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Contact Hours

How to Diagnose and Treat Depression

Abstract

Depression remains the most common problem in primary care; however, it's gravely underdiagnosed and undertreated. This article details how to diagnose and treat depression in adults and in special populations, including postpartum women, children and adolescents, the medically ill, and the elderly.

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Depression, a syndrome of episodic, clustered, psychological and physiological signs and symptoms, affects 3% to 5% of the general population and 5% to 15% of the primary care population.[1-3] Despite its pervasiveness, research indicates that primary care clinicians fail to recognize as many as 50% of depression cases.[4] Many patients with depression receive inadequeate treatment or no treatment at all, which results in decreased quality of life, high morbidity and mortality, and economic losses.[5] Considering these potential consequences, you should clearly understand the clinical manifestations and risk factors for depression in order to diagnose it accurately and early, prescribe appropriately, and minimize sequalae.

Barriers to diagnosis and treatment of depression are many. First, depression carries a stigma, though less today than in times past, and the general population may still view it as a character flaw. Hence, patients may be reluctant to accept a diagnosis of depression and may focus on the disease's physical manifestations rather than treatment.[3,5] Second, patients or their families often underestimate the severity of depression or believe they can self-treat it, which can delay treatment.[5] So, patient education is essential in accepting the diagnosis.

Third, clinicians may lack the skills and information to handle the emotional aspects of depression. Fourth, clinicians may fear alienation of their patients because a diagnosis of depression may invoke higher insurance premiums or refusal of insurance for the patient.[5,6] Time restrictions and lack of adequate reimbursement or inadequate insurance coverage create additional barriers. Finally, formularies can restrict your prescribing newer antidepressants, limiting treatment options.[5]

Identifying Depression

Depression can be a chronic and recurrent disease requiring ongoing treatment for relapses.[5,6] A single episode of depression isn't the norm, and the chance of having a second episode is 50% to 65%. Patients who have depression also frequently have another disorder such as anxiety.[7,8] Clinicians who treat patients for depression should evaluate them at frequent intervals to assess efficacy of pharmacologic therapy and provide support.[5]

The peak age for depression onset is 20 to 40, with the highest risk occurring in those who have a family history of the disease. Studies of monozygotic twins that show their parallel rate of depression is 65% support this genetic component.[1] Women are twice as likely as men to become depressed—especially those who bear a child at a young age.[1,8] Other risk factors include a prior episode or episodes of depression, prior suicide attempts, being in the postpartum period, medical comorbidity, lack of social support, stressful life events, and a history of sexual abuse or current substance abuse.[4,8]

Rx Of the 2.0 ANCC contact hours awarded for this activity, 1.0 is applicable toward a pharmacology requirement.

Symptoms

Depressed patients usually don't complain of sadness or despair. Instead, somatic complaints may contain hidden signs of depression, making it difficult to differentiate symptoms of depression from organic disease.[1] Common physical complaints presenting as depression, starting with the most common, are loss of energy or fatigue, unexplained pain, gastrointestinal symptoms, headache, insomnia, dizziness, palpitations, heartburn, numbness, loss of appetite, and premenstrual syndrome. Insomnia, specifically early morning awakening, is a reliable and early indicator of depression.[1] In general, the more somatic complaints and unexplained physical symptoms a patient has, the higher the likelihood of depression.[1,3,8]

Screening for Depression

Because of insufficient evidence to recommend for or against screening tests in asymptomatic patients, the U.S. Preventive Services Task Force doesn't recommend general screening. You should, however, have a high suspicion of symptoms in those who have risk factors for depression.[2,3] In these at-risk patients, several questionnaires can help detect depressive symptoms with a high index of sensitivity, but a low index of specificity. The Beck Depression Inventory (BDI), the Center for Epidemiological Studies Depression Scale (CES-D), and the Zung Self-Rating Depression Scale (SDS) can help detect depression in adult patients with a reliable measure of accuracy.[2] These brief questionnaires take 2 to 5 minutes for the patient to complete and have a point system that allows rapid scoring. Begin with one questionnaire and use others, if needed.

Another approach is the Two-Question Case-Finding Instrument. To use this method, ask the patient, "During the past month, have you often been bothered by feeling down, depressed, or hopeless?" and "During the past month, have you often been bothered by having little interest or pleasure in doing things?". If the answer to both questions is *no*, it's unlikely that the patient has depression. If the answer is *yes*, to one or both questions, follow up with the diagnostic criteria for a major depressive episode (see Table 1, "Criteria for Diagnosing a Major Depressive Disorder") to confirm the diagnosis. The reported sensitivity for this screening method is 96%, with specificity of only 57% for the two-question instrument.

Diagnosing Depression

A clinical diagnosis of depression involves taking a complete medical history and history of present illness, examining the patient physically and mentally, evaluating social history, reviewing pharmacologic therapies, testing to rule out substance abuse or any organic cause that can lead to a depressive episode, and differentiating depression from other psychiatric disorders.[8,9]

Organic causes to consider are hypothyroidism, neurosyphilis, substance abuse, and major organ system disease such as cardiovascular, liver, renal, and neuronal. Laboratory tests to consider include complete blood count with differential, sedimentation rate, venereal disease research laboratory test, chemistry profile, thyroid profile, and drug screen. Although the sedimentation rate is a nonspecific marker, it's elevated in collagen-vascular diseases, infections, neoplasms, inflammatory states, and hyperthyroidism.[9] A variety of drugs may also cause depression, for example, antihypertensives, anticonvulsants, beta-blockers, steroids, cancer chemotherapy, levodopa, and benzodiazepines.[8]

In addition to ruling out organic diseases, differentiate other mental health disorders from depression. The differential diagnosis of mental health disorders includes bipolar disorder, dementia, schizophrenia, schizoaffective disorder,

Criteria for Diagnosing a Major Depressive Disorder

Depressed mood or markedly decreased pleasure in most activities that occurs for 2 weeks or more defines a major depressive disorder. Patients will experience at least five of the following symptoms nearly every day. These symptoms cause clinically significant distress or impairment in social, occupational, or other functioning. To be considered a major depressive disorder, psychotropic drugs or a general medical condition aren't the cause of these symptoms and they don't occur within 2 months of the loss of a loved one:

- Depressed mood (irritability in children and adolescents) most of the day, nearly every day
- Markedly diminished interest or pleasure in almost all activities most of the day, nearly every day, as indicated either by subjective account or observation by others
- Significant weight loss or gain
- Insomnia or hypersomnia
- Psychomotor agitation or retardation
- Fatigue or loss of energy
- Feeling of worthlessness or guilt
- Impaired concentration or indecisiveness
- Recurrent thoughts of death or suicide.

somatoform disorder, anxiety, attention deficit hyperactivity disorder (ADHD), and personality disorder. With a bipolar disorder, the patient suffers from manic episodes. Dementia has an insidious onset with a progressive and irreversible impairment in cognitive function. In schizophrenia and schizoaffective disorders, patients experience hallucinations, delusions, and other psychotic symptoms, with the age of onset usually in the twenties.[1,8-11]

A somatoform disorder, generally occurring before age 30, is a pattern of recurrent multiple somatic complaints (a minimum of four) that persists over several years. With an anxiety disorder, the patient experiences excessive fear and worry with at least three somatic symptoms of restlessness, irritability, sleep disturbances, muscle tension, difficulty concentrating, or fatigability. Keep in mind that anxiety and depression often coexist. ADHD, which frequently manifests itself before age 7, is a persistent pattern of inattention or hyperactivity and impulsivity that is greater than expected for age. Patients with a personality disorder are narcissistic, selfish, inflexible, have poor impulse control, and their behavior deviates from the environmental cultural norm.[1,8-11]

Identifying Types of Depression

The criteria for a major depressive episode is defined by the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*) (see Table 1, "Criteria for Diagnosing a Major Depressive Disorder").[8,9,11] Most patients with depressive symptoms have a minor depressive disorder, which doesn't meet the criteria for major depressive disorder. Instead, patients with minor depressive disorder have fewer than 5 symptoms of a major depressive disorder that last 2 weeks, with symptoms not occurring during a psychotic episode.[8,9,11]

Dysthymia is a low-grade depression that often occurs with an anxiety disorder. It's a less severe but more chronic form of depression than other types, but it carries an increased risk of developing major depressive disorder. Major depression can be superimposed on dysthymia. This condition is called *double depression*.[1,11]

Symptomatology allows for differentiation of dysthymia from major or minor depression. Dysthymia diagnostic criteria requires that symptoms are present for 2 years or longer and include at least 2 of the following symptoms: poor appetite or overeating, insomnia or hyposomnia, lack of energy or fatigue, low self-esteem, poor concentration or difficulty making decisions, and hopelessness.[3,8,9,11]

Factors that Increase Suicide Risk

- Older than age 65
- Male sex
- White race or Native-American ethnicity
- Single, divorced, separated, or widowed (especially without children)
- Unemployment
- History of admission to a psychiatric ward
- Family or personal history of one or more suicide attempts
- Drug or alcohol abuse
- Severely stressful life event in recent past
- Panic attacks or severe anxiety
- Severe physical illness, especially of recent onset
- Severe hopelessness
- Anhedonia
- Specific plan for suicide
- Access to firearms or other lethal means

Addressing Suicide Risk

Consider suicide risk a primary consideration in patients with a depressive disorder. Patients with severe forms of major depression have a 15% risk of death from suicide.[5] Men, especially elderly white men, are more likely than others to commit suicide (see Table 2, "Factors that Increase Suicide Risk").[3]

In addition to an extensive list of risk factors, asking three questions may help evaluate a person's risk of committing suicide. "Do you ever think of hurting yourself or taking your own life?" If the answer is *yes*, the following two questions can further evaluate matters. "Do you currently have a plan?" and, if so, "What is your plan?" Clinicians may avoid these questions for fear of implanting the idea of suicide, but this fear is unfounded. Instruct the patient to inform you of any suicidal thoughts.[3]

Identifying Depression in Special Populations

Children and Adolescents

Children (younger than age 13) and adolescents (ages 13 to 19) don't present with the same depressive symptoms as adults. Because children often don't internalize their behavior as adults and adolescents do, early signs for them may be disobedience, excessive acting up or out, temper tantrums, or running away from home. Also, children's depression may manifest in school with dysfunction, inattention, restlessness, or aggression.[3,11]

Adolescents' depression may involve hypersomnia, hyperphagia, an irritable or labile mood, a change in friends or peer groups, substance abuse, and vegetative symptoms.[11]

The presence of comorbid disorders is common in both children and adolescents, with a reported 40% to 70% occurrence of associated anxiety, dysthymia, substance abuse, conduct disorder, and ADHD.[12] The National Institute of Mental Health recommends involvement of a child psychiatrist, psychologist, or psychiatric NP, or other qualified mental health specialist in the evaluation, diagnosis, and treatment of children and adolescents suspected of being depressed.[13]

Women in the Postpartum Period

Postpartum depression occurs in 5% to 20% of women within 6 months of delivery. Don't confuse it with postpartum blues, which as many as 85% of new mothers experience within 2 weeks of giving birth. Postpartum blues generally resolve spontaneously within 6 to 12 weeks.[3] With postpartum depression, the new mother's depressive symptoms center around concerns for the well-being of the infant and her own maternal inadequacy. Many dispel the associated physical symptoms of loss of appetite, lack of energy, and sleeplessness as the common experiences of a new mother. Failure to diagnose and treat postpartum depression deprives the new mother of a joyful period and may have long-term effects on the mother and her child.[14]

Those with Comorbid Conditions

Many diseases can trigger depression. For example, 25% of patients with cancer, as many as 50% of patients with Parkinson's disease, and 33% of those with dementia will have depression.[1] Stroke is another strong predictor of depression, with estimates ranging from 45% to 48%. A group of diseases called *depression-spectrum diseases* share a high comorbidity with depression. These diseases include chronic fatigue syndrome, fibromyalgia, and body dysmorphic disorder.[8] Appetite, sleep, and energy changes associated with illnesses overlap with the symptoms of depression and can make depression diagnosis a challenge. To help differentiate, look for feelings of worthlessness and guilt, which often occur with depression but not with other illnesses.[1,8]

Elderly

The elderly often have vague physical symptoms that could indicate depression. Because of polypharmacy in the elderly, distinguishing between drug reactions and depressive symptoms may be difficult. Drugs that cause depressive symptoms include steroids, antihypertensives, digoxin, anti-Parkinson's drugs, reserpine, antihistamines, and nonsteroidal antiinflammatory drugs.[3,9] Distinguishing dementia from depression may also prove challenging.[9] Of concern for this population is the increase in morbidity and mortality associated with depression and coronary artery disease.[3] Also, this population has a high risk of suicide.[15]

Nonpharmacologic Treatment Options

Treatment options for depression include nonpharmacologic interventions (including psychotherapy), antidepressant drug therapy, and electroconvulsive therapy.[12] Most patients respond to a combination of psychotherapy and antidepressant drugs.[7,12,16]

Some nonpharmacologic methods of treating depression include psychotherapy, exercise, support groups, self-help literature, and light therapy. Patients shouldn't undergo nonpharmacologic therapies unsupervised or for prolonged periods because longer periods of depression are more resistant to treatment and 70% to 80% of patients respond to an adequate trial of pharmacologic therapy.[7,12,16,17]

Psychotherapeutic strategies for depression include supportive, cognitive, interpersonal, behavioral, family, and group therapy and assertiveness training.[9,17] Cognitive or interpersonal therapy are the most effective psychotherapeutic means of treating depression, shortening depressive episodes.[7,9] Cognitive therapy can alter the negative outlook that many depressed patients hold toward themselves and their lives. Interpersonal therapy, which can help those with interpersonal problems, maximizes the patient's ability to function socially while avoiding social isolation, which may worsen the condition.[12] Both cognitive and interpersonal psychotherapy can reduce depression relapse.[17,18]

Well-motivated patients with insight into their problems tend to excel in psychotherapy, while the severely depressed (those displaying concentration and memory deficits, hopelessness, and inobjectivity) don't.[7,16] Cognitive and interpersonal therapy focus on the present and are problem-solving oriented. They may occur in many formats: group, family, marital, and behavior therapy. Refer candidates for psychotherapy to a psychotherapist. Patients can expect to attend hourly sessions every week for 1 to 10 weeks.[7]

Pharmacologic Treatment Options

Patients who meet the criteria for major depressive disorder may receive pharmacologic therapy. Antidepressant drug ther-

apy may be an option for those who don't meet major depressive disorder criteria but whose depression has lasted longer than a month or interferes with roles related to family, work, and, school.[8,9]

Consider several factors when selecting an antidepressant. Previous response to earlier antidepressant trials can help rule drug classes in or out. Evaluate the patient's clinical presentation in view of anticipated drug effects and adverse effects to maximize drug benefit, particularly those that may lead to drug termination. Also consider ease of administration and cost.[19]

Antidepressants fall into four classes, according to mechanism of action (see Table 3, "Four Functional Classes of Antidepressants").[8,9] You may initiate pharmacological treatment with any nonmonoamine oxidase inhibitor antidepressant.[12] The primary reason for antidepressant treatment failure is drug nonadherence. In most cases, patients stop taking antidepressants because of troublesome adverse effects.

Selective Serotonin Reuptake Inhibitors

Although most antidepressants are equally effective in reversing depression in outpatients, selective serotonin reuptake inhibitors (SSRIs) have become the initial drug of choice, because they act at fewer neurotransmitter sites, produce fewer adverse effects, and therefore patients tolerate them better than tricyclic antidepressants (TCAs).[7,8] Also, SSRIs are safer in overdose than TCAs and are simple to titrate up to optimal dosage.[7,12] Of the antidepressants prescribed, 50% are SSRIs.[7]

Although half of patients taking SSRIs don't report any adverse effects, these drugs can induce unpleasant and dan-

Table 3

Four Functional Classes of Antidepressants

Selective serotonin reuptake inhibitors

Citalopram (Celexa), fluoxetine (Prozac), fluvoxamine (Luvox), paroxetine (Paxil), sertraline (Zoloft) and escitalopram (Lexapro).
Advantages: Low side effect profile, wide therapeutic index, and simple titration.
Disadvantages: Sexual adverse effects, disturbs sleep physiology, cytochrome P450 inhibition, may cause serotonin syndrome, and withdrawal.

Tricyclic antidepressants tertiary amines

Amitriptyline (Elavil), clomipramine (Anafranil), doxepine (Sinequan), imipramine (Tofranil), and trimipramine (Surmontil).
Advantages: Effective for SSRI nonresponders, inexpensive, and treats comorbid conditions.
Disadvantages: Lethal in overdose, cardiotoxic, numerous adverse effects, needed drug titration and medical monitoring, and numerous drug interactions.

Tricyclic antidepressants norepinephrine selective reuptake inhibitors

Desipramine (Norpramin), maprotiline (Ludiomil), nortryptyline (Pamelor, Aventyl), and protriptyline (Vivactil).
Advantages: Effective for SSRI nonresponders, adjunctive use to SSRIs drugs, and less antihistamine/anticholinergic effects than tricyclic antidepressant tertiary amines.
Disadvantages: Lethal in overdose; cardiotoxic; prolonged QTc and can cause sinus and AV node dysfunction, leading to heart block and dysrhythmias. Closely monitor blood pressure.

Atypicals

Vanlafaxine (Effexor)
Advantages: Well-tolerated, wide therapeutic index, minimal cytochrome P450 inhibition, and no dose titration.
Disadvantages: SSRI adverse effects at low doses and NSRI adverse effects at high doses.

Nefazodone (Serzone)
Advantages: Treats comorbid anxiety
Disadvantages: Sedating

Trazodone (Desyrel)
Advantages: Wide therapeutic index, and safety with overdose.
Disadvantages: Can cause priapism in men (1 in 8,000)

Mirtazapine (Remeron)
Advantages: Safety with overdose, doesn't disturb sleep physiology, and doesn't cause sexual dysfunction
Disadvantages: Sedating; can cause increased appetite and weight gain.

Bupropion (Wellbutrin, Zyban)
Advantages: Doesn't disturb sleep physiology; causes little, if any, sexual dysfunction.
Disadvantages: Can cause seizures; has a narrow therapeutic index.

Monoamine oxidase inhibitors

Phenelzine (Nardil), tranylcypromine (Parnate)
Advantages: Effective in patients with depressive symptoms refractory to other drugs.
Disadvantages: Can cause hypotension; potentially fatal drug-drug interactions with serotonin agonists, norepinephrine agonists, and tyramine-rich foods.

gerous reactions, including diarrhea, dry mouth, and (most commonly) nausea.[20] But patients usually develop a tolerance to these effects. Appetite suppression is common early in therapy. And, some patients who have taken SSRIs for more than a year report weight gain. SSRIs may stimulate the central nervous system, causing symptoms such as motor restlessness, headache, nervousness, and insomnia. They may also alter sleep architecture, causing a shift from deep sleep to light sleep and reducing REM sleep. Some patients complain of sweating. Some 30% of patients taking SSRIs suffer effects such as anorgasmia, erectile dysfunction, or abnormal ejaculation.[8] Some 15% of SSRI users discontinue the drug because of adverse effects.[17]

MOST PATIENTS respond to a combination of psychotherapy and antidepressant drugs. Some nonpharmacologic methods of treating depression include psychotherapy, exercise, support groups, self-help literature, and light therapy. Patients shouldn't undergo nonpharmacologic therapies unsupervised. Consider several factors when selecting an antidepressant. Previous response to earlier antidepressant trials can help rule drug classes in or out.

SSRIs inhibit the cytochrome P450 isoenzymes and, hence, may inhibit the metabolism of other drugs. Unfortunately, depression often coexists with other illnesses that require other drug treatments. Screen patients for drug interactions and possible toxicity. Citalopram (Celexa) and sertraline (Zoloft) cause much less enzyme inhibition and are good SSRI choices for patients taking multiple drugs.[8]

Serotonin syndrome is a potentially fatal pharmacodynamic drug interaction involving SSRIs. Combining SSRIs with other serotonergic drugs (for example, meperidine [Demerol] and sumatriptan [Imitrex]) may induce this syndrome, which decreased mental status and neuromuscular and autonomic nervous system dysfunction characterize. Neuromuscular symptoms include restlessness, tremor, myoclonus, and incoordination. Sustained muscular contraction may be so extreme as to induce metabolic acidosis and rhabdomyolysis. Hyperthermia, sweating, and vital sign instability may indicate autonomic dysfunction.[20]

Tricyclic Antidepressants

Two classes of TCAs exist: the tricyclic antidepressant tertiary amines (TATCAs) and the tricyclic antidepressant norepinephrine selective reuptake inhibitors (NSRIs). TATCAs are the second most commonly prescribed class of antidepressant and are especially useful for comorbid conditions of chronic pain, migraine headaches, or insomnia.[8] Because of a broader range of pharmacologic activity, TATCAs may reverse depression that's unresponsive to SSRIs.

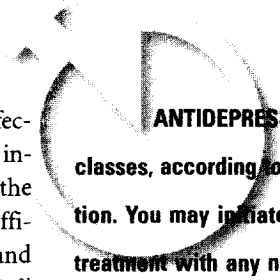
This same broad range of activity produces many poorly tolerated adverse effects. TATCAs block cholinergic muscarinic receptors, resulting in dry mouth, constipation, urinary retention, blurred vision, tachycardia, and memory impairment. TATCA blockage of histamine H1 receptors causes sedation. Alpha 1 adrenergic blockade may cause orthostatic hypotension or dizziness. Because patients must develop a tolerance for these adverse effects, titrate drug dosages until effective. Adverse effects may persist even following dose titration.[8]

TATCAs can be lethal in overdose and interact with many drugs, most notably potentiating antihypertensive and central depressant drugs. They are contraindicated for patients with narrow angle glaucoma, cardiac conduction disorders, and benign prostatic hypertrophy. Although TATCAs are inexpensive, the increased monitoring they require for dose titration and adverse reaction or drug interaction management may offset any savings. Obtain blood levels of these drugs early in treatment to see if the patient is a rapid or slow TCA metabolizer.

The NSRIs are TCAs with less antihistaminic and anticholinergic effects than TATCAs. They produce antidepressant effects by blocking the norepinephrine uptake pump, and, thereby, function as indirect norepinephrine agonists. Adverse effects include increased blood pressure, tachycardia, tremors, sweating, and anxiety. Discontinuation of NSRIs secondary to adverse effects compares to that of TATCAs users. These drugs can be fatal in overdose. NSRIs dangerously interact with fewer drugs than TATCAs, but potentiate other norepinephrine agonists. NSRIs benefit 50% of those who fail to get relief from SSRIs. Carefully use them with SSRIs for patients who partially respond.[8]

Atypical Therapies

The atypical antidepressant venlafaxine (Effexor) behaves like a dual SSRI/NSRI. SSRI effects and adverse effects predominate at low doses, while NSRI effects and adverse effects prevail at higher doses. Tolerability compares to the SSRIs. Patients



ANTIDEPRESSANTS fall into four classes, according to mechanism of action. You may initiate pharmacological treatment with any nonmonoamine oxidase inhibitor antidepressant. The primary reason for antidepressant treatment failure is drug nonadherence. In most cases, patients stop taking antidepressants because of troublesome adverse effects. Although most antidepressants are equally effective in reversing depression in outpatients, selective serotonin reuptake inhibitors have become the initial drug of choice.

may start taking venlafaxine at an effective dosage. It has a wide therapeutic index and minimal effects on the cytochrome P450 isoenzymes. Its efficacy equals other antidepressants, and one study even found it exceeds SSRIs.[21]

Nefazodone (Serzone), developed from trazodone to have less antihistaminic and more serotonin reuptake inhibitor qualities, is an alternative for patients troubled with SSRI adverse effects. Nefazodone doesn't disturb sleep architecture (it may even improve sleep) and it has minimal sexual adverse effects.[22] Nefazodone has a wide therapeutic index, and because it's sedating, it's particularly useful for anxious patients.[23]

Titrate up nefazadone to allow for tolerance to sedation, confusion, dizziness, and gastrointestinal adverse effects. It inhibits a cytochrome P450 enzyme. Variability of response to nefazodone is greater than with other antidepressants.[20]

Like nefazodone, mirtazapine (Remeron) is useful with anxious patients and those troubled by SSRI-induced sleep or sexual dysfunction. Mirtazapine is sedating and may interact with other drugs, but minimally effects cytochrome P450 enzymes. It has a wide therapeutic index, and patients may receive effective doses immediately. Mirtazapine may cause weight gain and, rarely, agranulocytosis.[8]

Bupropion (Wellbutrin, Zyban) is particularly useful for treating depressed patients with psychomotor retardation. It doesn't have any sexual adverse effects and doesn't disturb sleep physiology. It may cause seizures, particularly at higher doses. For this reason, divide its daily dosage, and make sure patients don't take doses closer than 4 hours apart.[8]

Monoamine Oxidase Inhibitors

Monoamine oxidase inhibitors (MAOIs) are inexpensive and useful in some patients with refractory depression.[15] These drugs, however, may be fatal in overdose or in interaction with other drugs. Refer MAOI candidates to a mental health specialist for treatment initiation.[9] The main adverse reaction of MAOIs is hypotension. Hypertension or serotonin syndrome may result from drug interaction.[8]

■ Following Up

Because of the potential sequelae of depression and adverse effects of treatment, close follow-up is crucial. In addition to implementing nonpharmacologic interventions, consider the pharmacologic treatment algorithm in the Figure, "Pharmacologic Treatment of Depression."[1,11] At 1 to 2 weeks, assess for adverse drug effects and suicide risk, and educate and support the patient at this and all subsequent visits.[8]

A patient interview or scales, such as the CES-D, BDI, or SDS, may help gauge response at 4 to 6 weeks. Patients who are somewhat better should show a reduction in depression severity, while those who respond to drug therapy shouldn't score in the depressive range on standardized scales.[8] Complete drug titration to therapeutic levels by 4 weeks after starting treatment.

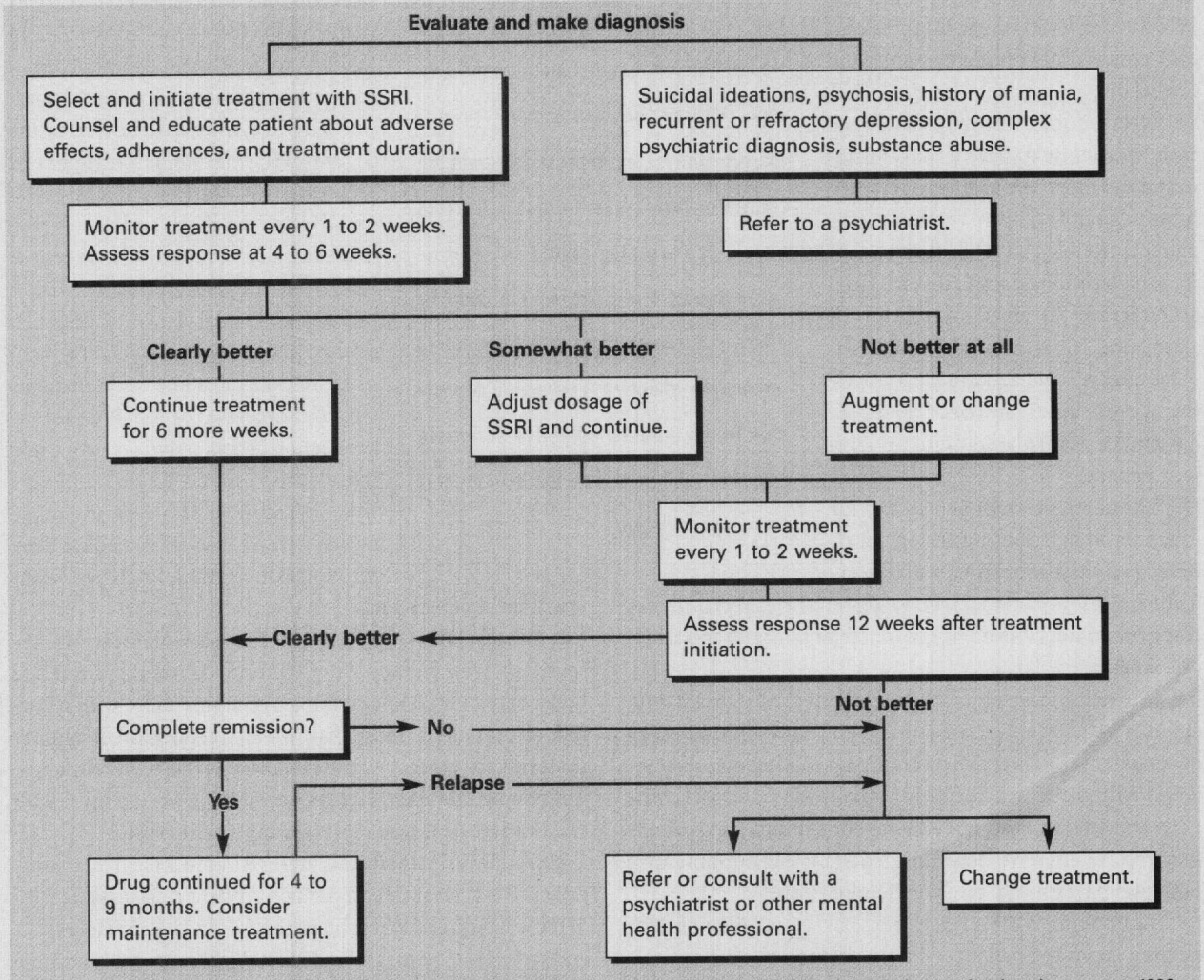
Initially, follow-up every 1 to 2 weeks with those who respond to drug therapy. After 6 months of therapy, evaluate the patient every 3 to 6 months.[9] Maintain those with recurrent or prolonged (more than 2 years) depression on antidepressants for at least 12 months, and perhaps indefinitely.[15] The relapse rate after drug discontinuation among those with two or more depressive episodes approaches 90%.[8]

Switch patients who fail to respond to a particular antidepressant to an antidepressant of a different class. Some 50% of those who failed to respond to an SSRI responded to a NSRI and vice versa.[8] Some theorize that depression is a syndrome resulting from heterogeneous factors and that some cases will respond to drugs with focused activity like the SSRIs, while others respond to drugs like TCAs with broader activity.

Patients on TATCAs and the SSRIs may suffer withdrawal symptoms if they stop taking them abruptly. Anticholinergic withdrawal syndrome (from TATCAs) may present as headache, hypersalivation, loose stools, or urinary frequency. SSRI withdrawal may also present with headache and loose stools, but also dizziness/sensory disturbance, agitation, sleep disturbance, and malaise. These vague symptoms can easily confuse the clinical picture. SSRIs with longer half-lives are less likely to produce withdrawal symptoms, but more likely to interact with a replacement antidepressant. The SSRI with

Figure

Pharmacologic Treatment of Depression



Adapted from the Agency for Healthcare Research and Quality: Quick reference guide for clinicians, no. 5; Depression in primary care, 1993.

the longest half-life, fluoxetine (Prozac), is active as a metabolite with a half-life of 9 days after chronic administration.[20]

Patients who fail to respond to an adequate trial on a second antidepressant may try a third drug from a different class, or they may need referral for possible electroconvulsive therapy. Electroconvulsive therapy is the most effective treatment for major depression with a response rate of 80%.[17] Consider electroconvulsive therapy referral for patients who are psychotic or in a catatonic state, lack the capacity to cooperate with therapy, are suicidal or violent, are psychosocially isolated, require drug or alcohol detoxification, refuse food, have comorbid psychiatric or other illnesses that require hospitalization for safety, or are unresponsive to pharmacologic therapy.[9]

Making a Difference

With appropriate diagnosis and treatment, we can decrease or eliminate the symptoms of depression, prevent a relapse, and avoid adverse sequelae. Over the past decade, SSRIs have replaced TCAs as first-line treatment for depression. They have a safer overdose profile, less adverse effects, and an efficacy that equals TCAs. SSRIs help make primary care management of depression attainable. NP

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TEST RESULTS MUST BE POSTMARKED BY SEPTEMBER 30, 2004.

ANCC
2.0/1.0 Rx
Contact Hours

How to Diagnose and Treat Depression

Purpose: To improve nursing practice and the quality of care by providing a learning opportunity that enhances a participant's understanding of diagnosis and treatment of depression. **Objectives:** After reading the article and taking this test, you should be able to: 1. Describe depression, including prevalence, symptoms, differential diagnoses, screening tools, and suicide risks. 2. Identify nonpharmacologic treatment options for depression. 3. Provide four functional classes of antidepressants and identify advantages, disadvantages, and mechanisms of action.

1. Research indicates that primary care clinicians fail to recognize
1. 5% of depression cases.
2. 15% of depression cases.
3. 50% of depression cases.
4. 65% of depression cases.

2. Which diagnosis includes hallucinations, delusions, and other psychotic symptoms?
1. anxiety
2. dementia
3. depression
4. schizophrenia

3. SSRIs account for the following portion of antidepressants prescribed by clinicians:
1. 5%.
2. 15%.
3. 50%.
4. 65%.

4. Which of the following may indicate autonomic dysfunction?
1. myoclonus
2. restlessness
3. sweating
4. tremor

5. The following drug class is the second most commonly prescribed by clinicians:
1. atypical antidepressants.
2. NSRIs.
3. SSRIs.
4. tricyclic antidepressant tertiary amines (TATCAs).

6. The following drug class produces antidepressant effects by blocking the norepinephrine uptake pump, functioning as indirect norepinephrine agonists:
1. atypical antidepressants
2. NSRIs
3. SSRIs
4. TATCAs

7. Which antidepressant doesn't have sexual adverse effects and doesn't disturb sleep physiology, but may cause seizures?
1. bupropion
2. mirtazapine
3. nefazodone
4. venlafaxine

8. Patients taking MAOIs may experience
1. hypertension.
2. hypotension.
3. nervousness.
4. sweating.

9. Patients with two or more depressive episodes who discontinue pharmacologic therapy have a depression relapse rate of
1. 20%.
2. 40%.
3. 70%.
4. 90%.

10. With a response rate of 80%, the most effective treatment for major depression is
1. antidepressant drug therapy.
2. electroconvulsive therapy.
3. exercise.
4. psychotherapy.

11. Well-motivated patients with insight into their problems can expect to attend hourly psychotherapy sessions every
1. week for 1 to 10 weeks.
2. 4 to 6 weeks.
3. 3 to 6 months.
4. 12 months.

12. Patients with severe forms of major depression have a
1. 5% risk of death from suicide.
2. 15% risk of death from suicide.
3. 50% risk of death from suicide.
4. 80% risk of death from suicide.

13. Patients with major depressive disorder may experience the following symptom nearly every day:
1. anorgasmia.
2. fatigue.
3. rhabdomylosis.
4. urinary retention.

14. A white, 65-year-old, widowed man has a high risk of
1. personality disorder.
2. schizoaffective disorder.
3. somatoform disorder.
4. suicide.

15. The peak age for depression onset is
1. before age 7.
2. between ages 13 to 19.
3. between ages 20 to 40.
4. older than age 65.

Carefully cut along dotted line.

Evaluation Listed below are statements about the CE offering. Please circle the number that best indicates your response.

	Disagree			Agree
1. I met objective 1.	1	2	3	4
2. I met objective 2.	1	2	3	4
3. I met objective 3.	1	2	3	4
4. The objectives related to the purpose of the activity.	1	2	3	4
5. The learning method was effective for me.	1	2	3	4

6. It took_____(hrs.) _____(mins.) to read and review the article, and take the test.

Any licensed nurse may submit this evaluation form directly to the Iowa Board of Nursing.

Name _____

Address _____ ☐ New

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(One of the above numbers is required in order to process this test.)

Phone Number (home) _____ (work) _____

☐ Fax my certificate to (fax number) _____ ☐ home ☐ work

☐ Visa

☐ MasterCard No. _____ Exp. Date _____
(Credit card must be used for faxed tests. Payment must accompany the tests.)

How to Diagnose and Treat Depression

1.	2.	3.	4.	5.	6.	7.	8.
☐1.	☐1.	☐1.	☐1.	☐1.	☐1.	☐1.	☐1.
☐2.	☐2.	☐2.	☐2.	☐2.	☐2.	☐2.	☐2.
☐3.	☐3.	☐3.	☐3.	☐3.	☐3.	☐3.	☐3.
☐4.	☐4.	☐4.	☐4.	☐4.	☐4.	☐4.	☐4.

9.	10.	11.	12.	13.	14.	15.
☐1.	☐1.	☐1.	☐1.	☐1.	☐1.	☐1.
☐2.	☐2.	☐2.	☐2.	☐2.	☐2.	☐2.
☐3.	☐3.	☐3.	☐3.	☐3.	☐3.	☐3.
☐4.	☐4.	☐4.	☐4.	☐4.	☐4.	☐4.

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℞ Of the 2.0 ANCC contact hours awarded for this activity, 1.0 is applicable toward a pharmacology requirement.

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