

Sleep Complications in Depression, Anxiety, and Psychotic Disorders and Their Treatment

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Sleep disorders are a common problem in the general population as well as the psychiatric population. Although these disorders don't have the same morbidity and mortality as the Axis I disorders, they are reported frequently and cause much discussion among psychiatrists. Patients find sleep disorders inconvenient at best, crippling at worst; they feel lonely and isolated with a poor night's sleep. To be up in the middle of the night when their significant other is asleep is profoundly disconnecting and demoralizing. The rest of the family will suffer if their family member is up half the night, or worse, tossing and turning in the same bed. Compounding this clinical issue, many physicians are not trained in sleep medicine and many of the FDA-approved medications have an abuse liability.

There are more than 80 sleep disorders officially recognized by the American Academy of Sleep Medicine.¹ They are logically laid out in *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition,² and follow the rationale used in other disorders. They may be



primary disorders or secondary disorders. The secondary disorders can be secondary to psychiatric, medical or substance use disorders. Mastering the DSM-IV classification will aid in management of both psychiatric and primary care patients. When patients rest more easily and families sleep more confidently, then everyone copes more effectively, making treatment for psychiatric patients more successful.

The Figure (see page 855) is a reminder that several common features in psychiatric patients affect sleep. It is important to work with patients comprehensively, to assess each of the four areas before making a plan.

MOOD DISORDERS

Manic episodes are defined as abnormal and persistently elevated, expansive, or irritable mood episodes lasting at least a week, or any shorter amount if

hospitalization occurs. In addition, three of the following must be present: inflated self-esteem or grandiosity, decreased need for sleep, pressured speech or more talkative, flight of ideas or a subjective experience of racing thoughts, distractibility, increased goal-directed activity or psychomotor agitation, or excessive involvement in pleasurable activities with high potential for problems.

Sleep deprivation may trigger mania or may be associated with early onset of mania.³ Primary insomnia may be a trigger and secondary insomnia may be a reinforcer. This physiology may cause a spiraling prodrome that ends in full-blown mania. It does suggest that management of this decreased need for sleep may ameliorate the manic episode. Recent studies suggest that sleep deprivation has antidepressant effects. It has had immediately antidepressant effects in 50% of patients, but napping or subsequent sleeping will reverse the effect just as quickly. Partial sleep deprivation and REM deprivation also seem to have antidepressant effects. It is not known if this is the cause or effect for the antidepressant effects.

Major depression is defined as depressed mood or anhedonia during the same two-week period; it is not part of a medical illness or psychosis. Furthermore, there must be significant weight loss or weight gain, insomnia, or hypersomnia nearly every day, psychomotor agitation or retardation nearly every day, fatigue or loss of energy nearly every day, feelings of worthlessness or excessive guilt nearly every day, diminished ability to think or concentrate nearly every day, recurrent thoughts of death or suicidal ideation or suicide plan or attempt. Although not a DSM criterion, a sleep disturbance may persist after the acute episode.

A polysomnogram (PSG) is an EEG tracing during sleep that includes leads that record breathing, pulse and limb movements. The PSG is the primary manner to record and measure the stages

of sleep, and to study the way that disturbances occur. In mood disorders, PSGs indicate several types of sleep abnormalities. On a gross level there are sleep continuity disturbances with prolonged sleep latency (longer time to sleep onset), increased wakefulness during sleep, early-morning awakening, sleep fragmentation, and decreased sleep efficiency (more time in bed is spent in wakefulness). Deep sleep is recorded as stage 3 and 4 on a PSG and is called slow-wave sleep (SWS). Decreases in deep sleep, SWS, are measured on the PSG in major depression, and much of this deficit is observed in the first period of non-rapid eye movement sleep (NREM), which occurs early in the sleeping episode.

In addition, REM sleep abnormalities are a robust finding observed on the PSG. Reduced REM latency (time to first REM sleep) and disturbing dreams are observed along with prolonged first REM and increased rapid eye movement during the first REM period. These correlate with subjective complaints of insomnia, difficulty falling asleep, difficulty remaining asleep, and early morning awakenings. It is interesting that manic patients show similar changes, but dysthymic patients show normal PSG patterns. Unfortunately, sleep patterns don't seem helpful diagnostically. Depressed mood that does not meet depressive disorder criteria did not correlate with PSG changes. Reduced REM latency and reduced SWS may persist well beyond the acute episode.

TREATMENT

On one level, treatment for sleeping problems includes anything that works. Successful strategies have been exercise to make one more tired; relaxation techniques to help stop racing thoughts; or sleep hygiene guidelines that encourage the person only to sleep in bed and not watch television, read, or toss and turn.

Pharmacological interventions can be directed at the underlying disorder

CME	EDUCATIONAL OBJECTIVES
	<ol style="list-style-type: none">1. Describe the effect of acute anxiety disorders on a patient's sleep.2. Discuss the effect of acute mood disorders on patient's sleep.3. Identify the effect acute schizophrenia may have on a patient's sleep.
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if there is a secondary sleep disorder, or they can be aimed at symptom relief regardless of the cause.

Antidepressants tend to normalize sleep as they change the depressed mood. However, serotonin syndrome, which is associated with the newer selective serotonin reuptake inhibitors (SSRIs) is a possible adverse event that could lead to sleep disruption.⁴ Because antidepressants cause REM suppression, and depression is associated with increases in early REM sleep, this effect may be associated with their overall effectiveness. Conversely, abrupt withdrawal of antidepressants may cause REM rebound with accompanying bizarre, intense or frightening dreams or sleep fragmentation. Trazodone, an antidepressant that has been popular as a soporific, does not suppress REM. On the other hand, bupropion increases REM sleep. Tricyclic antidepressants and SSRIs may increase restless leg or periodic leg movements and can exacerbate insomnia due to these disorders.

With bipolar illness, mood stabilizers may increase daytime sleepiness. Lithium may prolong REM latency and suppress REM sleep time. Even in manic patients, lithium increases SWS. As with SSRIs, lithium may exacerbate restless leg syndrome. Adjunctive benzodiazepines or neuroleptics may be used early in treatment of mania if there are prominent sleep complaints. This is especially true when sleep deprivation triggers manic symptoms. Common FDA-approved soporifics are benzodiazepines and selective GABA-receptor agonists. They do not cause many adverse events when combined with antidepressants, nor does trazodone. Potential interactions might occur with cytochrome P-450 enzymes. Patients with chronic sleep disturbances, as well as the mood disturbances with their secondary insomnias, may develop a psychophysiological insomnia when they develop performance anxiety about going to

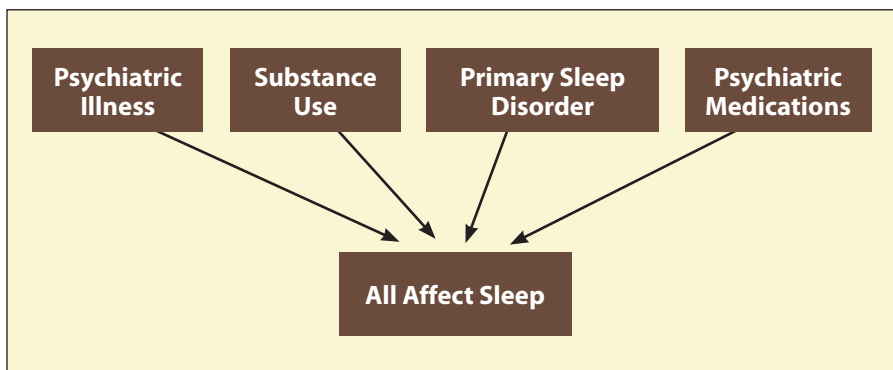


Figure. Factors in psychiatry that affect sleep.

sleep. Such patients can benefit from behavioral interventions that improve self-efficacy (a sense of competence), as well as the common soporifics. Failure to respond to pharmacology for mood disorders should suggest the possibility of a co-occurring primary sleep disorder (dyssomnia or parasomnia).

ANXIETY DISORDERS

In DSM-IV, panic attacks are defined as palpitations, pounding heart, sweating, trembling or shaking, sense of shortness of breath, feelings of choking, chest pain or discomfort, nausea or GI discomfort, dizziness or lightheadedness, derealization or depersonalization, fear of losing control, fear of death, paresthesias, and chills or hot flashes. Panic disorder, in addition, has at least one month of one of the following: persistent concern about more panic attacks, worry about the implications of the panic attacks, or panic avoidant behaviors. Although not a criterion for the disorder, 65% of these people have nocturnal panic attacks, or sleep panic attacks.⁵ These are associated with sleep awakenings and nonrestorative sleep. The sleep panic is associated with NREM sleep, while nightmares (dream anxiety attacks) are associated with vivid dreams and REM sleep. Both can be associated with autonomic activation and arousal. Sleep panic usually occurs within 3 hours of sleep onset during late stage 2 and early stage 3 sleep.

Patients usually have normal REM sleep physiology, as measured by PSG, and a normal percentage of time in each of the four stages of sleep. They may have trouble falling asleep, but the EEG evidence does not show many abnormalities despite the many subjective complaints. Sleep deprivation seems to worsen/increase the daytime and nocturnal panic attacks (sleep panic).⁶ These may be due to respiratory physiology that causes rising CO₂ and may be related to the physiology of relaxing. Recurrent nocturnal panic may cause fear of sleep and avoidant behaviors, accompanied by shame and embarrassment.

Treatment of the sleep disturbances associated with panic attacks includes avoiding caffeine, which is known to exacerbate panic disorder; treatment with medications that improve panic disorder symptoms; and instructions about sleep hygiene.

Social phobia is the fear of being in social or performance situations that expose the person to unfamiliar individuals; the patient fears he or she will react with anxiety and in a fashion that would be humiliating or embarrassing. Furthermore, exposure to this setting usually provokes anxiety. The person recognizes the fear is excessive or unreasonable. It lasts at least 6 months and causes significant functional impairment. As with panic disorder, the PSGs are rather normal looking. Alcohol consumption may be used as a mild anxiolytic, but it can cause awakenings and

nightmares when asleep, and increased anxiety while awake in proportion to the amount consumed. Although alcohol increases SWS in the early part of the night, it increases REM sleep in the later part of the night when it can cause more awakenings and nightmares. Treatment of social phobia usually involves medications for social phobia (benzodiazepines, monoamine oxidase inhibitors, SSRIs and beta blockers). If this treatment is not effective, then the combined use of medications and behavioral interventions is recommended.⁷

Case History 1: Sleep Panic

Ms. B is a 30-year-old woman with a 17-year history of panic disorder. Her first lifetime episode of panic attacks awakened her from sleep at age 13. Both the initial and subsequent sleep panic attacks were characterized by abrupt arousals from sleep without dream recall. She denied somnambulism. Her sleep panic attacks were associated with a sense of impending doom, heart palpitations, shortness of breath and lightheadedness that lasted for several minutes.

The patient experienced her first daytime panic attack at the age 15, approximately 2 years after her first sleep panic attack. Her daytime wake panic attacks, which were similar in quality to her sleep panic attacks, often were unpredictable or triggered by exercise. It was not unusual for Ms. B to have many episodes of daytime awake or sleep panic attacks within 24 hours. Despite her frequent daytime wake and sleep panic attacks, she reported minimal degrees of avoidance behavior.

During the month before her admission to the National Institute of Mental Health, Ms. B had 35 sleep panic attacks and 30 wake panic attacks. Treatment consisted of imipramine and alprazolam. She had a total response (ie, complete blockade of wake and sleep panic attacks) with this drug combination. She relapsed when the drugs were discontinued while she was pregnant.

Case History 2: Sleep Panic and Sleep Avoidance

Ms. C is a 26-year-old single woman with a 10-year history of panic disorder with agoraphobia. Her first daytime panic attacks developed at home while reading a newspaper. The occurrence of several additional panic attacks within the next several weeks led to an exten-

sive medical and neurological workup. No evidence of neurological or cardiovascular disease was identified. Her daytime wake panic attacks included heart palpitations, tachycardia, sweating, hand tremors, hot flashes, chest tightness, psychosensory disturbances, and shortness of breath. She was treated for several years with insight-oriented psychotherapy that improved her ability to deal with stressful family issues but had no effect on the frequency or severity of her daytime wake panic attacks. Ms. C gradually developed multiple fears typical of agoraphobia (i.e., fear of elevators, grocery shopping, bridges, crowded places and public transportation) and became unable to travel beyond a 1-mile radius of her home. These avoidance behaviors increasingly dominated her life, leading to complete disability in terms of work and social function.

Several years after the onset of daytime wake panic attacks, Ms. C developed sleep panic attacks. She had a history of nightmares but stated that her sleep panic attacks were quite different in quality from her nightmares. She denied any dream content associated with her sleep panic attacks. Ms. C described waking from sleep with a pounding chest, sweating, numbness in her hands, and a choking sensation. She also had a fear that she might die of suffocation. Ms. C quickly became frightened of sleeping (“I didn’t want to die in my sleep”) and developed a progressive pattern of bizarre sleep behaviors (eg, sleeping in a sitting position, sleeping in a chair, sleeping without bedspreads, lowering or raising the temperature in the room, and asking a friend

or family member to sleep in a nearby room). These behaviors were intentionally promulgated by the patient in an attempt to obtain rest without “sleeping to the point of panic.” A consequence of her sleep phobia, however, was the development of chronic intermittent sleep deprivation and an apparent increase in the frequency and severity of both sleep and wake panic attacks.

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Case History 3: Generalized Anxiety Disorder

Generalized anxiety disorder (GAD) involves excessive anxiety and worry, and it is difficult to control. There are symptoms of tension, irritability and restlessness, including sleep disturbance. The anxiety causes a significant impairment in social functioning and is not accounted for by another psychiatric disorder, including substance-induced anxiety. More than half of patients with GAD report sleep problems and can't stop worrying at bedtime. EEG studies show increased sleep latency and awakenings during the night with reduced SWS sleep. Treatment includes benzodiazepines, and SSRIs may be helpful.

Mr. D is a 43-year-old married engineer with a 15-year history of chronic tension and generalized anxiety. His anxiety symptoms began shortly after the death of his father. Additional signs or symptoms of his generalized anxiety disorder included chronic tension, constant worrying about either his job performance or marriage, bouts of fatigue (described by the patient as a consequence of chronic stress), shortness of breath, motor agitation (usually lower extremities), sweating, and hot flashes. Mr. D's concerns were almost always unrealistic; for example, he had received many outstanding evaluations regarding his work performance and was highly regarded within the industry. Nevertheless, he harbored a fear that he was inept and would be fired.

Major life events triggered a marked exacerbation in the severity of the above symptoms. During periods of stress, Mr. D also predictably developed "globus hystericus," urinary frequency and a wide array of abdominal complaints. He also sought the advice of several different physicians.

At the height of his distress, Mr. D had significant sleep disturbance (particularly early and middle insomnia) lasting for weeks or months. He denied suicidal ideation, melancholia, sleep panic at-

tacks, nightmares, somnambulism, loud snoring, sleep paralysis, obsessions, compulsions, or social anxiety.

Treatment consisted of cognitive behavioral therapy plus alprazolam. This treatment approach resulted in an immediate and sustained improvement in his sleep complaints, although complete control of his generalized anxiety and associated gastrointestinal problems required 12 months of intensive cognitive behavioral therapy.

Case History 4: Posttraumatic Stress Disorder

Posttraumatic stress disorder (PTSD) is characterized by reliving a traumatic experience or experiences, trying to avoid reminders that trigger the re-experience, and hyperarousal, such as difficulty falling asleep or staying asleep, irritability or angry outbursts, trouble concentrating, hypervigilance, or exaggerated startle response. If the trauma happened during sleep, there may be additional sleep problems with a conditioned fear of sleep as part of the attempts to avoid re-experiencing the trauma. About two-thirds of the patients report frequent nightmares. Nightmares associated with PTSD seem to be reliving the content and emotions of the traumatic experience. These nightmares seem to be REM nightmares, although awakenings during NREM sleep may also occur. REM behavior disorder may occur with the nightmares.

Treatment is most effective with combined psychotherapy and pharmacotherapy. Although typical anxiolytics are commonly used, there is a high rate of co-occurring alcohol dependence. This complicates the choice of medications when as many as 70% of these patients have alcoholism. Both benzodiazepine dependence and alcohol dependence are associated with substance-induced sleep disorder complications. Management of these must occur simultaneously with treatment of the PTSD, making this a complicated

and awkward treatment. Patient requests for relief in the form of benzodiazepines can reach desperate proportions, but the doctor can resist such requests out of the compassionate wish to avoid the complications of benzodiazepine dependence. Use of non-addictive medications is preferred, and most of these medications only help people initiate sleep; a few are associated with helping patients maintain sleep. Management of the sleep disturbance is often ineffective by the normal benchmark of 8 hours of sleep, and the goal may have to be obtaining a manageable level of sleep disturbance, ie, 5 hours of sleep per night instead of 3 hours per night.

Mr. A is a 53 year-old Vietnam War veteran. He was an infantryman in the Army and served in combat for 11 months. He was in many firefights and often thought he might die. Several of his Army friends were killed in combat during these encounters. He had a stormy period of adjustment to civilian life in the early 1970s. He drank heavily in an effort to blank out the intrusive thoughts of combat that haunted him back in the States. He had tremendous difficulty falling asleep without alcohol and frequently awakened after 1 to 3 hours of sleep. He had a recurrent nightmare of being overrun by the Viet Cong and would awaken in a cold sweat. Before his divorce, his wife said she would get afraid at night because of his nightmares. At times he grabbed her and choked her until she woke him. She would then go into another room to sleep.

Twenty-five years after his combat experience he was still having difficulty sleeping, averaging about 3 hours per night and frequently awakening to nightmares of combat. He had trouble falling back to sleep and would often go from window to window to make sure everything was safe outside. He was able to nap briefly in the day, but he felt tired during the day. He came to the psychiatrist after completing a residential treatment pro-

gram for his alcohol dependence. Without his alcohol, he was much more aware of his insomnia and felt out of control.

The psychiatrist prescribed increasing doses of trazodone, beginning at 50 mg at bedtime. When the dose reached 150 mg he was able to sleep about 5 hours per night and did not feel “hungover” or “drugged up” in the daytime, live he did when he took 200 mg. He did not feel too tired during the day and did not need to nap often. After six months of abstinence, it was obvious that he needed specialty treatment for his posttraumatic stress disorder, and he was referred to the PTSD residential program at the local Veterans Affairs Medical Center. Although he was helped symptomatically, he still needed the trazodone for his 5 hours of sleep at night.

OBSESSIVE-COMPULSIVE DISORDER (OCD)

Obsessive-compulsive disorder (OCD) is characterized by obsessions and/or compulsions that are recognized as excessive or unreasonable. The symptoms are distressing or impair normal functioning and are not due to another psychiatric disorder.

Many patients with OCD do not have sleep disturbance; those who do have a sleep disorder report awakenings associated with obsessions or compulsions, such as having to check the locks at night. Their EEGs were similar to those of depressed patients. Sleep seems to improve when obsessive compulsive symptoms are treated successfully, with SSRIs and/or psychotherapy. There is some suggestion that clomipramine is more sedating, while fluoxetine and fluvoxamine may worsen insomnia and require additional pharmacotherapy. Behavioral interventions may also be useful in combination with these medications.

SUBSTANCE-INDUCED ANXIETY DISORDER

Substance induced anxiety occurs when anxiety is a prominent symptom, and it occurs with a substance known to

cause anxiety with intoxication or withdrawal. Removal of the substance is the best treatment. Common substances that cause intoxication-induced anxiety:

- Caffeine,
- Cocaine and stimulants,
- Chronic alcohol use,
- Drug panic associated with hallucinogens (marijuana, hash, LSD, etc.),
- Serotonin syndrome (caused by a variety of medications), and
- Rebound anxiety with the shorter-acting benzodiazepines.

Withdrawal-induced anxiety can involve physiological dependence on any of these agents:

- Alcohol and sedatives,
- Opioids, and
- Nicotine (usually mild).

Management of these disorders involves removing the toxic substance or treating the disturbing withdrawal syndrome. Sleep disturbances associated with opioid dependence can take weeks of abstinence to resolve. With opiate substitution therapy, the sleep disturbance may never go away. However, opioid-dependent individuals may still have a treatable sleep disorder that is not substance-induced.

PSYCHOTIC DISORDERS

Schizophrenia is the most common diagnosis among psychotic disorders. The diagnosis is made when two or more of the following are present for a significant portion of time during a one-month period:

- Delusions;
- Hallucinations;
- Disorganized speech (frequent derailment or incoherence);
- Grossly disorganized or catatonic behavior; and
- Negative symptoms such as affective flattening, avolition or anhedonia.

Furthermore, there is marked deterioration in functioning that persists for at least 6 months. Most importantly, it is not due to another illness like affective disorder,

schizoaffective disorder, developmental disorder, or psychoactive substances.

Although not part of the criteria, sleep disorders are prominent in the prodrome and the acute illness with schizophrenia.⁸ REM abnormalities are inconsistent in empirical studies, but SWS abnormalities are more consistently found. PSGs show markedly reduced deep sleep and delayed sleep onset, often delayed by 50 to 100 minutes (normal is 10 to 20 minutes). Reduced serotonin metabolites are associated with reduced stage 4 sleep (deep sleep) in unmedicated schizophrenia, but these deficits are not responsive to newer antipsychotic medications, even though these medications may improve time asleep and quality of sleep. The stage 4 deficits persist with the atypical neuroleptics even though there is a marked increase in stage 4 sleep for normal sleepers when these medications are studied. Newer neuroleptics have serotonin antagonist effects and reduce SWS but increase total sleep time and NREM sleep. These medications have activity at a variety of receptors, and daytime sleepiness may well be related to antihistaminic effects.

When managing the dyssomnias among the schizophrenic population, it is important to remember that there is considerable co-occurrence of sleep disorders and psychotic disorders. Several studies have reported high rates of sleep-disordered breathing and periodic limb movement. These studies also reported that sleep-disordered breathing problems can be treated with the positive airway treatments with the same degree of compliance as nonschizophrenic patients. Because many of the newer antipsychotic medications have long half-lives, they can be dosed at bedtime and used to induce sleep and prolong time asleep before awakening. Neuroleptics can be mixed with bedtime benzodiazepines in the early recovery phase when anxiety can be high, as long as there is no history of psychoactive substance abuse/dependence. If there is

such a history, then greater care, careful informed consent and close monitoring are required before prescribing them, if at all. Ruling out sleep-related breathing disorders is also important when considering the use of benzodiazepines, which can exacerbate the sleep-related breathing disorders. For those patients with an impulse-control problem or schizoaffective disorder, a mood stabilizer can have a beneficial effect on sleep.

Akathisia is a side effect of antipsychotic medications that can produce challenging dilemmas when it interferes with sleep. Dose reductions of medications may be tried but this reduces sedation at bedtime and may lead to decompensation. Anticholinergics and propranolol can be used in the management of akathisia. If there is asthma present, propranolol is not indicated and amantadine is another option. The newer atypical antipsychotic medications have reduced extrapyramidal symptoms and preclude this problem.

Case History 5: Schizophrenia

Ms. E is a 27-year-old woman who was on outpatient probate. She had suffered several psychotic episodes in the past year and was unable to care for herself during these episodes. She did not sleep much and was up all night in fear of the voices that told her to kill herself and her neighbors. Last year she was put on clozapine after a trial of several atypical neuroleptics did nothing to help her psychotic symptoms of auditory hallucinations and paranoia. She did well on clozapine but stopped taking it a month after discharge when she moved

and did not make connections with her new caregivers. She was rehospitalized and responded quickly to a restorative dose of clozapine. But she decided to return to the clinic for her follow-up care, which disrupted her medication compliance once again.

Ms. E was assigned a case manager and this case manager made quick ar-

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rangements to have her continued on clozapine. She did well for two months and was coming up on her 90-day hearing at probate court. She also had a vacation planned with her family. As the vacation approached, the case manager thought that the patient might be skipping some doses of clozapine since her mother reported that she was up all hours of the night. This was addressed immediately

in the weekly clozapine medication monitoring with the psychiatrist, and she did well without needing hospitalization.

SUMMARY

Although the focus of this article is on the sleep problems of psychiatric disorders, the message is: Be comprehensive in assessment. Sometimes a thoughtful history can differentiate what is substance-induced and what is secondary to psychiatric illness. Obtaining a lifetime history of sleep can help determine that a primary sleep disorder preceded psychiatric illness. Some of the Axis I disorders in DSM have sleeping problems associated with acute illness that are treated effectively once the acute phase is over. When this is inadequate for management, then further exploration and treatment are required. It is here that behavioral interventions are invaluable.

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