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Anxiety and Anxiety-Related Disorders in the Adolescent Population: An Overview of Diagnosis and Treatment

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Anxiety is a common component of visits to the doctor's office by adolescents; however, it is often overlooked as a possible causative agent of the presenting complaint. With a high index of suspicion and proper questioning, a clinician can analyze the contribution that anxiety plays in the life of an adolescent patient. Behavioral and pharmacologic interventions exist for this group of disorders, and with proper diagnosis and treatment the symptoms of these disorders can possibly be ameliorated. The following provides a concise guide to the diagnosis and treatment of anxiety and anxiety-related disorders including hyperventilation syndrome, syncope, sleep disorders, panic disorder, and obsessive compulsive disorder (OCD).

ANXIETY

Anxiety is one of the most common causes of symptomatology resulting in an office visit. When an adolescent presents with loss of consciousness, mental status change, cardiac problems, stomachaches, headaches, or any set of symptoms that seem to be disparate, a diagnosis of anxiety disorder should be considered. Anxiety ought to be treated as a piece of diagnostic information that may or may not require treatment and should be considered as a possible cause, or at the very least a component, of any chronic medical condition.

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Approaching the history of the presenting problem, it is the responsibility of the clinician to maintain an awareness of the differing perspectives that will be offered as the patient or family describes the problem. In the case of anxiety, it is likely that the level of anxiety in the family will affect the adolescent, but the adolescent will have his or her own perspective on the extent of the disability. In the interest of eliciting candid answers from an adolescent patient, we encourage the clinician to try to interview the patient without the adult present.

History

The clinician should consider the possibilities of metabolic disturbances (eg, thyroid disease), substance abuse or substance exposure, or psychiatric conditions as part of the differential diagnosis when evaluating for anxiety disorder. As always, the clinician ought to question how long this symptom has been present. Was the onset associated with any other occurrence (eg, illness, emotional or physical trauma)? Questions such as "When and in what situation does anxiety usually occur?" and "Is anxiety more likely to occur at home, school, or other social situations?" will help to further focus the evaluation. It is important for the clinician to explore whether anxiety is more likely to occur in social situations that are peer related or in family situations. It is our experience that school is the most likely setting for the adolescent experiencing anxiety.

Next, the clinician can try to discern what patients actually experience when they say they are feeling "anxious." One can attempt to elicit an explanation of symptomatology as well as what the patient can do to "make it better" or "prevent" the feeling. To discern the chronicity of the problem, one may ask, "Is the anxiety there all the time for you, or is it episodic?" If it is episodic, one should elicit a description of the last 2 to 3 times that anxiety occurred.

Frequently, a precipitating factor in adolescent anxiety is the stress of school. To discern whether the stress is academic or social, it is important that the clinician ask about grades, family expectations, and personal expectations. Commonly, one might hear single-syllable answers to these queries. Regardless of the answer, the clinician can question more deeply (eg, by asking questions such as "What are your grades like?" "How do you do on tests?" and "Do you think you study more or less than your peers?") Exploring social stress, the clinician can ask questions such as, "Is the teacher picking on you?" "Are peers making fun of you?" "Have you had any recent altercations with any peer(s) at school?" "Are you being ostracized?" "Are you getting into trouble in school because of misbehavior?" and "Are your parents pressuring you to do better in school?" There are a plethora of questions that can be asked to shed light on the source of the anxiety. It is our hope that the above-listed questions give the clinician several tools to aid in eliciting a more precise history of the condition.

Last, in obtaining a history from the adolescent it may be important to ask the patient to describe recent episodes that occurred in interactions with peers. With this style of questioning, the clinician is attempting to get every bit of information, because one may never know what piece of information will provide an important clue. As we have all experienced, the most important piece of data might not seem important to the patient and may not be offered without directed history questions.

Treatment

Depending on the level of anxiety, one may wish to consider helping a patient get the nonpharmacologic help they need to cope with anxiety levels. Although counseling can be quite effective in dealing with disabling levels of anxiety, it is often rejected by family or patients for many different reasons (eg, unobtainable for financial reasons or the social stigma associated with counseling). Relaxation techniques including meditation and yoga have been shown to reduce stress in adolescents.[1,2] Nonpharmacologic modalities can aid the adolescent in developing coping skills. In this way, he or she is not merely a passive victim of anxiety; rather, these skills allow the patient to actively engage in treatment.

If nonpharmacologic therapies are not an option or are not successful, one can consider using pharmaceuticals to ease the symptoms of anxiety.[3,4] Treatment may vary depending on whether the anxiety is chronic or acute and recurring.

One of the significant concerns in a patient who suffers from anxiety is that the patient will self-medicate if anxiety is significant enough. This should be taken into account when considering pharmaceutical treatment. The most beneficial treatments for acute anxiety are the benzodiazepines, but this class of drug also carries a significant risk of abuse. That said, for episodic anxiety, the benzodiazepines are the best choice because of the quick-acting nature of the chemical. Unfortunately, with chronic use, a patient may develop a tolerance, and the dose will need to be increased periodically to remain effective. Suggested benzodiazepines include alprazolam (Xanax [Pfizer, New York, New York]) and lorazepam (Ativan [Biovail, Bridgewater, NJ]). Alprazolam (0.25–1 mg orally three times per day), which has a half-life of 6 to 20 hours, is the most effective and quick acting. Lorazepam (0.5–2 mg PO orally three times per day) has a half-life of 10 to 20 hours and is also helpful but not as quick acting as alprazolam. Again, we need to emphasize the need to be extremely cautious with benzodiazepines because of the potential for abuse. These drugs can cause physical and psychological dependence. Withdrawal can occur, especially if benzodiazepines are used for 3 to 6 months at relatively high doses. When used intermittently, the dose can remain the same, and addiction and tolerance issues are less likely to occur.

Note that in the case of an adolescent with a comorbid seizure disorder, one should consider if this patient is being treated chronically with a benzodiazepine.

If this patient were to have an episode of status epilepticus, benzodiazepines may be less effective, because the patient may have a developed tolerance to this class of medications.

When starting a patient on these medications, we suggest beginning the medication regimen on a non–school day or after school hours to try to avoid the possibility of problematic sedation.

The selective serotonin reuptake inhibitors (SSRIs) have been shown to be effective in treating generalized and social anxiety. There is a black-box warning on SSRI medications[5] concerning an increase in suicidal ideation associated with the use of these medications; however, there are also published reports regarding increased suicide in adolescents caused by decreased prescribing of SSRIs by primary care physicians.[6–9] It is worth noting that a warning is not a contraindication, and SSRIs can be used in adolescents with caution and careful follow-up.[10] SSRIs may take as long as 4 weeks to show some effectiveness and, therefore, are useful for chronic anxiety but not for intermittent acute anxiety. Therefore, these medications are not useful when taken on an as-needed basis. Depending on the severity of the acute symptoms, the clinician might need to begin treatment by providing the patient with dual coverage of a short-acting benzodiazepine in conjunction with an SSRI, with the goal of tapering the benzodiazepine.

One major problem with SSRI medications is that they can decrease sexual libido, which can be a frequent reason for discontinuation. Other adverse effects can include headaches, sweating, abdominal discomfort, and other less common adverse effects (refer to the Physicians' Desk Reference[11] for a full listing of adverse effects). The clinician is encouraged to educate the patient about these possible effects. To some clinicians the question of sexual activity may seem premature for many of these patients; however, it is important to inquire about sexual activity. Compliance with medications has its share of challenges in the best of situations, and if the patient sees decreased sexual libido as problematic, then it should be discussed openly in the clinical setting. In addition, the clinician needs to ask the patient directly about suicidal ideations.

When prescribing an SSRI, the clinician should be attentive to the importance of a slow titration upward and also downward to avoid serotonin syndrome, an adverse drug reaction resulting in excess serotonin. Symptoms of serotonin syndrome include seizures, restlessness, muscle twitches and myoclonus, confusion, exaggerated reflexes, sweating, and gastroenterological symptoms. Due to its long half life, fluoxetine (Prozac) is the only SSRI which is safe to discontinue abruptly. While this is not recommended, fluoxetine should be considered to be the first choice because of the significant risk of noncompliance in the adolescent population. Recommended follow up visits every week for 4 weeks, every other week for 4 weeks, and then again at 12 weeks. After 12 weeks, the clinician can

make a judgment concerning follow-up, but monthly visits for the next 2 months and then a minimum of every 3 months may be best. Last, SSRI medications can cause hypomania and behavioral disinhibition.[12,13] Behavioral disinhibition might include doing things that one thought about doing in the past but were not done, such as fighting, destroying property, running away, etc—in short, getting into trouble.[12,13]

If the clinician is going to treat an anxious patient, consideration should be given to placing strong demands on the family to go for psychological counseling so that a trained professional in the behavioral health field is also involved. In the case of mild anxiety, behavioral therapy alone might be sufficient. If a counselor or therapist sees something problematic with a patient, it can be addressed in a timely manner. In addition, the relationship between the mental health provider and the clinician is paramount in the successful treatment of these adolescents, not just for suicidal concerns or hypomania. The goal of treatment is to get the patient off medications. In our opinion, the best way to do that is to get these patients the psychological help they need. Medication is only a temporary treatment that will help to reduce anxiety in the patient and, therefore, improve counseling outcomes.

If the clinician decides to use SSRI medications, one can start adolescents on 10 mg of fluoxetine (Prozac [Eli Lilly and Company, Indianapolis, IN]) orally once per day. Because of its long half-life, abrupt discontinuation of fluoxetine is the least likely of the SSRIs to cause serotonin syndrome. Although abrupt discontinuation is not recommended, fluoxetine should be considered to be a good choice because of the risk of noncompliance in the adolescent population. A recommended follow-up visit schedule is every week for 4 weeks, every other week for 4 weeks, and then again at 12 weeks. After 12 weeks, the clinician can make a judgment concerning follow-up, but monthly visits for the next 2 months and then a minimum of every 3 months may be best. We strongly encourage the clinician to explore the possibility of hypomania as well as any other adverse effects at each follow-up visit.

If the patient is improved, one can stabilize the dose, but it is imperative that the clinician see the patient for follow-up, as per above. If there is no sign of relief in 2 to 4 weeks, the dose can be increased to 20 mg orally once per day. The adult maximum is 80 mg. If fluoxetine does not work, one can switch to a different SSRI medication such as fluvoxamine (Luvox, Jazz Pharmaceuticals, Inc, Palo Alto, CA), sertraline (Zoloft [Pfizer]), or paroxetine (Paxil [GlaxoSmithKline, Philadelphia, PA]).

If the adolescent's condition fails to respond to SSRIs and the patient has been diagnosed with uncomplicated anxiety disorder or determined to have a history of substance abuse, the clinician can consider buspirone (Buspar [Bristol-Myers Squibb, New York, NY]) as a potential treatment. However, it should be noted that buspirone is only recommended for patients older than 18 years. If the

clinician decides to use buspirone in adolescents, consider that the starting dose in adults is 5 mg 2 to 3 times per day or 7.5 mg twice per day, and it can be titrated with 5-mg increases per day every 2 to 3 days. The usual adult dose is 20 to 30 mg orally every day in divided doses, with a maximum of 60 mg each day. Buspirone is used in uncomplicated patients with generalized anxiety disorders whose conditions have failed to respond other anxiolytic agents or those who have a history of substance abuse.

Although there are several pharmaceutical treatment options available, the rapid onset of action of the benzodiazepines makes this class of medications the most efficacious for the treatment of acute intermittent anxiety.

HYPERVENTILATION SYNDROME

Hyperventilation is an extremely common problem in the adolescent patient. The chief complaint is usually a loss of consciousness with no apparent or precipitating event. However, most often, hyperventilation syncope will result from an anxiety-provoking event. Before making this diagnosis, there are several conditions that the clinician should rule out, including neurologic, cardiac, or respiratory problems and medications, drugs, or alcohol. In addition, the possibility of an epileptic event having caused the episode(s) should be considered. In the case of hyperventilation syncope, the adolescent may remember feeling light-headed or a sensation of darkening vision before losing consciousness. In the case of an epileptic event, it is unlikely that the patient will report these symptoms. If the patient describes true vertigo, this might be indicative of an epileptic event and should be explored as such. In addition, when exploring a syncopal episode the clinician should discern what the patient means by "black out" or "vision loss" as compared with a true loss of consciousness. Associated symptoms may include darkening of vision, tingling and numbness (especially of hands, feet, and face), stomachache, and increased heart or respiratory rate. The clinician can ask the patient, or one who observed the event, questions such as, "Tell me what you remember about the fall. Did you fall like a log or like a leaf?" Falling like a leaf implies a slower and more controlled fall and is more likely to imply that there was some sense of self-protection during the episode. In the case of hyperventilation syncope, the patient is usually aware that they are blacking out and is more likely to describe falling like a leaf. In the case of a seizure event, the patient is unlikely to remember the fall. Any description of such a fall is usually obtained by questioning an observer, who will commonly answer that the patient fell like a log. This type of fall would imply a true ictal event.

There may be pallor associated with the hyperventilation episode, but there will be no postictal phase. Those patients with hyperventilation syncope will have mental clarity within a few seconds of coming out of the episode. The history should explore where the patient was at the time of the episode (eg, in an emergency department, a dissecting laboratory, a dentist office). The clinician

can ask what the patient was doing when the episode occurred (eg, "Did you see blood?" "Were you watching a scary movie?" "Were you dissecting in biology laboratory?"). In what type of situation was the patient and what were the associated symptoms, if any? If the episode was observed by someone else, did the observer think that the patient was mentally clear within a short time after the episode occurred? Continuing to discern if this was an epileptic event, the clinician needs to question if there was any sort of tonic/clonic activity during the episode. Although tonic/clonic activity is possible during hyperventilation syndrome, it is not common.

Ask the patient if he or she was breathing heavily during the episode. Did heart rate increase? Although patients may not be aware of an increase in heart rate and/or of breathing quickly in the moments leading up to the event, when questioned the adolescent may describe that he or she had trouble catching his or her breath. One should also explore the possibilities of heart disease or respiratory problems in the patient or family.

It is important to ascertain if there have been any previous syncopal episodes or any diagnoses made at previous times. If so, was there any follow-up, or were any tests done? Question the patient as to any history of illicit drug use, anxiety, or stress. Be sure to ask what, if any, medications the patient is taking. As always, the clinician should have a healthy suspicion regarding possible drug or alcohol involvement in the episode. The clinician should ask questions about school (eg, "Are there any educational problems?" "Are your grades declining?" "Are you feeling pressure or stress from family or boy/girlfriends?").

Family history questions should include an exploration of epilepsy, cardiac conditions, or fainting. One might ask if there is anyone in the family who blacks out, passes out, or has seizures or "spells."

In the case of hyperventilation syncope, physical and neurologic examination results will be within the normal range. The best test to perform in the office setting is a controlled hyperventilation test to evaluate if one is able to reproduce the symptoms. During this test, patients are asked to hyperventilate and to not speak while they are hyperventilating but to answer all questions with a yes or no nod of the head and then to raise a hand when they are beginning to feel funny. At the time of feeling funny ask, "Are you feeling dizzy?" and "Is your vision darkening?" If yes, stop him or her from further hyperventilation. If no, have him or her continue to hyperventilate until symptoms appear or hyperventilation has gone on for 2 minutes. Afterward, ask the patient if the symptoms have been reproduced. Many patients will say yes, but the symptoms experienced in the office were not as severe as the event itself. Note that the event is short-lived, with a quick recovery.

An explanation to the patient of what is occurring might include the following: "When we breathe normally, the purpose is to increase oxygen and decrease carbon dioxide. When we hyperventilate, we lose excessive amounts of CO_2, and as CO_2 decreases, blood flow also decreases. Less blood flowing to the eyes causes a transient loss of vision, decreased blood flow to the brain causes a feeling of light-headedness, a decrease to the stomach causes a stomachache, and a decrease to the extremities causes a tingling sensation. If we hyperventilate enough, we pass out. In the passed-out state, one can no longer hyperventilate, and so the body auto-corrects."

For treatment, we recommend that the patient hold his or her breath to retain carbon dioxide; a brown paper bag is likely to be socially embarrassing. If the patient is experiencing frequent syncopal episodes and appears to be overly anxious, then we recommend psychological and chemical intervention as a cotreatment until the underlying issues are resolved. For a discussion on medications to be used for anxiety, see "Anxiety" above.

SYNCOPE

Syncope most often occurs in times of stress or anxiety. Triggers might include seeing blood, visiting a hospital, getting an immunization, going to an emergency department, or perhaps going to a dentist's office. Less commonly, a syncopal episode caused by a vasovagal response can be triggered by hair brushing (thus, "hair-brushing syncope"). The changes in blood flow from cephalad to caudad that occur with micturition can also result in a temporary loss of consciousness.

History questions begin with exploring the symptoms experienced by the patient before the episode. Did the patient feel cold or clammy with sweating? If observed by another, the clinician may question if the patient appeared pale before the syncopal episode. The clinician should discern where the patient was, what activity he or she was doing when the event occurred, and at what time the event occurred. Was the patient in a biology laboratory, say, dissecting at the time? Was there psychic or physical trauma? Was there visual trauma (eg, a gory movie)? The clinician should seek clues as to whether there was any associated anxiety with the syncopal episode. It is imperative that the clinician further explore even the "no" answers to any of these questions with follow-up questions as described above.

If someone else observed the event, the clinician should ask the observer how long the episode lasted and what, if any, changes occurred in the skin color of the patient during the episode. Did the patient go pale? Was the patient mentally okay before and after the event? As described in "Hyperventilation Syncope," the clinician can explore if the patient fell like a log or a leaf. The observer might respond that the patient seemed confused and did not know what had happened. The clinician should clarify what the observer means by confused. Importantly,

the clinician should ask the same questions of the observer to further delineate and clarify the episode. It is completely within the range of normal responses for a patient to have felt slightly disoriented at the time of the event. If questioned, the patient will usually report being aware of who was present at the time and any subsequent events. It is common for the patient to say, "What happened to me?" However, the clinician should discern the length and extent of the postepisode confusion.

In the case of complex syncope, the patient may have some motor or tonic-clonic activity during the episode and thus appear to be having a true seizure. This type of syncope is triggered most frequently when the patient receives an immunization or has a dental procedure. Complex syncope appears epileptic in presentation, but the preceding and following events will usually delineate or define whether the loss of consciousness was an electrical episode (seizure) or an anoxic episode (syncope). We strongly encourage the clinician to rule out a seizure disorder before making the diagnosis of complex syncope if the history of the event is not clear-cut.

In the differential diagnosis of an episode of loss of consciousness, the clinician should consider the possible presence of orthostatic hypotension. The diagnosis is in the history, and in almost all instances, the patient will report that symptoms occur when he or she goes from a lower to higher body positioning. In simple syncope, there will be no change in body position before the syncopal episode. In the case of simple or complex syncope or orthostatic hypotension, there should be no neurologic findings. On physical examination, particular attention should be given to cardiac, pulmonary, and vascular conditions that might exacerbate or induce the problem. It is our experience that orthostatic hypotension is not uncommon in the depressed adolescent. We strongly encourage the clinician to question the patient at length regarding any depressive symptoms that he or she might be experiencing.

There is no pharmacologic treatment for simple or complex syncope. The adolescent should either try to avoid trigger situations or learn techniques to deal with the problem.

SLEEP DISORDERS

Insomnia is defined as difficulty in maintaining or initiating sleep or have nonrestorative sleep for 1 month.[14] In all patients who suffer with insomnia, studies have shown that 40.5% have concomitant psychiatric disorders, and more than 50% of those will have anxiety disorders.[15] An adolescent who presents with a sleep problem should be questioned as to what exactly he or she is describing regarding difficulties sleeping. Is the patient having difficulty falling asleep? Does he or she describe falling asleep easily but is unable to stay asleep? Or perhaps the patient is having difficulty waking up after a night's sleep. As we

have highlighted, it is important that the clinician explore exactly what is meant by difficulty sleeping or sleep problems. Patients with brain damage such as cerebral palsy may often present with sleep disorders. Unfortunately, insomnia in those individuals with structural brain damage can be quite difficult to treat. The clinician should inquire about brain damage or recent brain trauma as a possible causative factor when an adolescent presents with sleep problems. It is important for the clinician to maintain a high index of suspicion regarding abuse of medications, drugs, and alcohol[16] or the consumption of caffeinated high-energy drinks such as Red Bull[17] or 5-Hour Energy.[18]

In the general population of those with insomnia, 8.6% are reported to have dysthymia, 14.0% have major depression, and 23.9% have anxiety.[15] With numbers as high as these, we encourage the clinician to screen for these psychiatric comorbidities. Of those with nonpsychiatric disorders, one should consider physical illness, restless leg syndrome, iron-deficiency anemia, and appropriate or inappropriate use of medications as possible contributing factors in sleep disorders.

After clarifying the actual problem, the clinician should take a thorough sleep history and question what times the adolescent goes to sleep at night and what time he or she awakens. Does the patient nap during the day (hypersomnia), and if so, at what times? Included in the causes of hypersomnias are physical illnesses such as mononucleosis, chronic fatigue syndrome, etc, sleep apnea, and medication use. It is important for the clinician to explore if the adolescent experiences night terrors, sleep walking, or sleep talking (parasomnias). Inquire as to any sources of stress that might be affecting the patient: Would the patient describe his or her family situation as a stable one? Is he or she in a new school? Is the patient experiencing trouble in school or with friends? External stressors are a common source of sleep disorders in the adolescent patient. The primary clinician needs to thoroughly explore the possibility of anxiety or depression to better discern the cause of the problem.[19] A sleep diary can be quite helpful in attempting to ascertain the causative factors of sleep disturbance in the adolescent patient.

The patient who has difficulty falling asleep can consider using a white-noise machine or bright-light therapy (2000–2500 lux for 2–3 hours starting from 6 AM) to induce somnolence and relaxation in the evening. Alternatively, the adolescent can be taught relaxation techniques that might assist with the problem. However, some patients experience a sleep-phase disorder with which it seems that their individual circadian rhythm is dysfunctional (circadian rhythm disorder). If behavioral therapies are not successful, the clinician may consider giving melatonin to the patient to help induce somnolence. Melatonin 1 to 3 mg orally 1 to 2 hours before sleep time has been shown to affect the circadian rhythm in adult patients, thereby inducing sleep.[20–22] Other medications may be used that cause sedation as an adverse effect (eg, diphenhydramine [Benadryl (McNeil PPC, Fort Washington, PA)] or chlorpheniramine [Chlor-Trimeton (Schering-Plough,

Kenilworth, NJ)]). Although clonidine (Catapres [Boehringer Ingelheim, Ingelheim, Germany]) is not recommended for the treatment of insomnia, it can be prescribed for those patients whose sleep problems are recalcitrant to over-the-counter medications.[23] Used as a sleep aid, the starting dose of clonidine is 0.025 to 0.05 mg orally, given 30 minutes before bedtime. This dose is given for 3 to 5 days. If results are not sufficient in this time, the clinician can increase the dosage by 0.05 mg every 3 to 5 days up to a maximum dosage of 0.3 mg each day. Clonidine and the antihistamines can cause the patient to feel tired or a little foggy, so the clinician should assess the daily routines of the patient, particularly as regards driving, when considering prescribing clonidine or other sedation-inducing medications. The patient should be instructed not to abruptly discontinue clonidine, because it may result in rebound hypertension. When a clinician is discontinuing this medication, it is suggested that it be tapered in a stepwise fashion to avoid this potential complication.[23] Although clonidine is a hypotensive agent, it is the experience of the authors that it rarely causes hypotension in normotensive adolescents. However, it is important that the clinician and the patient be aware of the possibility of an increase in hypotensive episodes while taking clonidine. Consideration should be given to pretesting the dose during the day to evaluate for any signs or symptoms of hypotension. When a patient is on clonidine, it is recommended that pulse and blood pressure be monitored every 2 weeks for the first 2 months and at 3-month intervals thereafter.[23]

The patient who is able to fall asleep but unable to stay asleep could be suffering from depression or a parasomnia. Although insomnia may trigger depression, it is also true that depression may trigger insomnia.[24] We strongly encourage any clinician who is evaluating an adolescent for sleep disorder to evaluate the patient for depression. One helpful piece of information regarding depression and sleep disorders is that depression usually causes awakening between 12 AM and 2 AM. Parasomnias are most likely to occur 90 to 120 minutes after the patient has fallen asleep.[25] The patient is likely to have no recall of an event of parasomnia, and the event does not usually interfere with a feeling of restfulness. With this in mind, it is clear that a diagnosis of a parasomnia can only come from an observer rather than from the patient. However, if a parent of the patient describes “something funny” occurring just after the adolescent has fallen asleep, the clinician should probe further and consider the possibility of an epileptic event. If the initial parasomnia presents after the age of 10 years, the differential should include the possibility of a complex partial seizure having occurred.

The patient who has difficulty waking up in the morning is usually going to sleep too late at night and, therefore, is not getting the necessary amount of sleep required to wake up fully refreshed in the morning. With appropriate history taking the clinician can likely rule in or out other illnesses that cause somnolence. A particularly common reason for difficulty waking up is that the adolescent is on the computer or telephone late into the night. Other common reason for this problem are a dislike of school or depression.

Table 1
Panic attack symptoms

- Palpitations, tachycardia
- Shortness of breath
- Chest pain or discomfort
- Nausea or abdominal distress
- Sweating
- Trembling and shaking
- Feeling of choking
- Dizziness, lightheadedness, or faintness
- Paresthesias
- Chills or hot flashes
- Derealization and depersonalization
- Fear of losing control or "going crazy"
- Fear of dying

Data source: "Psychopharmacology" course booklet. September 30–October 2, 2005. Boston, MA. Massachusetts General Hospital, Department of Psychiatry, Harvard Med-CME, PO Box 825, Boston, MA 02117:565

If the parent is trying to wake the adolescent and the youngster has trouble waking or is speaking gibberish or nonsense, the clinician should consider other possibilities, such as atypical migraine or seizure in the differential diagnosis. Headache on waking could be a resultant symptom of an ictal event or of migraine. If the patient went to bed with a headache, then migraine, as opposed to an ictal event, is the more likely cause of difficulty awakening. In the case of an ictal event, the clinician can explore the nature of the event itself and the seizure history of the patient and family and can proceed accordingly to evaluate the youngster for an epileptic disorder.

We strongly encourage any clinician to explore the possibility of a comorbid depression or a history of drug use in an adolescent patient who presents with a history of sleep disorder, because this may help to avert a serious crisis in the life of the patient.

PANIC

Panic disorder is a syndrome characterized by recurrent panic attacks (ie, discreet episodes of intense anxiety associated with at least 4 other symptoms of autonomic arousal and anxiety that develop rapidly and typically peak within 10 minutes)[26] (see Table 1).

Some patients have limited symptom attacks. Rather than experiencing 4 symptoms, these patients may experience 1 or 2 symptoms such as tachycardia, dyspnea, and/or light-headedness. These symptoms cause distress and disability and respond to the same treatments as those for a full-blown panic attack. Panic can have comorbid symptoms such as agoraphobia or claustrophobia, which can

function as triggers to an attack. Again, it is important to ascertain details regarding place of occurrence, situation, precipitating factors, and symptomatology when taking the history. If the patient describes an episode of panic that includes tachycardia or tachypnea, the clinician needs to explore if this was an isolated episode of panic or if a preexisting condition was reactivated.

Use of dietary and nutritional supplements should be explored, given that several supplements can be the cause of autonomic symptoms. Consider if the child is on a weight-loss regimen and perhaps using over-the-counter diet supplements that might contain stimulant-type medications. In the workup for panic disorder, consideration should be given to an electrocardiogram, resting blood pressure and pulse, and a thyroid screen, if indicated. If these test results are normal and the clinician is confident that there are no other metabolic causes, then therapy for panic disorder should be considered.

Treatment includes cognitive-behavioral therapy, benzodiazepines, and SSRI medications. For sporadic, longer-lasting but infrequent attacks, benzodiazepines are a good first choice. For chronic frequent episodes, either the benzodiazepines or SSRIs are helpful, although the latter are considered safer. For a fuller discussion of benzodiazepines and SSRIs please see the discussion on use of SSRIs in the adolescent population in "Anxiety."

OBSESSIVE COMPULSIVE DISORDER

Defined as pathologic levels of fears, phobias, and obsessions, OCD is reported to occur in 2% to 3% of all children and adolescents.[27] Many people have fears and phobias that do not impair their quality of life. However, adolescents who suffer from OCD have pathologic levels of fears, phobias, or obsessions that can impair their quality of life.

Diagnosing OCD requires a careful history, particularly because of the difficulty of detecting subtle differences between so-called normal obsessions or compulsions and those that cause impairment. An example would be the adolescent girl who might take 30 to 40 minutes to get dressed in the morning. It is easy to see that this example could well be considered typical behavior in an adolescent girl. However, on further questioning, one might find that this child has to have her hair absolutely perfect or she will not leave the house. The clinician is encouraged to explore if a particular behavior is rooted in fears and phobias. Another example is a child who does not want to leave for school. Is this because the patient is afraid that no one will be home when he arrives home, or is he worried that the bus will crash or perhaps that he will get on the wrong bus and get lost?

OCD in adolescents can present as a sleep problem. One might find that a patient is afraid to go to sleep because of a fear that he or she, or a family member, will not live through the night; or perhaps he or she is afraid of ghosts or thieves. Such

patients often experience teasing by family members because the fears and phobias seem to make no rational sense. Clinicians are encouraged to interview the patient separately from the family. Pathologic levels of fear and phobia can cause deep embarrassment for young people, and patients may or may not be forthcoming about the levels of terror they experience.

Although screening tools can be helpful in delineating OCD, it is important that the clinician consider the subjectivity of the person who fills out the diagnostic tools. The clinician is encouraged to screen the family for a history of fears, phobias, obsessions, and tics. As with many other disorders, the diagnosis of OCD is made largely on the history of the patient as well as the family history. The family history can be quite helpful, because OCD is generally an inherited condition. Adolescents who suffer with OCD have a greater chance of having comorbid tic disorder and attention-deficit/hyperactivity disorder (ADHD) than the general population. Given a high rate of comorbidities between these conditions, the clinician might wish to consider the possibility of a diagnosis of OCD in any adolescent diagnosed with ADHD or tic disorder and a diagnosis of ADHD in any adolescent diagnosed with OCD and tic disorder.[23]

Cognitive-behavioral therapy has been shown to be as effective, or more effective, than SSRI medications for patients with OCD. When using SSRI medication, the clinician should be aware of the black-box warning, as discussed earlier, regarding the possible increase in suicidal ideation. For the patient on SSRI medications, it is imperative that the clinician see the patient frequently for follow-up visits to assess for the presence of suicidal ideation. Fluoxetine (Prozac), sertraline (Zoloft), and fluvoxamine (Luvox) have all been approved by the US Food and Drug Association (FDA) for treatment of OCD in adolescents. Non–FDA-approved treatments for OCD in adolescents include paroxetine (Paxil), citalopram (Celexa [Forest Pharmaceuticals, Inc, New York, NY]), escitalopram (Lexapro [Forest Pharmaceuticals, Inc], the levo-isomer of citalopram), and clomipramine (Anafranil [Patheon Inc. Whitby, Ontario, Canada]).[9]

CONCLUSIONS

It is common for the internist or general practitioner to be on the front lines of treatment for anxiety and anxiety-related disorders. This article is intended to impart a basic working knowledge of the manifestations of anxiety as well as available treatments, thus providing the clinician with useful information and pointing out some of the pitfalls in making diagnoses.

REFERENCES

1. Larun L, Nordheim LV, Ekeland E, Hagen KB, Heian F. Exercise in prevention and treatment of anxiety and depression among children and young people. *Cochrane Database Syst Rev*. 2006;(3):CD004691

2. Smith C, Hancock H, Blake-Mortimer J, Eckert K. A randomised comparative trial of yoga and relaxation to reduce stress and anxiety. *Complement Ther Med*. 2007;15(2):77–83
3. Connolly SD, Bernstein GA; Work Group on Quality Issues. Practice parameter for the assessment and treatment of children and adolescents with anxiety disorders. *J Am Acad Child Adolesc Psychiatry*. 2007;46(2):267–283
4. Winerip M. Child anxiety that goes beyond the norm. *New York Times*. July 20, 2008. Available at: www.nytimes.com/2008/07/20/nyregion/nyregionspecial2/20Rparent.html. Accessed October 1, 2008
5. Food and Drug Administration. FDA public health advisory: suicidality in children and adolescents being treated with antidepressant medications—October 15, 2004. Available at: www.fda.gov/cder/drug/antidepressants/SSRIPHA200410.htm. Accessed August 13, 2008
6. Gibbons RD, Hur K, Bhaumik DK, Mann JJ. The relationship between antidepressant prescription rates and rate of early adolescent suicide. *Am J Psychiatry*. 2006;163(11):1898–1904
7. Are SSRIs safe for children? *Med Lett Drugs Ther*. 2003;45(1160):53–54
8. Which SSRI? *Med Lett Drugs Ther*. 2003;45(1170):93–95
9. Green WH. *Child and Adolescent Clinical Psychopharmacology*. 4th ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2007
10. Bolfek A, Jankowski JJ, Waslick B, Summergrad P. Adolescent psychopharmacology: drugs for mood disorders. *Adolesc Med Clin*. 2006;17(3):789–808; abstract xiii–xiv
11. *Physicians' Desk Reference*. Montvale, NJ: Thompson PDR; 2009
12. Ramasubbu R. Antidepressant treatment-associated behavioural expression of hypomania: a case series. *Prog Neuropsychopharmacol Biol Psychiatry*. 2004;28(7):1201–1207
13. Akiskal HS, Hantouche EG, Allilaire JF, et al. Validating antidepressant-associated hypomania (bipolar III): a systematic comparison with spontaneous hypomania (bipolar II). *J Affect Disord*. 2003;73(1–2):65–74
14. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. Primary Care Version. Washington, DC: American Psychiatric Association; 1994
15. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders. An opportunity for prevention? *JAMA*. 1989;262(11):1479–1484
16. Shibley HL, Malcolm RJ, Veatch LM. Adolescents with insomnia and substance abuse: consequences and comorbidities. *J Psychiatr Pract*. 2008;14(3):146–153
17. Red Bull Energy Drink. Ingredients. Available at: www.redbullusa.com/#page=ProductPage. Ingredients. Accessed August 8, 2008
18. 5-Hour Energy Drink. Home page. Available at: www.5hourenergy.com. Accessed August 8, 2008
19. Breslau N, Roth T, Rosenthal L, Andreski P. Sleep disturbance and psychiatric disorders: a longitudinal epidemiological study of young adults. *Biol Psychiatry*. 1996;39(6):411–418
20. Mindell JA, Emslie G, Blumer J, et al. Pharmacologic management of insomnia in children and adolescents: consensus statement. *Pediatrics*. 2006;117(6). Available at: www.pediatrics.org/cgi/content/full/117/6/e1223
21. Owens JA, Rosen CL, Mindell JA. Medication use in the treatment of pediatric insomnia: results of a survey of community-based pediatricians. *Pediatrics*. 2003;111(5 pt 1). Available at: www.pediatrics.org/cgi/content/full/111/5/e628
22. Shneerson JP. *Handbook of Sleep Medicine*. Oxford, United Kingdom: Blackwell Science; 2000
23. Leckman JF, Cohen DJ. *Tourette's Syndrome—Tics, Obsessions, Compulsions: Developmental Psychopathology and Clinical Care*. New York, NY: John Wiley & Sons; 1999
24. Riemann D, Voderholzer U. Primary insomnia: a risk factor to develop depression? *J Affect Disord*. 2003;76(1–3):255–259
25. Deray M. Management of parasomnias. *Int Pediatr*. 1997;12(3):161–163
26. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*. 4th ed. Primary Care Version. Washington, DC: American Psychiatric Association; 1994:47–63
27. Stewart SE. Questions & answers about OCD in children and adolescents. Available at: www.ocfoundation.org/UserFiles/File/Questions-Answers-OCD-In-Children-Adolescents.pdf. Accessed August 18, 2008