Recognition and Treatment of Anxiety Disorders
In the Primary Care Setting

Anxiety disorders are a diverse set of syndromes characterized by different clinical features. Most begin in childhood, adolescence, or early adulthood and continue throughout adulthood. There are 11 anxiety disorders classified in the Diagnostic and Statistical Manual of Mental Disorders, 4th edition. Text Revision (DSM-IV-TR). The anxiety disorders that are observed most often in the primary care setting are social anxiety disorder (SAD)/social phobia, generalized anxiety disorder (GAD), panic disorder.

Epidemiology and Economic Impact of Anxiety Disorders

According to the National Comorbidity Survey Replication, the lifetime prevalence of any anxiety disorder is approximately 28.8%. Additional data from this survey, which estimated the 12-month prevalence, severity, and comorbidity of DSMIV-TR disorders, showed that anxiety disorders are the most prevalent class of disorders (18.1%),
followed by mood disorders (9.5%), impulse control disorders (8.9%), and substance disorders (3.8%). The prevalence of the anxiety disorders combined actually exceeds 10% of the population. Among individuals with SAD, the annual cost of anxiety disorders to be approximately $42.3 billion in the United States, or $1,542 per sufferer. Work impairment is one of the most common and costly consequences of anxiety disorders.

**Pathophysiology of Anxiety Disorders**

Anxiety disorders appear to result from an interaction between biological factors and external situations, stress, or trauma to produce clinically significant symptoms. The pathophysiologic mechanisms underlying anxiety disorders are complex and involve multiple neurotransmitter systems, the amygdala and the hippocampus oper at a level beyond what is adaptive for modern society. As a result of the output from these areas of the brain, activation of corticotropin-releasing factor and cortisol occurs.

The fact that SAD often precedes other anxiety disorders is now being challenged by more sophisticated models that are known to provoke anxiety and/or panic on challenges. The amygdala and the hippocampus operate at a level beyond what is adaptive for modern society. As a result of the output from these areas of the brain, activation of corticotropin-releasing factor and cortisol occurs.

The noradrenergic theory of anxiety contends that increased noradrenergic release leads to anxiety via excess or dysfunctional arousal. Serotonin (5-HT) has been implicated in the neurotransmission of anxiety for many years, but interest in the role of the serotonergic system in anxiety disorders has increased during the last decade. The relationship between anxiety and 5-HT is indeed complex; the classic theory that anxiety is secondary to excessive 5-HT activity is now being challenged by more sophisticated models that place emphasis on different serotonergic neural circuitry and receptors mediating different aspects of anxiety.

**Presentations of Anxiety Disorders in Primary Care**

The presentations of anxiety in primary care are more frequent and broader in spectrum than the presentations of depression, challenging the primary care physician (PCP) to find a diagnosis from a full spectrum of anxiety disorders. Person with anxiety disorders often experience physical symptoms and distress and thus present to their PCP with somatic complaints, which may temporarily distract the clinician from the underlying symptoms of anxiety. Three frequent modes of presentation of anxiety disorders in primary care are the following:

- **Psychiatric:** In a less common presentation, patients consider their symptoms to be emotional in origin.
- **Somatic:** This is the most common presentation and includes a variety of somatic complaints, such as pain syndromes, headaches, fatigue, irritability, sweating, sleep disturbance, and gastrointestinal symptoms.

**Figure 1. Relationship between common physical symptoms and psychiatric disorders in primary care patients.**

**Figure 2. One-month and lifetime DSM-III-R diagnoses of distressed high utilizers.**
Comorbidity and Anxiety Disorders

Comorbidity is the rule rather than the exception in anxiety and depressive disorders. Anxiety disorders are often comorbid with other psychiatric disorders and with one another. Moreover, anxiety disorders frequently coexist with chronic medical conditions such as cardiovascular disease and diabetes mellitus. The coexistence of anxiety disorders and major depressive disorders is associated with barriers to treatment, including treatment resistance, increased risk for suicide, greater chance for recurrence, greater utilization of medical resources, and worse psychiatric outcomes.

The long-term clinical course of anxiety disorders and the influence of comorbid psychiatric disorders on recovery or recurrence of panic disorder, GAD, and social phobia have been examined in a prospective, naturalistic, longitudinal study conducted over 12 years. Of 711 patients assessed at intake, 473 remained in the study at the 12-year follow-up. Patients were evaluated at 6-month intervals during the first 2 years, annually during years 3 to 6, and every 6 months during years 7 to 12. Patients were considered to have recovered from an anxiety disorder if they exhibited psychiatric status ratings of 2 or less on a rating scale of 1 to 6 (1, without any residual symptoms of disorder; 2, presence of symptoms of no more than mild degree) for 6 consecutive weeks. Twelve years after study entry, estimates showed that patients with panic disorder without agoraphobia were likely to have had a recovery at all time points (recovery probability, 0.82), whereas the recovery probabilities during 12 years of follow-up were much lower in patients with GAD, panic disorder with agoraphobia, or social phobia (0.58, 0.48, and 0.37, respectively; Figure 3). A higher probability of recovery from major depressive disorder than from any of the anxiety disorders was observed, with the exception of panic disorder without agoraphobia. In terms of comorbidity, investigators found that patients with comorbid major depressive disorder were half as likely to subsequently recover from GAD or panic disorder with agoraphobia (P<0.01) as patients without comorbid major depressive disorder.

Clinical Symptoms and Diagnostic Features Of Anxiety Disorders Seen in Primary Care

The PCP plays a significant role in the initial screening and diagnosis of anxiety disorders. Some of the challenges to the detection of anxiety disorders by PCPs include the DSMV terminology, patient resistance to diagnostic test procedures, translating the symptoms into a diagnosis, and eliminating organic possibilities. For a patient with symptoms of anxiety to be given a proper diagnosis, the DSMV-TR criteria must be met. The diagnosis is often complicated by the simultaneous presence of another psychiatric disorder, such as depression or substance abuse. Because several anxiety disorders have overlapping signs and symptoms, it is important for clinicians to use several lines of questioning to determine the primary diagnosis. Obtaining a good medical and psychiatric history is fundamental to the diagnosis of an anxiety disorder. Medical disorders that may be contributing to the distress must be ruled out, in addition to substance abuse or dependence.

Social Anxiety Disorder

SAD, also referred to as social phobia, is the anxiety disorder that presents most often in the primary care setting. SAD is characterized by a marked and persistent fear of social or performance situations in which embarrassment may occur. Common associated features of SAD include hypersensitivity to criticism, negative evaluation or rejection, difficulty being assertive, and low self-esteem or feelings of inferiority. Associated features may include poor social skills (eg, poor eye contact) and observable signs of anxiety (eg, cold clammy hands, tremors, shaky voice). A diagnosis of SAD is appropriate only if an individual’s normal routines, occupational functioning, or social activities or relationships are significantly impaired as a result of such fears (Table 1).

### Table 1. Diagnostic Criteria for Social Anxiety Disorder

- Patient experiences extreme anxiety in social or performance situations
- Individual recognizes that the fear is excessive or unreasonable
- The feared social or performance situations are avoided or else are endured with intense anxiety or distress
- Individual finds that normal routines, occupational functioning, or social activities or relationships are significantly impaired as a result of his/her fears

### Table 2. Diagnostic Criteria for Generalized Anxiety Disorder

- Anxiety and worry are out of proportion to likelihood of feared event
- Patient finds anxiety difficult to control
- Symptoms occur more days than not for at least 6 months
- Adults experience 3, or children experience 1, of the following:
  - restless, keyed up, or feeling on edge
  - easily fatigued
  - difficulty concentrating
  - irritable
  - muscle tension
  - sleep disturbance
Generalized Anxiety Disorder

GAD is characterized by chronic, excessive, uncontrollable worry and anxiety about everyday issues (eg, health, money, family, work) that are not provoked by any specific event. Although persons with GAD generally realize that their anxiety is more intense than the situation warrants, they find it difficult to control their worry and concerns. Constant worry affects daily functioning and frequently manifests as physical complaints. Persons with GAD are unable to relax, startle easily, have difficulty concentrating, and often have trouble falling asleep or staying asleep. GAD does not characterize result in avoidance of certain situations, and the individual is usually able to function in social settings or on the job if the impairment is mild. However, persons with severe GAD may have difficulty carrying out the simplest daily activities. Individuals with GAD generally present to their PCPs when the anxiety peaks and they can no longer cope. As many as 83% of patients present initially to their PCP with general, vague, or nonspecific somatic complaints, such as pain syndromes, symptoms of autonomic arousal (chest pain, palpitations, hyperventilation), headache, fatigue, insomnia, and gastrointestinal symptoms. The diagnostic criteria for GAD are shown in Table 2.1

Panic Disorder

Recurrent, unexpected panic attacks—and discrete periods of intense fear or discomfort in the absence of real danger—are the essential features of panic disorder. A panic attack has a rapid onset and generally lasts 10 to 15 minutes, and it may produce a sense of unreality, a fear of impending doom, or a fear of losing control. Physical symptoms such as a pounding heart, sweating, weakness, faintness, and dizziness often accompany a panic attack. By itself, a panic attack is not considered a mental disorder and does not necessarily constitute panic disorder. Panic disorder is differentiated from single panic attacks by the anticipatory anxiety that one will have future panic attacks and the restriction of function that results from that concern. The diagnostic criteria for panic disorder are shown in Table 3.1

Anxiety Secondary to Organic Causes

Anxiety secondary to an organic cause should be considered in patients in whom symptoms begin after age 35 and in whom the following are lacking: a personal or family history of anxiety disorder; a childhood history of anxiety, phobia, or separation anxiety; a significant life event that would precipitate an anxiety disorder; avoidance behavior. An organic cause should also be considered in patients who fail to respond to psychiatric treatment. Frequently, individuals with an organic cause of anxiety deny a history of or a tendency to have feelings of anxiety or an anxiety disorder. In such cases, the PCP should look beyond a psychiatric diagnosis to rule out other possible origins of the anxiety, such as hyperthyroidism, undiagnosed cancer, a cardiac event, or any number of other medical conditions.

Treatment of Anxiety Disorders

In general, 2 types of therapy—pharmacotherapy and psychotherapy—are used, either alone or in combination, for the treatment of anxiety disorders. Pharmacologic agents allow patients with anxiety disorders to bring their symptoms under control, whereas some forms of psychotherapy help patients to explore the beliefs or behaviors that maintain their anxiety disorder. Several factors increase the effectiveness of treatment, including:

- patient compliance
- absence of comorbid conditions
- continuation of treatment to prevent relapse
- ease of use
- patient education.

When the clinician chooses an appropriate treatment for anxiety, the patient must first be evaluated for the presence of a comorbid condition. The appropriateness of the treatment for the individual patient should be considered. For example, some patients with severe anxiety may require the rapid relief afforded by benzodiazepines, whereas patients with a substance abuse problem may require a selective serotonin reuptake inhibitor (SSRI). Some clinicians will escalate the dose when a response is not achieved. The duration of treatment should be extended beyond the point of symptom relief to prevent relapse. The International Consensus Group on Depression and Anxiety recommends an 8-week initial treatment phase to establish efficacy and response, followed by a continuation phase of 1 year or more to achieve remission.20

Cognitive Behavioral Therapy

Of the various forms of psychotherapy, cognitive behavioral therapy (CBT) is perhaps the one for which the most evidence is available to support its effectiveness for anxiety disorders. The cognitive component of CBT helps to control unrealistic or negative patterns of thinking that contribute to anxiety and prevent the patient from overcoming his or her fears. In the behavioral component of CBT, the event or fear is recalled or experienced in a safe, controlled situation to help defuse the anxiety if it provokes. CBT is useful alone and can be a valid first-line treatment for anxiety disorders, or it may be used in combination with medication for refractory symptoms; persistent cognitive factors, behavioral patterns, and anxiety sensitivity; and comorbid conditions. CBT may facilitate the discontinuation of anxiolytic medication.

Pharmacotherapy

The drug classes that have been found to be effective for the treatment of anxiety disorders include the benzodiazepines, buspirone, serotonin norepinephrine reuptake inhibitors (SNRIs), and SSRIs. Drugs with an FDA-approved indication for the treatment of anxiety are shown in Table 4.

Benzodiazepines

Benzodiazepines have been used extensively for the treatment of anxiety disorders since the 1960s; newer benzodiazepine formulations, such as extended-release tablets and orally disintegrating tablets, offer alternative dosing and delivery options. As anxiolytics, benzodiazepines potentiate the inhibitory effects of GABA through their action on the GABA-A receptor.21 Benzodiazepines are generally considered effective and have other advantages, although this class also has a number of drawbacks that need to be considered (Table 5).22

The long-term use of benzodiazepines has raised concerns about the development of therapeutic tolerance, although results of follow-up studies do not suggest a significant loss of therapeutic anxiolytic effect over time or provide evidence for escalation of dose.21,22 A study of 2,440 New Jersey Medicaid patients examined the long-term use of benzodiazepines with a focus on dose escalation (an increase to >40 mg of diazepam per day or its equivalent or to 20 mg of diazepam per day in the elderly over a 2-year period).21 Investigators observed that the dose was increased above the prespecified level of concern in 1.6% of patients. For the majority of patients, the doses tended to remain stable over time, with the exception of those treated concomitantly with antidepressants and those taking duplicate prescriptions for benzodiazepines at different pharmacies. Nagy and colleagues22 studied 60 patients with panic attacks or panic disorder who completed a 4-month combined program of alprazolam and behavior therapy and were interviewed 1.7 to 4 years after discharge. At follow-up, 60% of patients were taking alprazolam at a lower dose, 30% had discontinued alprazolam therapy altogether, and 5% had increased their dose. The abuse of benzodiazepines can be divided into 2 patterns: intentional abuse by persons who use drugs for their euphoriant effects, and unintentional abuse by patients who begin using benzodiazepines to treat an anxiety disorder and then use them inappropriately.22 Intentional abusers generally take high doses of benzodiazepines and engage in the abuse of multiple drugs and alcohol. Most unintentional abusers are unaware of their benzodiazepine dependence until they try to abruptly discontinue the drugs.

Table 3. Diagnostic Criteria for Panic Disorder

<table>
<thead>
<tr>
<th>Recurrent unexpected panic attacks</th>
</tr>
</thead>
<tbody>
<tr>
<td>At least one of the attacks has followed by 1 month (or more) of one (or more) of the following:</td>
</tr>
<tr>
<td>Persistent concern about having additional attacks</td>
</tr>
<tr>
<td>Worry about the implications of the attack or its consequences</td>
</tr>
<tr>
<td>A significant change in behavior related to the attacks</td>
</tr>
<tr>
<td>The panic attacks are not due to the direct physiologic effects of a substance (eg, a drug of abuse, a medication) or a general medical condition (eg, hyperthyroidism)</td>
</tr>
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</table>

Table 4. Drugs With FDA-Approved Indications for Anxiety Disorder

<table>
<thead>
<tr>
<th>Benzodiazepines</th>
<th>Panic</th>
<th>GAD</th>
<th>SAD</th>
<th>PTSD</th>
<th>OCD</th>
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<td>✓</td>
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<td>✓</td>
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<td>✓</td>
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<tr>
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</tr>
<tr>
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</tr>
<tr>
<td>Paroxetine CR</td>
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<td>✓</td>
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<td>✓</td>
</tr>
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<td>SNRI</td>
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<td>✓</td>
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<tr>
<td>Other</td>
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<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
</tr>
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</table>

CR, controlled release; GAD, generalized anxiety disorder; ODD, obsessive compulsive disorder; ODT, orally disintegrating tablet; PTSD, post-traumatic stress disorder; SAD, social anxiety disorder; SNRI, serotonin norepinephrine reuptake inhibitor; SSR, selective serotonin reuptake inhibitor; XR, extended release.
To help patients avoid withdrawal symptoms when it is time to discontinue a benzodiazepine, the drug must be tapered very gradually. When withdrawing patients from a benzodiazepine, clinicians must observe carefully for the following: (1) recurrence of the anxiety symptoms being treated, (2) a temporary rebound in the number of anxiety attacks (eg, panic), (3) withdrawal symptoms associated with abrupt discontinuation, and (4) pseudowithdrawal due to a psychological dependence on the medication. It is important to keep in mind that if the taper involves a fixed-dose decrease, the percentage of the drug dose being removed increases as lower doses are reached. Therefore, as lower doses are approached, a smaller amount should be removed with each step of the taper. During the process of benzodiazepine discontinuation, CBT can be valuable in helping patients transition off the medication.

Buspirone

After the benzodiazepines, buspirone, classified as an azapiron, was the first agent to gain FDA approval for the treatment of GAD. Buspirone is classified as a 5-HT1A agonist; although its mechanism of action is rather complex and not completely understood, its main effects are mediated by the serotonin-1A (5-HT1A) receptors. Buspirone is an agonist primarily at postsynaptic 5-HT1 receptors and is a partial agonist at postsynaptic 5-HT1 receptors in several brain regions involved in stress, fear, and anxiety.24,25 Additionally, buspirone acts as a dopamine agonist, possessing a weak affinity for both dopamine D1 and D2 receptor subtypes. Buspirone has been demonstrated to have efficacy in the treatment of GAD, but not in other anxiety disorders or depression. Buspirone has a benign side-effect profile, does not potentiate alcohol or have any potential for abuse, and is relatively safe in overdose (Table 5).25 Buspirone does not induce sedation, psychomotor or cognitive impairment, or physical dependence or tolerance, and it does not interact with alcohol. The 3- to 4-week lag before the onset of anxiolytic activity limits the utility of buspirone in patients requiring intervention for acute anxiety.

SSRIs and SNRIs

Before the discovery and approval of the SSRIs, 2 other classes of antidepressants, the tricyclic antidepressants (TCAs) and the monoamine oxidase inhibitors (MAOIs), were sometimes prescribed for the treatment of anxiety disorders. However, certain side effects associated with TCAs (eg, drowsiness, dry mouth, weight gain) and MAOIs (eg, potentially severe interactions with certain medications and foods), in addition to their potential lethality in overdose, have made the SSRIs more desirable when antidepressants are indicated for the treatment of anxiety disorders. The efficacy of the SSRIs in the treatment of depression has been well established in numerous controlled clinical trials and through clinical experience. The SSRIs and SNRIs have well-documented efficacy in the treatment of depression, and are effective in alleviating symptoms of anxiety. Their drawbacks include delayed onset of benefit, side effects such as sexual dysfunction, and lack of utility as agents that can be used on an as-needed basis (Table 5).25 Some SSRIs and SNRIs have a high potential for drug-drug interactions, and cost may also be a concern. Although the advantages of SSRIs in treating anxiety disorders are not as well documented as their clinical utility in treating depression,24 most agents in this class now have FDA approval for several anxiety disorders (Table 4). Buspirone, while effective for depression, is not efficacious as monotherapy for anxiety disorders, and it may even exacerbate agitation.

The SSRIs have a common mechanism of action, although they differ considerably in chemical structure, metabolism, and pharmacokinetics. Perhaps the most important way in which the drugs in this class differ is in their potential to cause drug-drug interactions through the inhibition of cytochrome P-450 isoenzymes (ie, CYP1A2, CYP2C9, CYP2D6, CYP3A4), which are responsible for the oxidation and metabolism of numerous substances and drugs.26 Because of the chronic nature of anxiety disorders and depression, most patients require long-term treatment, which increases the likelihood that SSRIs will be prescribed together with other medications. Thus, the potential for clinically significant drug-drug interactions, which may be pharmacokinetic or pharmacodynamic, is considerable. An understanding of the cytochrome P-450 isoenzymes involved in the metabolism of the SSRIs and coadministered drugs can help the PCP anticipate and avoid potentially dangerous drug-drug interactions. As shown in Table 6, citalopram is associated with minimal or no inhibition of CYP2C9, CYP2C19, CYP2D6, and CYP3A4 and only mild inhibition of CYP1A2; escitalopram has little or no effect on any of the enzymes, so that its potential to cause drug-drug interactions is limited.27,28 Fluvoxamine is a potent inhibitor of CYP1A2 and CYP2C19 and a mild inhibitor of CYP2C9. CYP2D6, CYP3A4. Fluoxetine and paroxetine are potent inhibitors of CYP2D6, whereas sertraline is a mild inhibitor of CYP2D6. Because of the long half-life of fluoxetine and its metabolite norfluoxetine, its inhibitory effects on cytochrome P-450 activity can persist for several weeks after discontinuation.

Treatment of Social Anxiety Disorder

As noted in Table 4, 2 SSRIs (paroxetine and sertraline) and the SNRI venlafaxine are approved for the treatment of SAD. Double-blind, randomized, placebo-controlled studies have demonstrated the efficacy of these compounds for the treatment of SAD.29,30 Patients treated with paroxetine, sertraline, or venlafaxine over 12-week study periods report significant decreases in SAD symptoms as assessed with the Clinical Global Impression Improvement (CGI-I) Scale and/or the Liebowitz Social Anxiety Scale (LSAS) in comparisons with patients given placebo.

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Table 5. Benefits and Drawbacks of Anxiolytic Agents20,25

<table>
<thead>
<tr>
<th>Benefits</th>
<th>Drawbacks</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Benzodiazepines</strong></td>
<td>Sedation, decreased reaction time, cognitive impairment</td>
</tr>
<tr>
<td>Effective</td>
<td>Discontinuation difficulties</td>
</tr>
<tr>
<td>Short response latency</td>
<td>Potential for abuse in predisposed individuals</td>
</tr>
<tr>
<td>Well tolerated; high level of patient acceptance</td>
<td>Interaction with alcohol</td>
</tr>
<tr>
<td>Rapid dose adjustment feasible</td>
<td>Not effective for comorbid depression</td>
</tr>
<tr>
<td>Can be used PRN</td>
<td><strong>Buspirone</strong></td>
</tr>
<tr>
<td>Reduce antidepressant-induced activation</td>
<td>Delayed onset of benefit</td>
</tr>
<tr>
<td><strong>Drawbacks</strong></td>
<td>Dosing 2 or 3 times daily</td>
</tr>
<tr>
<td><strong>Buspirone</strong></td>
<td>Limited spectrum of effect</td>
</tr>
<tr>
<td><strong>SSRIs/SNRIs</strong></td>
<td>Drug interactions (CYP3A4 substrate)</td>
</tr>
<tr>
<td>Well-documented efficacy in major depression</td>
<td><strong>Drawbacks</strong></td>
</tr>
<tr>
<td>Effective in alleviating anxiety symptoms</td>
<td>Delayed onset of benefit</td>
</tr>
<tr>
<td>Some have multiple indications for anxiety disorders</td>
<td>Cost</td>
</tr>
</tbody>
</table>

Table 6. In Vitro Inhibition of Cytochrome P-450 Enzymes by SSRIs

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>Citalopram</th>
<th>Escitalopram</th>
<th>Fluoxetine</th>
<th>Fluvoxamine</th>
<th>Paroxetine</th>
<th>Sertraline</th>
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<tbody>
<tr>
<td>1A2</td>
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</table>

**CYP, cytochrome P-450 enzyme; GAD, generalized anxiety disorder; PRN, as needed; SNRI, serotonin–norepinephrine reuptake inhibitor; SSRI, selective serotonin reuptake inhibitor.**
rapid and sustained improvement, whereas buspirone produced a more gradual effect. More recently, the newer SSRI escitalopram was evaluated for the treatment of GAD in 315 outpatients who were randomized in double-blind fashion to treatment with escitalopram at a dosage of 10 mg per day or placebo.38 Patients treated with escitalopram showed a statistically significant improvement on all efficacy parameters compared with patients given placebo. Response rates at week 6 were 68% for escitalopram and 41% for placebo (P<0.01).

**Conclusion**

Anxiety disorders typically begin early in life, tend to be chronic and unremitting, and are often comorbid with depression and substance abuse. Persons with anxiety disorders are high utilizers of health services in the primary care setting and frequently present to their PCP with myriad somatic complaints. The main goal of treatment is to achieve remission. The 2 forms of therapy used for the treatment of anxiety disorders are pharmacotherapy, which treats the symptoms of anxiety, and CBT, a form of psychotherapy that helps patients explore and control behaviors that sustain the disorder. Drug classes that have been found to be effective for the treatment of anxiety disorders are the benzodiazepines, the azapirone buspirone, and the SSRI s and SNRIs. Benzodiazepines are effective for GAD and panic disorder and are often used concomitantly with an antidepressant. Buspirone is effective for GAD. SSRI s and SNRIs are effective for various anxiety disorders and are of particular benefit in patients with comorbid depression.

**References**

8. Lebowitz JD. Fear and the brain: where have we been, and where are we going? Biol Psychiatry. 1989;26:1229-1238.
4. The rationale for adding a benzodiazepine to an SSRI
into memories is the ________
A. cerebellum
B. hippocampus
C. hypothalamus
D. amygdala

2. All of the following are features of an anxiety disorder secondary to an organic cause except:
A. absence of significant life events generating anxiety symptoms
B. poor response to psychiatric treatment
C. personal or family history of an anxiety disorder
D. onset of anxiety symptoms after age 35 years

3. Each of the following is considered a potential drawback associated with the SSRIs except:
A. increased potential for nephrotoxicity
B. potential for drug–drug interactions (eg, cytochrome P-450 metabolism)
C. delayed onset of action
D. side effects (eg, sexual dysfunction)

4. The rationale for adding a benzodiazepine to an SSRI for the treatment of panic disorder is based on which of the following:
A. Antidepressants are associated with a lag time of several weeks before they achieve therapeutic effect.
B. Benzodiazepines have a rapid onset of action and may reduce panic attacks during initiation of an antidepressant.
C. B only
D. A and B

5. The SSRIs that strongly inhibit the CYP2D6 enzyme are ________.
A. sertraline and fluvoxamine
B. escitalopram and citalopram
C. paroxetine and fluoxetine
D. none of the above

6. Which of the following statements describe(s) the treatment of anxiety disorders most accurately?
A. Benzodiazepines must be tapered very gradually upon discontinuation to avoid withdrawal symptoms.
B. Antidepressants should be initiated at a low dose and titrated slowly.
C. Using a benzodiazepine to augment antidepressant therapy slows the treatment response and may enhance activating symptoms of SSRIs
d. A and B
E. A and C

7. Which of the following anxiety disorders presents most frequently in the primary care setting?
A. Social anxiety disorder
B. Posttraumatic stress disorder
C. Generalized anxiety disorder
D. Obsessive-compulsive disorder

8. Each of the following characterizes patients with GAD except:
A. They present to the primary care physician when anxiety peaks and they can no longer cope.
B. Constant worry affects daily functioning and frequently manifests as physical complaints.
C. Anxiety and worry are associated with the presence of at least 6 physical symptoms for 3 months or longer.
D. Despite the realization that their anxiety is more intense than the situation warrants, they find it difficult to control worry and concerns.

9. In a study that examined the association of long-term benzodiazepine use and dose escalation, what percentage of patients increased their dose over a 2-year period?
A. 10%
B. 2%>
C. 25%

10. Each of the following statements is true regarding the diagnosis of anxiety disorders except:
A. The symptoms of the various anxiety disorders are discrete and easily recognized.
B. The diagnosis is often complicated by comorbidity such as depression and/or substance abuse.
C. The DSM-IV-TR criteria must be met for a patient to be given a proper diagnosis.
D. Obtaining a good medical and psychiatric history is fundamental to the diagnosis of an anxiety disorder.