapies

Light Therapy

Light therapy is the first-line treatment for seasonal affective disorder (SAD) (see Table 12-1). People with SAD often live in climates in which there are marked seasonal
differences in the amount of daylight. Seasonal variations in mood disorders in the Southern Hemisphere are the reverse of those in the Northern Hemisphere. Light therapy also may be useful as an adjunct in treating chronic MDD or dysthymia with seasonal exacerbations (APA, 2000b).

Light therapy is thought to be effective because of the influence of light on melatonin. Melatonin is secreted by the pineal gland and is necessary for maintaining and shifting biological rhythms. Exposure to light suppresses the nocturnal secretion of melatonin, which seems to have a therapeutic effect on people with SAD (Zahourek, 2000). Treatments consist of exposure to light balanced to replicate the effects of sunlight for 30 to 60 minutes a day.

**St. John’s Wort**

*St. John’s wort* (*Hypericum perforatum*) is a whole plant product with antidepressant properties that is not regulated by the FDA. St. John’s wort has superior efficacy compared with placebo and was generally comparable in effect to low-dose TCAs, and less so to SSRIs (Mischoulon, 2007). The herb is not to be taken in certain situations (e.g., MDD, pregnancy, age younger than 2 years) (Fuller & Sajatovic, 2000). Nor should St. John’s wort be taken with certain substances, such as amphetamines or other stimulants, other antidepressants (MAOIs, SSRIs), warfarin, theophylline, or digoxin (Fuller & Sajatovic, 2000; Mischoulon, 2007). Research suggests that St. John’s wort may be less effective in cases of severe or chronic depression and that people who have the milder forms of depression are the best candidates (Mischoulon, 2007).

**Exercise**

There is substantial evidence that exercise can enhance mood and reduce symptoms of depression and anxiety (Mayo Foundation for Medical Education and Research, 2005). It may take at least 30 minutes a day for at least 3 to 5 days a week in order to reduce the symptoms of depression and anxiety; however, shorter periods of time (10 to 15 minutes) have shown to reduce depression and anxiety in the short term (Mayo Foundation for Medical Education and Research, 2005).

**The Future of Treatment**

There is a great need for earlier detection and intervention, achievement of remission, prevention of progression, and integration of neuroscience and behavioral science in the treatment of depression (Greden, 2004). High-risk ages and groups, including the following, are in need of screening:

- Individuals in late adolescence and early adulthood
- Women in their reproductive years
- Adults and older adults with medical problems
- People with a family history of depression

There is also a need for education, particularly about the linkage between physical symptoms and depression. Psychopharmacological treatment should be augmented with cognitive-behavioral therapies, and there is need for more supplementary strategies, such as the following:

- Promotion of sleep hygiene
- Increase in exercise
- Better total health care

Continual research will bring more genetic screening tools and pharmacogenetics understanding; the use of neuroimaging will become a common diagnostic tool and will not be restricted to research. These and other advances are consistent with the goals of the President’s New Freedom Commission on Mental Health (2003) for transforming mental health care in America. (www.mentalhealthcommission.gov/reports/finalreport/toc.html).

**Transcranial Magnetic Stimulation**

Transcranial magnetic stimulation (TMS) applies the principles of electromagnetism to deliver an electrical field to the cerebral cortices, but unlike ECT, the waves do not result in generalized seizure activity (Rosenbaum, 2004). Early studies of this technique support further research into its use in the treatment of serious, relapsing, medication-resistant depression. Whereas some studies of TMS find significant antidepressant effect in individuals with medication-resistant major depression (Avery et al., 2006).

**Brain Imaging**

A study from the University of Wisconsin–Madison (Johnstone et al., 2007), perhaps the first study to use brain imaging, revealed a breakdown in normal patterns of emotional processing in people who are depressed. Using a functional magnetic resonance imaging scanner, the researchers found that healthy people are able to regulate their negative emotions through conscious efforts, such as envisioning a more positive outcome or reframing a negative situation. The scan revealed that high levels of regulatory activity correlated with low levels of activity in the emotional response centers. They found that some depressed individuals lacked the ability to regulate emotions. In these individuals, high levels of regulatory activity did not change the levels of activity in the emotional centers, demonstrating the neural circuits regulating emotion in some depressed individuals are dysfunctional.

**EVALUATION**

Short-term indicators and outcome criteria are frequently evaluated. For example, if the patient comes into the unit with suicidal thoughts, the nurse evaluates whether the
patient still has suicidal thoughts, is able to state alternatives to suicidal impulses in the future, and is able to explore thoughts and feelings that precede suicidal impulses. Outcomes relating to thought processes, self-esteem, and social interactions are frequently formulated because these areas are often problematic in people who are depressed.

Physical needs warrant nursing or medical attention. If a person has lost weight because of anorexia, is the appetite returning? If a person was constipated, are the bowels now functioning normally? If the person was suffering from insomnia, is he or she now getting 6 to 8 hours of sleep per night? If the indicators have not been met, an analysis of the data, nursing diagnoses, goals, and planned nursing interventions is made. The patient should be reassessed and the care plan reformulated when necessary.

### KEY POINTS TO REMEMBER

- Depression is the most commonly seen psychiatric disorder in the health care system.
- There are a number of subtypes of depression and depressive clinical phenomena. The two primary depressive disorders are major depressive disorder (MDD) and dysthymic disorder (DD).
- The symptoms in major depression are usually severe enough to interfere with a person’s social or occupational functioning (inability to experience pleasure [anhedonia], significant weight loss, insomnia or hypersomnia, extreme fatigue [anergia], psychomotor agitation or retardation, diminished ability to think or concentrate, feelings or worthlessness, recurrent thoughts of death).
- A person with MDD may or may not have psychotic symptoms, and the symptoms a person usually exhibits during a major depression are different from the characteristics of the normal premorbid personality.
- In DD, the symptoms are often chronic (lasting at least 2 years) and are considered mild to moderate. Usually, a person’s social or occupational functioning is not as greatly impaired as they are in MDD, although they may cause significant distress or some impairment in these areas. The symptoms in a dysthymic depression are often congruent with the person’s usual pattern of functioning.
- Many theories exist about the cause of depression. The most accepted is the psychophysiological theory; however, cognitive theory, learned helplessness theory, and psychodynamic and life events issues help explain triggers to depression and maintenance of depressive thoughts and feelings.
- Nursing assessment includes the evaluation of affect, thought processes (especially suicidal thoughts), feelings, physical behavior, and communication. The nurse also needs to be aware of the symptoms that mask depression.
- Nursing diagnoses can be numerous. Depressed individuals are always evaluated for Risk for suicide. Some other common nursing diagnoses are Disturbed thought processes, Chronic low self-esteem, Imbalanced nutrition, Constipation, Disturbed sleep pattern, Ineffective coping, and Disabled family coping.
- Interventions with patients who are depressed involve several approaches, including using specific principles of communication, planning activities of daily living, administering or participating in psychopharmacological therapy, maintaining a therapeutic environment, and teaching patients about the biochemical aspects of depression and medication teaching.
- Several short-term psychotherapies are effective in the treatment of depression, including IPT, CBT, and some forms of group therapy.
- Electroconvulsive therapy (ECT) is an effective treatment for people with major depression with psychotic features and patients refractory to other treatments. Vagus nerve stimulation (VNS) can be a valuable adjunctive treatment in treatment-resistant depression. Light therapy is the first line of treatment for seasonal effective disorders (SAD).
- Evaluation is ongoing throughout the nursing process, and patients’ outcomes are compared with the stated outcome criteria and short-term and intermediate goals. The care plan is revised by use of the evaluation process when desired outcomes are not being met.

### CRITICAL THINKING

1. You are spending time with Mr. Plotsky, who is being given a workup for depression. He hardly makes eye contact and slouches in his seat, and his expression appears blank, but sad. Mr. Plotsky has had numerous bouts of major depression in the past and says to you, “This will be my last depression. I will never go through this again.”

   A. If safety is the first concern, what are the appropriate questions to ask Mr. Plotsky at this time?

   B. Give an example of the kinds of signs and symptoms you might find when you assess a patient with depression in terms of behaviors, thought processes, activities of daily living, and ability to function at work and at home?

   C. Mr. Plotsky tells you that he has been on every medication there is but that none have worked. He asks you about the herb St. John’s wort. What is some
CRITICAL THINKING—cont’d

information he should have about its effectiveness for severe depression, its interactions with other antidepressants, and its regulatory status?
D. What might be some somatic options for a person who is resistant to antidepressant medications?
E. Mr. Plotsky asks what causes depression. In simple terms, how might you respond to his query?
F. Mr. Plotsky tells you that he has never tried therapy because he thinks it is for babies. What information could you give him about various therapeutic modalities that have proven effective for some other depressed patients?

2. When you are teaching Ms. Mac about her SSRI sertraline (Zoloft), she asks you, "What makes this such a good drug?"
A. What are some of the positive attributes of SSRIs?
What is one of the most serious, although rare, side effects of the SSRIs?
B. Devise a teaching plan for Ms. Mac.

CHAPTER REVIEW

Choose the most appropriate answer.
1. If a nurse subscribes to the theory that learned helplessness is a major factor in the development of depression, which statement best represents her belief?
   1. TCAs, MAOIs, and SSRIs are the most useful tools to combat depression.
   2. Depression develops when a person believes he or she is powerless to effect change in a situation.
   3. Depressive symptoms result from experiencing significant loss and turning aggression against the self.
   4. Psychosocial stressors and interpersonal events trigger neurophysical and neurochemical changes in the brain.

2. Which response to a patient experiencing depression would be helpful from the nurse?
   1. "Don’t worry, we all get down once in a while."
   2. “Don’t consider suicide. It’s an unacceptable option.”
   3. “Try to cheer up. Things always look darkest before the dawn.”
   4. “I can see you’re feeling down. I’ll sit here with you for a while.”

3. Which of the following is considered a vegetative symptom of depression?
   1. Sleep disturbance
   2. Trouble concentrating
   3. Neglected grooming and hygiene
   4. Negative expectations for the future

4. For a person with severe depression, which statement about cognitive functioning is true?
   1. Reality testing remains intact.
   2. Concentration is unimpaired.
   3. Repetitive negative thinking is noted.
   4. Ability to make decisions is improved.

5. When the nurse is caring for a depressed patient, the problem that should receive the highest nursing priority is:
   1. powerlessness.
   2. suicidal ideation.
   3. inability to cope effectively.
   4. anorexia and weight loss.

REFERENCES

Johnstone, T., van Reekum, C.M., & Urry, H.L. (2007). Failure to
Golden, R.N., Dawkins, K., Nicholas, L. (2006). Trazodone and
Golden, R.N., Dawkins, K., Nicholas, L. (2006). Trazodone and

Mood Disorders: Depression  ■  ■  ■  CHAPTER 12  243