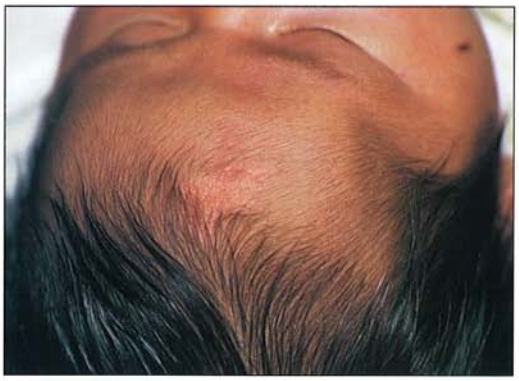

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Panic disorder: Debilitating, underdiagnosed, and treatable

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ABSTRACT Panic disorder is a chronic, recurring condition of acute episodes of panic attacks accompanied by a range of physical and psychological symptoms. Because these symptoms may mimic a variety of medical conditions, panic disorder often goes undiagnosed. The clinical picture is further clouded by the frequent psychological and medical comorbidities. Patients' use of all health care services is markedly increased, as they search for a diagnosis to account for their symptoms. Once diagnosed, panic disorder is highly treatable with pharmacotherapy, psychosocial treatments, or a combination of these. Appropriate management can reduce or eliminate panic attacks in 70%-90% of cases, but only about one third of patients receive appropriate treatment. Physicians need to hone their skills in recognizing and treating this common condition.

But the thing that made me so frightened, I think, was just not knowing what was wrong with me.—Patient Alicia

Panic disorder is a common condition in both the community and the primary care setting.[1,2] About 15% of the general population experiences panic attacks; 1.6%-3.2% of women and 0.4%-1.7% of men have panic disorder,[3] which will afflict 3 million American adults at some time during their life.[4] Peak age of onset is in young adulthood, at an average age of 25 years. It is more common in the less educated and in nonwhites. Coexisting agoraphobia is seen in 5.8% of women and 2.5% of men.[5]

Panic disorder is characterized by the occurrence of panic attacks—sudden, unexpected periods of intense fear or discomfort. The attacks are accompanied by dyspnea, dizziness, palpitations, nausea, or gastrointestinal distress. If undiagnosed, panic disorder can have deleterious effects on the patient's work, family, and social life.[6] Although rates of successful suicides are not increased in panic disorder, coexisting major depression substantially heightens suicide risk.

Because of the confusing mix of symptoms and frequent medical and psychiatric comorbidities, panic disorder is notoriously difficult to detect. But once the disorder is diagnosed,

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Table 1 Diagnostic criteria for panic disorder without agoraphobia*

Recurrent, unexpected panic attacks
AND
At least one attack has been followed by at least 1 month of one (or more) of the following:
- Persistent concern about experiencing more attacks
- Worry about the meaning of the attack or its consequences (fear of losing control, having a heart attack, or "going crazy")
- A significant behavioral change related to the attacks

AND
Absence of agoraphobia
AND
Direct physiological effects of a substance (drug abuse or medication) or general medical condition has been ruled out as a cause of the attacks
AND
The panic attacks cannot be better accounted for by another mental disorder

*Criteria for panic disorder with agoraphobia are identical to these except that they include "presence of agoraphobia" instead of "absence of agoraphobia."

Source: American Psychiatric Association. Anxiety disorders. In: Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, DC: American Psychiatric Association, Inc; 1994:393-444..

treatment can reduce or prevent the occurrence of acute attacks.[4] Since its prevalence in primary care centers has been reported as high as 11%, physicians need to recognize its characteristic features and be familiar with currently available treatment options.

Defining the disorder

Panic disorder affects three psychological spheres: motivational (for example, the wish to be as far from the traumatic situation as possible); affective (a subjective feeling of terror); and behavioral (inhibited speaking or thinking). Occasional panic attacks are common phenomena that most people cope with successfully. Panic disorder is distinguished by the frequency of the attacks, sudden onset of symptoms that occur without warning, and the absence of any causal connection to a precipitating event.

Panic attacks, as defined in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV),[7] are manifested by the sudden onset of an overwhelming fear, accompanied by feelings of impending doom, for no apparent reason (Table 1). Nonpathological fear, on the other hand, involves an agitated foreboding, often of some real or specific peril.

The essential criterion for panic attack is the presence of 4 of 13 cardiac, neurologic, gastrointestinal, or respiratory symptoms that develop abruptly and reach a peak within 10 minutes (Table 2). The physical symptoms are the result of autonomic nervous system hyperactivity, and most often include shortness of breath, dizziness or faintness, palpitations, accelerated heart rate, and sweating. Trembling, choking, nausea, numbness, flushes, chills, or chest discomfort are also common, as are cognitive symptoms such as fear of dying or losing control.

DSM-IV recognizes three types of panic attacks: unexpected (uncued), situationally bound (cued and more characteristic of social and specific phobias), and situationally predisposed. Attack-related persistent concern, worry, or behavioral changes is another criterion, along with inter-episode anticipatory anxiety—characterized by inhibition, fear of medical illness, role dysfunction, and feelings of helplessness or humiliation. The fear of an impending panic attack usually causes more distress and disability than the attack itself.

The diagnosis of panic disorder requires recurrent, full-blown discrete anxiety attacks, followed by at least 1 month of persistent fear of another attack. One third of patients develop agoraphobia,[8] or a fear of places where escape may be difficult, such as bridges, trains, buses, or crowded areas. Agoraphobia, which is manifested by phobic restriction of activity, is a long-term consequence of original or successive panic attacks. Its presence usually indicates a more severe form of panic disorder.

Quality of life and health care costs

Panic disorder is associated with substantial impairment of personal happiness and role

functioning. Patients have a self-perception of poor physical or emotional health[9] and a markedly increased use of all health services, much of which is inappropriate, unnecessary, and costly.[10] These patients also have higher rates of alcohol abuse, marital and financial problems, and medication use.[11]

Persons with panic disorder may visit their primary care physicians with complaints of cardiac, respiratory, gastrointestinal, and neurologic symptoms.[12] Medical symptoms typically persist and escalate until the panic disorder is adequately treated. Patients tend to self-medicate and search for a general medical solution to their problems.

Extensive diagnostic evaluations are costly, such as a complaint of chest pain that mandates an extensive workup. Better screening for panic disorder—especially in young women who have few cardiac risk factors—may reap substantial savings. Indeed, baseline panic disorder has been found the best predictor of continued functional disability from chest pain 2 years after negative cardiac testing.

Recognizing and evaluating patients at high risk for panic disorder thus becomes a cost-effective measure. When it is correctly diagnosed, appropriate treatment can reduce the direct and indirect costs of care, increase patients' work productivity, and improve their mental and physical quality of life.

Inherent challenges of the evaluation process

By definition, panic attacks are associated with a number of symptoms that may suggest other physical conditions. An average of 12-14 medical symptoms is not uncommon.[13] Medications, substance abuse, and general medical conditions such as hyperthyroidism must be ruled out as a cause of the patient's symptoms before a diagnosis of panic disorder can be made. Among patients commonly seen in primary care, a high risk of panic disorder appears to be associated with the following: 1) complaints of anxiety and tension; 2) a recent history of increased hypochondriacal concerns; 3) cardiac (tachycardia, chest pain), neurologic (headache, dizziness), or gastrointestinal (epigastric pain, irritable bowel syndrome) symptoms; and 4) disorders such as labile hypertension, mitral valve prolapse, migraine headaches, peptic ulcer disease, and asthma.

Table 2 Criteria for panic attack

A discrete period of intense fear or discomfort in which four or more of the following symptoms developed abruptly and reached a peak within 10 minutes.
Chest pain or discomfort
Choking
Depersonalization or derealization
Dizziness, faintness, or unsteadiness
Fear of "going crazy" or being out of control
Fear of dying
Flushes or chills
Nausea or gastrointestinal distress
Palpitations or tachycardia
Paresthesias
Shortness of breath (or feelings of smothering)
Sweating
Trembling or shaking

Source: American Psychiatric Association. Anxiety disorders. In: Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, DC: American Psychiatric Association, Inc; 1994:393-444.

Patients often experience a cognitive reaction that has been referred to as catastrophic misinterpretation. Along with self-consciousness and hypervigilance, patients describe an overwhelming sense of terror or a fear of losing control, dying, or going crazy. This is sometimes accompanied by frightening visual images. The mind is hazy, objects are blurred and distant, and the environment seems unreal.

Affectively, patients refer to a panic attack as the most terrifying event they ever experienced, one they will do almost anything to avoid repeating. As a result, they are edgy, impatient, wound-up, jittery, and jumpy. Common behavioral responses include the urge to flee the setting of the attack, seek help, or freeze up. Many have sought emergency medical help,

Panic disorder: The historical perspective

Written reports of panic attacks date back to at least the 17th century. The phenomenon was described by Robert Burton in his Anatomy of Melancholy[1]: "this fear causeth in Man as to be red, pale, tremble, sweat." It was not until 1871 that the concept of panic disorder appeared in the medical literature, when a cardiovascular syndrome that clearly resembled panic was documented among Civil War soldiers.[2]

Panic disorder became a subject of clinical research during the middle part of this century. In 1964, tricyclic antidepressants (TCAs) were proven effective in blocking panic attacks, but not in relieving anticipatory anxiety.[3] The results of the first placebo-controlled clinical trial comparing TCAs and monoamine oxidase inhibitors for treating panic disorder were published in 1980.[4] Later that year, the disorder became part of official nomenclature in the Diagnostic and Statistical Manual of Mental Disorders, third edition (DSM-III).

1. Burton R. The Anatomy of Melancholy. New York, NY: Empire State; 1624.
2. DaCosta JM. On irritable heart: A functional form of cardiac disorder and its consequences. Am J Sci 1871;61:14-51.
3. Klein DF. Delineation of two drug-responsive anxiety syndromes. Psychopharmacologia 1964;17:397-408.
4. Sheehan DV, Ballenger J, Jacobsen C. Treatment of endogenous anxiety with phobic, hysterical and hypochondriacal symptoms. Arch Gen Psychiatry 1980;37:51-59.

convinced they are suffering from a catastrophic medical illness.

Since no laboratory findings are associated with the disorder,[7] diagnosis must rely on a careful history and brief physical examination. The history should include details of the panic attack, its onset and course, history of panic, and any treatment. Questioning about a family history of panic disorder, agoraphobia, hypochondriasis, or depression is important to determine if the patient is at increased risk. Because panic disorder may be triggered by marijuana or stimulants such as cocaine, a history of substance abuse must be identified. A careful medication history, including prescription, over-the-counter, and herbal preparations, is essential. The patient should be asked about stressful life events or problems in daily life that may have preceded onset of the disorder.

Finally, the physician should determine the extent of any avoidance behavior that has developed since symptom onset and ask about suicidal ideation, self-medication, or exacerbation of an existing medical disorder.

Common medical and psychiatric comorbidities

A strong causal association has been documented between panic disorder and cardiac morbidity and mortality.[14] Mitral valve prolapse is seen in as many as 30%-40% of patients with panic disorder,[15] and a Canadian study using DSM-IV criteria found that 12.5% of cardiac outpatients also had panic disorder.[16] When heart disease develops in already anxious patients, it may provide the stress that triggers the onset of panic attacks and panic disorder.[14] In turn, the disorder may worsen cardiac conditions by provoking increased heart rate and blood pressure. Ischemia can exacerbate panic symptoms by causing increased electrical discharge of the locus ceruleus. Treatment of the panic disorder may also relieve the cardiovascular symptoms.

Irritable bowel syndrome is also associated with significantly higher rates of panic disorder (28%) and agoraphobia (41%).[17] And panic disorder is diagnosed in 5.5% of young people with headaches.[18]

Comorbid psychopathology contributes to treatment resistance and symptom maintenance. Approximately 15% have problems with drug and alcohol abuse.[19] Rates of personality disorders (axis II pathology) in panic disorder—as determined by self-report questionnaires—are in the range of 40%-70%. Avoidant, borderline dependent, and histrionic disorders are the most common diagnoses.[19]

About 1 in 5 panic disorder patients suffers from another anxiety disorder; 30% have comorbid depression.[20] The efficacy of antidepressants in the treatment of anxiety disorders suggests that anxiety and depression may share a

Panic disorder: An elusive etiology

The pathophysiology of panic disorder is still uncertain,[1] but research suggests that both biological and psychological components are involved.[2] Family and twin studies have pointed to a genetic predisposition. And some believe the cause of panic may represent a neurochemical imbalance of serotonin, norepinephrine, and other neurotransmitters.[3]

• *Neurobiological theories* Dysregulation in certain fear-associated central nervous system (CNS) circuits may be associated with panic disorder. A 1979 animal study showed that direct stimulation of the nucleus locus ceruleus—which contains the cells of origin of noradrenergic projections to the cerebral cortex, limbic system, and brain stem—produced panic-like reactions and increased autonomic function in monkeys.[4] The authors concluded that abnormally high reactivity of this noradrenergically mediated "alarm response" may figure significantly in the etiology. Also of interest, the locus ceruleus normally atrophies with aging, possibly explaining the decreased prevalence of panic disorder among the elderly.

Serotonergic abnormalities have also been implicated in the genesis of the disorder. A major challenge to the noradrenergic hypothesis came with the discovery of the potent antipanic effects of selective serotonin reuptake inhibitors—drugs that have no direct effect on the noradrenergic system. Several studies have also documented responses to the serotonin agonist, mCPP (m-chlorophenylpiperazine), which binds to a variety of serotonin receptor subtypes, in persons with panic disorder. In one study, this agent precipitated panic attacks in 30% of healthy subjects.[5]

Patients with panic disorder often hyperventilate during an acute attack and are hypersensitive to the effects of inhaled carbon dioxide. Inhalation of carbon dioxide has been shown to increase firing of the locus ceruleus, and experimental depletion of serotonin produces hyperventilation and increased carbon dioxide sensitivity in animal brains.[6] Reduced serotonergic and increased noradrenergic activity in the CNS may thus explain the marked tendency to hyperventilation during a panic attack.

Finally, positron-emission tomography (PET) measurements have detected increased metabolic activity in the septohippocampal system of persons with lactate-induced panic attacks.[7] They also had abnormal hemispheric asymmetries of parahippocampal blood flow, blood volume, and oxygen metabolism in panic disorder patients.

• *Genetic susceptibility* An increased risk of developing the condition has been seen in first- and second-degree relatives of panic disorder patients. Studies have found a higher concordance in monozygotic twins than in dizygotic twins (5:1).[8] In female monozygotic twins, the rate of concordance is 30%-40%, demonstrating modest heritability.

• *Psychological theories* Panic disorder is characterized by an overactive cognitive schema that is continually structuring external or internal experiences as signs of danger. Panic is thus similar to the physiologic fight-or-flight reactions to threatening situations that can be summarized as "the four Fs"—fight, flight, freeze, or faint. Panic often involves reflexes designed to ward off potential hazards: eye-blink, gagging, coughing, bronchospasm, vomiting, and diarrhea.

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neurobiologic substrate. Since panic may serve as a prodrome to depression, panic disorder should be treated aggressively. Each of these conditions is more severe when concurrent than when either occurs alone.

The lengthy differential diagnosis

Disorders such as hyperthyroidism, hypoglycemia, congestive heart failure, cardiac arrhythmias, pheochromocytoma, audiovestibular dysfunctions, complex partial seizures, caffein-

Table 3 Differential diagnosis of panic disorder

Medical conditions
Audio-vestibular dysfunction
Caffeinism
Cardiac arrhythmias
Complex partial seizures
Congestive heart failure
Epilepsy
Hyperthyroidism
Hypoglycemia
Pheochromocytoma
Psychiatric conditions
Claustrophobia
Dissociative disorders
Generalized anxiety without panic
Severe depression
Simple phobia
Social phobias
Medications
Acute intoxication or withdrawal from alcohol, caffeine, or illicit drugs (amphetamines, cannabis, cocaine)

Sources: National Institutes of Health, National Institute of Mental Health; 1994. NIH Publication 94-3642.
Hale AS. ABC of mental health. Anxiety. BMJ 1997;314:1886-1889.

ism—as well as excessive use of bronchodilators or substance abuse—may produce panic-like symptoms and complicate the treatment of panic disorder[21] (Table 3).

Among the factors suggesting an organic pathology are: onset after age 40; absence of personal or family history of anxiety disorders, or significant life events that have triggered or worsened symptoms; lack of avoidance behavior; and poor response to known anti-panic agents. Treatment for these patients should include correction of the underlying medical condition and removal of any provocative treatment agents.

Management approaches

The recovery rate from episodes of panic disorder is generally lower than that from depression. A prospective, naturalistic, longitudinal study charting the course and outcome of panic-related disorders found 18% of panic disorder patients with agoraphobia and 43% without agoraphobia recovered, compared with 80% of patients with depression.[22]

Nevertheless, panic disorder is a treatable condition. Appropriate treatment can eliminate or reduce panic attacks in 70%-90% of patients, particularly when the condition is recognized early.[4] Recurrent attacks can also be treated effectively, but only about one third of patients receive appropriate treatment.[4] If inadequately treated, possible sequelae may include phobic avoidance, anticipatory anxiety, depression, or drug or alcohol abuse.

It is important to approach panic disorder patients with a calm, reassuring manner and keep in mind that familial factors may complicate management. A husband may fear that if his wife is cured of her symptoms, the marriage may break up, for example. Or a child who is the companion of the phobic parent may derive self-esteem from that role. Recruiting the family into treatment or considering family therapy is thus important.

Patients should be encouraged to reduce or eliminate caffeine consumption, including coffee and tea, cold medications, analgesics, and beverages with added caffeine. Alcohol use is a particularly insidious problem because patients may use drinking to alleviate the panic. Then, as the effects of alcohol wane, the patient may experience a panic rebound that precipitates even more drinking.

Research on the nature and treatment of panic disorder has expanded throughout the past decade, with the emergence of new pharmacologic strategies and multicomponent cognitive behavioral interventions. The fundamental tenet to guide treatment is collaboration. Dichotomization of disorders into biological and psychological components must be discarded in favor of a model that recognizes both as contributing factors.

Although patients usually respond quickly to appropriate therapy, some treatments may work better than others for certain patients. Thus close monitoring of the patient's response is indicated so that the treatment strategy can

Table 4 Pharmacologic treatment of panic disorder

Drug	Dosage range (mg/d) Initial	Therapeutic
SSRIs		
Fluoxetine HCl (Prozac)	5-10	10-60
Fluvoxamine maleate (Luvox)	25-50	25-300
Paroxetine HCl (Paxil)	10-20	20-50
Sertraline HCl (Zoloft)	25-50	50-200
Benzodiazepines		
Alprazolam (Xanax)	0.5*	1-4*
Clonazepam (Klonopin)	0.5†	1-4†
Diazepam (Valium)	2.0‡	2-20‡
Lorazepam (Ativan)	0.5†	1-4†
TCAs		
Amitriptyline HCl (Elavil)	10	10-300
Clomipramine HCl (Anafranil)	25	25-300
Desipramine HCl (Norpramin)	10	10-300
Imipramine HCl (Tofranil)	10	10-300
Nortriptyline HCl (Pamelor)	10	10-300
MAOIs		
Phenelzine sulfate (Nardil)	15	15-90
Tranylcypromine sulfate (Parnate)	10	10-30

*In divided doses, tid-qid
†In divided doses, bid-tid
‡In divided doses, bid
Key: MAOIs = monoamine oxidase inhibitors; SSRIs = selective serotonin reuptake inhibitors; TCAs = tricyclic antidepressants.

Sources: Muzina DJ, Malone DA. Panic disorder in primary care: A cause of unexplained symptoms. Cleve Clin J Med 1997;64:437-443.
Rubin A, Chassey M. When anxiety attacks: Treating hyperventilation and panic. Phys Sportsmed 1996;24:54-65.

be reassessed if the patient does not improve after 6-8 weeks.[4]

Pharmacotherapeutic options

Biological treatment is directed at regulating the physiologic system, addressing the underlying constitutional vulnerability, managing complications, and reducing severe impairment and distress to the point of remission—or to the degree that other therapies become realistic options. The goals of pharmacotherapy are to inhibit panic attacks, address comorbid psychiatric conditions, and get the patient into remission or recovery. Sometimes the aim is to decrease the intensity and frequency of panic attacks. Medications can reduce chronic arousal, diminish reactivity, and blunt or block climactic extremes of self-escalating arousal processes or panic attacks. They are less effective in managing anticipatory anxiety.

Efficacy from a single pharmacologic treatment trial—as measured by panic-free rates—is 50%-70%.[23] Relapse is common once the drug is discontinued. Treatment of panic disorder is, however, important because mastery of distressing situations is an important contributor to self-esteem throughout life. Medications allow many people to overcome anxiety so severe that it had rendered role functioning impossible.

• *Treatment phases* The acute phase of treatment, which extends from its initiation for an acute episode until symptoms remit, may vary from 2-3 weeks to 2-3 months. The continuation phase begins at the time of acute symptom remission and lasts until symptoms have been eliminated or reduced and function has been restored (Table 4). This phase varies from 2 months to 1 year. The maintenance phase is used to prevent the recurrence of initial symptoms and maintain accrued benefits. Patients whose symptoms recur during or after medication tapering are the main candidates for maintenance. Doses should always be reduced relatively slowly, not stopped abruptly. Increasing the frequency of follow-up visits during the period of tapering allows the physician to provide guidance and support and to assess carefully for rebound anxiety, withdrawal phenomena, and symptom recurrence.

• *Tricyclic antidepressants (TCAs)* have demonstrated efficacy in treating panic for more than 30 years. They are, however, associated with a delayed onset of action and side effects—particularly orthostatic hypotension, anticholinergic effects, weight gain, and cardiac toxicity—that may compromise tolerance of and compliance with therapy. The starting dose for an agent

such as imipramine HCl (Tofranil) is 10 mg hs, to avoid possible early-onset anxiety exacerbation and tachycardia. The rate of short-term control of panic attacks with TCAs is 63%; the attrition rate is 28%, usually related to nuisance side effects.[24] Generally, the dose is titrated (up to 250 mg/d) until the patient is asymptomatic. TCAs have little potential for abuse.

• *Selective serotonin reuptake inhibitors (SSRIs)* are an effective, well-tolerated alternative to benzodiazepines and TCAs. A meta-analysis of 32 randomized, placebo-controlled studies involving more than 2,300 patients found SSRIs superior to either imipramine or alprazolam.[23] They lack the cardiac toxicity and anticholinergic effects of TCAs. Four of the available SSRIs—fluoxetine HCl (Prozac), fluvoxamine maleate (Luvox), paroxetine HCl (Paxil), and sertraline HCl (Zoloft)—have shown efficacy for the treatment of panic disorder. The newest SSRI, citalopram (Celexa), has not been systematically studied for panic disorder, although it probably is efficacious. To minimize increases in anxiety, treatment must be initiated at a low dose.

• *Monoamine oxidase inhibitors (MAOIs)* Some consider MAOIs such as phenelzine sulfate (Nardil) to be the most comprehensively effective agents for blocking panic attacks and for relieving the depression and concomitant social anxiety of panic disorder. Recommended doses range from 45-90 mg/d. MAOI use is, however, limited by adverse effects such as orthostatic hypotension, weight gain, and insomnia. Since the need for careful dietary monitoring and risk of hypertensive crisis may be daunting for many patients, MAOIs are often reserved for patients who do not respond to the safer and better-tolerated drugs.

• *Benzodiazepines,* such as clonazepam (Klonopin), alprazolam (Xanax), and lorazepam (Ativan), are also effective in blocking panic attacks. Advantages include a rapid onset of therapeutic effect and a safe, favorable, side-effect profile. Among the drawbacks are the potential for abuse and dependency, worsening of depressive symptoms, withdrawal symptoms on abrupt discontinuation, anterograde amnesia, early relapse on discontinuation, and inter-dose rebound anxiety. Although contraindicated for anyone with substance abuse problems, benzodiazepines are not generally abused by persons with panic disorder. In fact, they are usually underutilized in these patients.

The starting dose of alprazolam is generally 0.5 mg bid. Approximately 70% of patients will experience a discontinuance reaction characterized by increased anxiety, agitation, and insomnia when alprazolam is tapered. Clonazepam's long duration of effect diminishes the need for multiple daily dosing. Starting low doses of 0.25 mg/d are gradually titrated to the usual effective dose of 1-5 mg/d. Initial symptoms of sedation and ataxia are usually transient.

• *β-Blockers* are useful in moderating heart rate and decreasing dry mouth and tremor; they are less effective in relieving subjective anxiety. The abuse potential is low, and memory and psychomotor performance is usually unaffected. But these agents are contraindicated in patients with asthma, heart blocks, and low cardiac output states.

• *Treatment for refractory patients* Monotherapy is ineffective in 20% of patients.[24] Reduced response to treatment is more likely in patients whose anxiety disorders coincide with another psychiatric disorder. Interventions for refractory cases may consist of adding buspirone (BuSpar) to an SSRI, or switching to an MAOI.

Behavioral therapy and psychotherapy

Cognitive behavioral therapy and other forms of psychotherapy appear effective for some or all panic disorder symptoms.[25] Psychotherapy can elucidate unconscious conflicts that may prompt avoidance behavior or the panic-stricken cessation of functioning. Response may be enhanced if a period of drug treatment is used to control symptoms.[25]

• *Cognitive behavioral therapy* is based on the principle that what is important for enduring recovery is mastering anxiety, not avoiding it. Mastery usually translates into reduction of arousal. Cognitive behavioral therapy is a short-term treatment, typically lasting 4-6

weeks. Good evidence indicates its efficacy in reducing the number and severity of panic attacks, and in arresting avoidance behavior. On average, about 60% of patients improve with this form of treatment.[26] Only 10%-20% of patients drop out of therapy, but attrition rates are higher for patients with agoraphobia.[24]

• *Psychoeducation* provides a foundation for the development of a new way of viewing the panic experience and empowering the panic sufferer. Patients should be given information about their disorder from the outset. They are usually relieved to discover that their symptoms are not indications of life-threatening medical conditions or signs of mental illness.[27] Shame can be mitigated by explaining that 15% of Americans have had a panic attack—about 3% of them within the past month.[1] Education about the adaptive function of anxiety and the fight-or-flight response helps patients understand that a panic attack is a protective coping response that is activated unnecessarily.

• *Other behavioral therapies* Biofeedback and other therapies that use progressive muscle relaxation, meditation, guided imagery, and hypnosis can also be successful in reducing arousal. Relaxation techniques that include breathing retraining are the single most helpful aspect of biofeedback. The patient is taught to use the diaphragm, not the intercostal muscles, to breathe. Because it involves substantial hardware and training, biofeedback requires referral to a specialist.

Conclusion

Panic disorder is a prevalent, often recurrent, psychiatric condition that is occasionally chronic. It affects adults, primarily women, and usually begins in early adulthood. One third of patients develop agoraphobia. Because panic attacks often involve somatic symptoms, patients commonly seek treatment from primary care physicians. Persistent, medically unexplained physical symptoms may distract the physician from recognizing the underlying psychiatric disorder.

In the approach to the patient with symptoms of panic disorder, the first task is to rule out medical illness and psychosis and make the diagnosis. The second is to control the metabolic core of the disease with medication. The third is to control phobic restrictions with behavioral therapy. Single drug therapy combined with cognitive behavorial therapy has been found effective in 60% of patients. Psychotherapy can also address associated psychosocial problems and uncover unconscious aspects of anxiety. Relapse prevention includes educating the patient about the nature of the disorder and ensuring long-term management. If panic disorder is inadequately treated, it can compromise the patient's well-being, vocational capacity, and family relationships. §

SELF-EXAMINATION

1. All except _____ are diagnostic criteria for panic disorder.
   a) the abrupt development of intense fear or discomfort accompanied by at least four somatic or cognitive symptoms that peak within 10 minutes
   b) attack-related persistent concern, worry, or behavioral changes
   c) accompanying specific phobias
   d) a panic attack that occurs for no apparent reason

2. In patients with panic disorder, the fear of an impending attack is more debilitating than the attack itself.
   a) true b) false

3. All except _____ are common comorbid diagnoses in patients with panic disorder.
   a) mitral valve prolapse
   b) irritable bowel syndrome
   c) vestibular dysfunction
   d) personality disorders

4. To prevent a recurrence of symptoms, maintenance pharmacotherapy is indicated for

all panic disorder patients.

a) true b) false

5. Which of the following statements about pharmacotherapy is false?

   a) The side effects of TCAs may compromise compliance with therapy.
   b) Studies have found SSRIs superior to imipramine and alprazolam.
   c) MAOIs are usually reserved for patients who have not improved with other medications.
   d) Because of their potential for abuse and dependency, benzodiazepines should be avoided.

Answers at end of reference list.

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Answers: 1)c, 2)a, 3)c, 4)b, 5)d