Current PSYCHIATRY

‘Night owls’: Reset the physiologic clock in delayed sleep phase disorder

Therapies can help adjust inherent ‘eveningness’ in drowsy young patients

CARL MENZ FOR CURRENT PSYCHIATRY

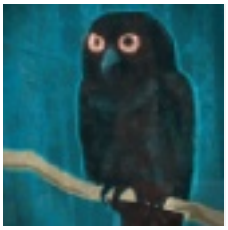
R. Robert Auger, MD
Assistant professor of psychiatry and medicine
Mayo Clinic College of Medicine
Consultant, Mayo Center for Sleep Medicine
Rochester, MN

Jason, age 16, has had difficulty with sleep initiation for 2 years. He describes going to bed at 10:30 PM on school nights but falling asleep no sooner than midnight and typically after 1:30 AM. He denies contributions from an “active mind” or environmental disturbances, and his bedroom contains no TV, computer, or other media devices. He does not sleep better with a change in environment. He denies pervasive low mood symptoms and believes his mood hinges predominantly on his ability to achieve sufficient sleep.

Once asleep, Jason generally enjoys good sleep consolidation until he needs to arise at 6:30 AM. His mother awakens him with difficulty, as he often sleeps through his alarm. He sleeps approximately 5 hours nightly during the school week, endorses impaired concentration, and often dozes during his first several classes. When he returns home from school, he finds it very difficult to resist napping.

On weekends he retires at 1 AM or later and typically falls asleep within 30 minutes. He usually awakens at noon but can sleep as late as 4:30 PM. He feels slightly more refreshed on weekends and describes his mood then as improved. During a recent spring break, he felt much better when allowed to sleep as much as he wanted.

Delayed sleep phase disorder (DSPD)—characterized by a pathological “night owl” circadian preference—is seen most commonly in adolescents and is associated with psychiatric morbidity, psychosocial impairment, and poor academic performance. Proper identification of the condition can be enhanced with a variety of assessment tools, and successful treatment



Delayed sleep phase disorder

Clinical Point

Studies link DSPD with personality and affective disorders, psychosocial impairment, and poor academic performance

Box 1

What causes delayed sleep phase disorder?

In individuals normally entrained to the light/dark cycle, circadian rhythms are:

- delayed by evening exposure to bright light (≥ 2,500 lux) prior to the core body temperature minimum (Tmin)
- advanced by morning light exposure after the Tmin.[1]

These opposing effects attune most people to the light/dark cycle, with sleep and wakefulness occurring on a conventional schedule. Persons with delayed sleep phase disorder (DSPD) live at a delayed phase that resists advancement and is incompatible with their personal and social obligations.

Theories have been proposed, but DSPD's etiology has not been fully explained. Affected adolescents may exhibit an extreme in circadian preference. Case reports also describe DSPD emerging after traumatic brain injury.[2]

Intriguing evidence supports various pathophysiologic explanations for DSPD. An abnormally long intrinsic circadian period (>25 hours) was recently demonstrated during temporal isolation in 1 individual with DSPD.[3] Both this case report and controlled studies describe deviations from expected relationships between the sleep/wake cycle and physiologic circadian markers. Most consistently described are longer intervals from Tmin[4] to sleep offset (final rise time) in DSPD patients compared with controls.

Other research suggests:

- hypersensitivity to nocturnal photic stimulation in select DSPD patients[5]
- impaired homeostatic sleep processes, as DSPD patients show a diminished ability to initiate sleep after sleep restriction, compared with controls.[6]

requires an awareness of potential endogenous and exogenous contributors.

This article describes what is known about DSPD and uses the case example to illustrate diagnostic assessment and treatment choices. Intriguing data support various pathophysiologic explanations for DSPD (***Box 1***).[1-6] Facilitating the adjustment of patients' physiologic clocks is the overall goal in managing DSPD.

Extreme 'eveningness'

Because of their extreme seemingly innate preference to retire and arise at relatively late clock hours (an "eveningness" trait), school-aged patients with DSPD represent a high-risk population for problematic sleepiness. In a survey of 612 high school students, the 63% who felt they needed more sleep on school nights showed a strong eveningness preference (as assessed by questionnaire), compared with students who described getting sufficient sleep.[7] Other studies have revealed psychiatric morbidity (including affective and personality disorders), psychosocial impairment, and poor academic performance associated with the condition.[8-10]

DSPD may affect 7% to 16% of patients presenting with insomnia complaints in sleep medicine clinics.[11] The condition appears most common among young cohorts and has been reported to affect up to 7% of adolescents in the United States.[12] Its high frequency in this age group may be a pathologic exaggeration of the normal tendency toward delayed timing of sleep and wakefulness linked with pubertal development.[13]

Sleep and wakefulness regulation

Conceptually, 2 processes govern sleep and wakefulness:

- The homeostatic drive to sleep (process S) is proportional to the duration of sleep restriction and becomes maximal at about 40 hours.
- Circadian regulation (process C) creates a drive for wakefulness that variably opposes process S and depends upon intrinsic rhythms.[14]

Neurons of the suprachiasmatic nucleus in the hypothalamus exert master coordination of this sleep/wake rhythm, along with other behavioral and physiologic variables.[15] Because the typical intrinsic period is slightly longer than 24 hours, synchronization to the 24-hour day (entrainment) is accomplished by environmental inputs (zeitgebers, or "time givers"), the most important of which is exposure to light.[16]

Misalignment between endogenous circadian rhythms and the light/dark cycle can result in circadian rhythm sleep disorders, such as:

- delayed sleep timing (DSPD)
- advanced sleep timing (advanced sleep phase disorder)
- erratic sleep timing (irregular sleep/wake rhythm)
- complete dissociation from the light/dark cycle (circadian rhythm sleep disorder, free-running type).

These 4 conditions are thought to involve predominantly intrinsic mechanisms, but circadian dysrhythmias also can be induced by exogenous factors. Extreme work schedules or rapid travel across time zones can challenge the circadian system's ability to acclimate and the individual's ability to achieve a desired sleep schedule.[17]

Differential diagnosis

Because DSPD relates primarily to an aberration in timing of sleep, it is characterized as a disorder only if the individual's preferred schedule interferes substantially with social or occupational functioning. The International Classification of Sleep Disorders (ICSD) provides detailed diagnostic criteria (***Table***).[17]

Depression and anxiety often manifest with sleep difficulties, as do inadequate sleep hygiene and other conditions associated with prolonged sleep initiation. According to ICSD criteria, primary insomnia can be differentiated from DSPD if the patient readily initiates and maintains sleep when allowed to sleep on his/her desired sleep/wake schedule. Accumulated evidence has largely debunked this notion, however, as polysomnographic studies have demonstrated both prolonged sleep latency and impaired sleep efficiency in DSPD patients versus matched controls.[3]

Assessment tools can complement the clinical history in diagnosing DSPD. Either a sleep log or actigraphy is required to demonstrate a stable phase delay, but actigraphy typically generates more reliable data.[18] Actigraphs are compact "motion detectors" whose output while being worn by patients allows longitudinal assessment of sleep/wake parameters.

Eveningness tendencies of presumptive DSPD patients can be further verified with the Morningness-Eveningness Questionnaire (MEQ) (***Box 2, page 61***).[19] Low scores are associated with evening types—felt to correspond to the endogenous circadian period—and can help narrow the differential diagnosis of sleep-initiation complaints.[20]

Table

Diagnostic criteria for delayed sleep phase disorder

A. Delay exists in the phase of the major sleep period in relation to desired sleep time and wake-up time, as evidenced by:
- a chronic or recurrent complaint of inability to fall asleep at a desired conventional clock time AND
- inability to awaken at a desired and socially acceptable time.

B. When allowed to choose a preferred schedule, patients exhibit normal sleep quality and duration for age and maintain a delayed but stable phase of entrainment to the 24-hour sleep/wake pattern.

C. Monitoring with a sleep log or actigraphy (including sleep diary) for at least 7 days demonstrates a stable delay in the timing of the habitual sleep period.

D. The sleep disturbance is not better explained by another sleep disorder, medical or neurologic disorder, mental disorder, medication use, or substance use disorder.

Source: Adapted and reprinted with permission from International classification of sleep disorders. Diagnostic and coding manual. 2nd ed[17]

CASE CONTINUED

'Definite evening type'

Jason scores 28 on the MEQ, consistent with a "definite evening type." Actigraphic monitoring is scheduled during a school holiday, when he is instructed to sleep according to his preferred schedule with the least possible restriction.

A clearly delayed sleep phase is evident, with the habitual sleep period occurring between 5 AM and 1 PM. Even on days when he was quite sleep-restricted because of an enforced wake time, sleep onset on the ensuing evening was substantially delayed, suggesting

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Clinical Point

DSPD is considered a disorder only if the individual's preferred schedule interferes substantially with occupational or social functioning

NSAIDs, Aspirin, and Warfarin)- Serotonin release by platelets plays an important role in hemostasis. Epidemiological studies of case-control and cohort design have demonstrated an association between use of psychotropic drugs that interfere with serotonin reuptake and the occurrence of upper gastrointestinal bleeding. These studies have also shown that concurrent use of an NSAID or aspirin may potentiate this risk of bleeding. Altered anticoagulant effects, including increased bleeding, have been reported when SSRIs and SNRIs are coadministered with warfarin. Patients receiving warfarin therapy should be carefully monitored when Pristiq is initiated or discontinued. **Ethanol-** A clinical study has shown that desvenlafaxine does not increase the impairment of mental and motor skills caused by ethanol. However, as with all CNS-active drugs, patients should be advised to avoid alcohol consumption while taking Pristiq. **Potential for Other Drugs to Affect Desvenlafaxine-**Inhibitors of CYP3A4 (ketoconazole)- CYP3A4 is a minor pathway for the metabolism of Pristiq. Concomitant use of Pristiq with potent inhibitors of CYP3A4 may result in higher concentrations of Pristiq. Inhibitors of other CYP enzymes- Based on *in vitro* data, drugs that inhibit CYP isozymes 1A1, 1A2, 2A6, 2D6, 2C8, 2C9, 2C19, and 2E1 are not expected to have significant impact on the pharmacokinetic profile of Pristiq. **Potential for Desvenlafaxine to Affect Other Drugs-** Drugs metabolized by CYP2D6 (desipramine)- *In vitro* studies showed minimal inhibitory effect of desvenlafaxine on CYP2D6. Clinical studies have shown that desvenlafaxine does not have a clinically relevant effect on CYP2D6 metabolism at the dose of 100 mg daily. Concomitant use of desvenlafaxine with a drug metabolized by CYP2D6 can result in higher concentrations of that drug. Drugs metabolized by CYP3A4 (midazolam)- *In vitro*, desvenlafaxine does not inhibit or induce the CYP3A4 isozyme. Concomitant use of Pristiq with a drug metabolized by CYP3A4 can result in lower exposures to that drug. Drugs metabolized by CYP1A2, 2A6, 2C8, 2C9 and 2C19- *In vitro*, desvenlafaxine does not inhibit CYP1A2, 2A6, 2C8, 2C9, and 2C19 isozymes and would not be expected to affect the pharmacokinetics of drugs that are metabolized by these CYP isozymes. **P-glycoprotein Transporter-** *In vitro*, desvenlafaxine is not a substrate or an inhibitor for the P-glycoprotein transporter. The pharmacokinetics of Pristiq are unlikely to be affected by drugs that inhibit the P-glycoprotein transporter, and desvenlafaxine is not likely to affect the pharmacokinetics of drugs that are substrates of the P-glycoprotein transporter. **Electroconvulsive Therapy-**There are no clinical data establishing the risks and/or benefits of electroconvulsive therapy combined with Pristiq treatment. **USE IN SPECIFIC POPULATIONS: Pregnancy-** Patients should be advised to notify their physician if they become pregnant or intend to become pregnant during therapy. Teratogenic effects – Pregnancy Category C- There are no adequate and well-controlled studies of Pristiq in pregnant women. Therefore, Pristiq should be used during pregnancy only if the potential benefits justify the potential risks. Non-teratogenic effects- Neonates exposed to SNRIs (Serotonin and Norepinephrine Reuptake Inhibitors), or SSRIs (Selective Serotonin Reuptake Inhibitors), late in the third trimester have developed complications requiring prolonged hospitalization, respiratory support, and tube feeding. Such complications can arise immediately upon delivery. Reported clinical findings have included respiratory distress, cyanosis, apnea, seizures, temperature instability, feeding difficulty, vomiting, hypoglycemia, hypotonia, hypertonia, hyperreflexia, tremor, jitteriness, irritability, and constant crying. These features are consistent with either a direct toxic effect of SSRIs and SNRIs or, possibly, a drug discontinuation syndrome. It should be noted that, in some cases, the clinical picture is consistent with serotonin syndrome [*see Warnings and Precautions (5.2)*]. When treating a pregnant woman with Pristiq during the third trimester, the physician should carefully consider the potential risks and benefits of treatment [*see Dosage and Administration (2.2)*]. **Labor and Delivery-** The effect of Pristiq on labor and delivery in humans is unknown. Pristiq should be used during labor and delivery only if the potential benefits justify the potential risks. **Nursing Mothers-** Desvenlafaxine (O-desmethylvenlafaxine) is excreted in human milk. Because of the potential for serious adverse reactions in nursing infants from Pristiq, a decision should be made whether or not to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother. Only administer Pristiq to breastfeeding women if the expected benefits outweigh any possible risk. **Pediatric Use-** Safety and effectiveness in the pediatric population have not been established [*see Box Warning and Warnings and Precautions (5.1)*]. Anyone considering the use of Pristiq in a child or adolescent must balance the potential risks with the clinical need. **Geriatric Use-**Of the 3,292 patients in clinical studies with Pristiq, 5% were 65 years of age or older. No overall differences in safety or efficacy were observed between these patients and younger patients, but greater sensitivity of some older individuals cannot be ruled out. For elderly patients, possible reduced renal clearance of desvenlafaxine should be considered when determining dose [*see Dosage and Administration (2.2) and Clinical Pharmacology (12.6) in the full prescribing information*]. **Renal Impairment-** In subjects with renal impairment the clearance of Pristiq was decreased. In subjects with severe renal impairment (24-hr CrCl < 30 mL/min) and end-stage renal disease, elimination half-lives were significantly prolonged, increasing exposures to Pristiq; therefore, dosage adjustment is recommended in these patients [*see Dosage and Administration (2.2) and Clinical Pharmacology (12.6) in the full prescribing information*]. **Hepatic Impairment-**The mean $t_{1/2}$ changed from approximately 10 hours in healthy subjects and subjects with mild hepatic impairment to 13 and 14 hours in moderate and severe hepatic impairment, respectively. No adjustment in starting dosage is necessary for patients with hepatic impairment.

OVERDOSAGE: Human Experience with Overdosage- There is limited clinical experience with desvenlafaxine succinate overdosage in humans. In premarketing clinical studies, no cases of fatal acute overdose of desvenlafaxine were reported. The adverse reactions reported within 5 days of an overdose > 600 mg that were possibly related to Pristiq included headache, vomiting, agitation, dizziness, nausea, constipation, diarrhea, dry mouth, paresthesia, and tachycardia. Desvenlafaxine (Pristiq) is the major active metabolite of venlafaxine. Overdose experience reported with venlafaxine (the parent drug of Pristiq) is presented below; the identical information can be found in the *Overdosage* section of the venlafaxine package insert. In postmarketing experience, overdose with venlafaxine (the parent drug of Pristiq) has occurred predominantly in combination with alcohol and/or other drugs. The most commonly reported events in overdosage include tachycardia, changes in level of consciousness (ranging from somnolence to coma), mydriasis, seizures, and vomiting. Electrocardiogram changes (e.g., prolongation of QT interval, bundle branch block, QRS prolongation), sinus and ventricular tachycardia, bradycardia, hypotension, rhabdomyolysis, vertigo, liver necrosis, serotonin syndrome, and death have been reported. Published retrospective studies report that venlafaxine overdosage may be associated with an increased risk of fatal outcomes compared to that observed with SSRI antidepressant products, but lower than that for tricyclic antidepressants. Epidemiological studies have shown that venlafaxine-treated patients have a higher pre-existing burden of suicide risk factors than SSRI-treated patients. The extent to which the finding of an increased risk of fatal outcomes can be attributed to the toxicity of venlafaxine in overdosage, as opposed to some characteristic(s) of venlafaxine-treated patients, is not clear. Prescriptions for Pristiq should be written for the smallest quantity of capsules consistent with good patient management, in order to reduce the risk of overdose. **Management of Overdosage-**Treatment should consist of those general measures employed in the management of overdosage with any SSRI/SNRI. Ensure an adequate airway, oxygenation, and ventilation. Monitor cardiac rhythm and vital signs. General supportive and symptomatic measures are also recommended. Gastric lavage with a large-bore orogastric tube with appropriate airway protection, if needed, may be indicated if performed soon after ingestion or in symptomatic patients. Activated charcoal should be administered. Induction of emesis is not recommended. Because of the moderate volume of distribution of this drug, forced diuresis, dialysis, hemoperfusion, and exchange transfusion are unlikely to be of benefit. No specific antidotes for desvenlafaxine are known. In managing an overdose, consider the possibility of multiple drug involvement. The physician should consider contacting a poison control center for additional information on the treatment of any overdose. Telephone numbers for certified poison control centers are listed in the Physicians Desk Reference (PDR®).

This brief summary is based on Pristiq Prescribing Information W10529C002, revised April 2008.

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Box 2

A morning or evening person? A self-assessment questionnaire

The Morningness-Eveningness Questionnaire (MEQ) developed by Horne and Ostberg[19] can be used to verify eveningness tendencies of patients with presumptive delayed sleep phase disorder. The MEQ is a 19-item self-assessment tool with responses that are assigned values totaling up to 86 points. Examples of the questions include:

- Considering only your own 'feeling best' rhythm, at what time would you get up if you were entirely free to plan your day?
- Considering only your own 'feeling best' rhythm, at what time would you go to bed if you were entirely free to plan your day?
- How easy do you find it to get up each day?
- When you have no commitments the next day, how much later do you go to bed compared to your usual bedtime?
- One hears about 'morning' and 'evening' types of people. Which ONE of these types do you consider yourself to be?

Lower scores are associated with evening types—felt to correspond to the endogenous circadian period—and can help in narrowing the differential diagnosis of sleep-initiation complaints.[20] Scores on the MEQ are interpreted as:

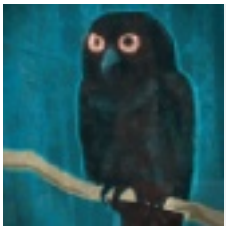
- 70 to 86: definite morning type
- 59 to 69: moderately morning type
- 42 to 58: neither type
- 31 to 41: moderately evening type
- 16 to 30: definite evening type

an obligate nature for the delayed sleep/wake schedule. Overall, Jason had few complaints with respect to impaired alertness while on this unrestricted schedule and experienced a much more stable mood.

Interventions

Without physiologic assessments, understanding the patient's "natural" sleep schedule can allow for rational recommendations about using phototherapy and oral melatonin (***Figure,***[21] ***page 62***). However, referral to a sleep specialist is required unless the general psychiatrist has experience in treating circadian rhythm sleep disorders.

Morning phototherapy. Properly timed morning bright light therapy (≥2,500 lux) has been shown to help DSPD patients achieve physiologically measured sleep phase advances, objective improvements in daytime alertness, and earlier reported bedtimes



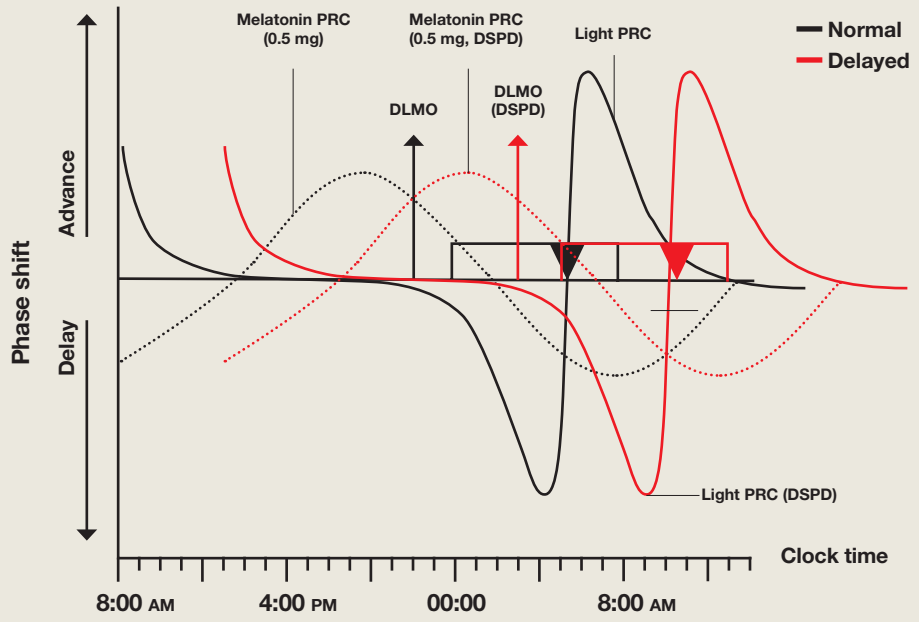
Delayed sleep phase disorder

Clinical Point

A rational approach is to recommend that patients take ≤0.5 mg melatonin 8 hours after their natural wake time

Figure

Light and melatonin phase response curves: Normal vs. delayed



This schematic compares 'normal sleep' phase response curves (PRCs) to light and exogenous melatonin with postulated PRCs for an individual with delayed sleep phase disorder (DSPD), presumed to be 5 hours 'out of phase.' Y-axis shows the direction and relative magnitude of phase shifts produced by light or melatonin at times shown on the x-axis. X-axis covers >24 hours to better illustrate the PRCs.

Relationships between 'normal sleepers' and DSPD patients are depicted by:

- rectangles (sleep period)
- triangles (core body temperature minimum [Tmin])
- arrows (dim light melatonin onsets [DLMOs]).

'Normal' sleep is shown to occur from midnight to 8 AM, and the DSPD patient's sleep from 5 AM to 1 PM; DLMO and Tmin are similarly delayed by 5 hours in the DSPD patient. This schematic assumes that phase relationships are maintained in DSPD patients, which is not a certainty.

Source: Adapted from reference 21

compared with controls.[22] Unfortunately, the described 2-hour treatment duration make this research protocol clinically impractical, and most clinicians commence with a 30-minute duration of therapy, as described in the seasonal affective disorder literature.

Relatively new and widely available blue light boxes have been reported to exhibit at least equivalent efficacy to bright light devices (as reported in the literature pertaining to seasonal affective disorder), but with markedly decreased light intensity and fewer associated adverse effects.[23] As the research addressing their use in the treatment of circadian rhythm sleep disorders is still emerging, their future role remains uncertain.

Precautions. Most psychiatrists would not perform a physiologic determination of a patient's circadian phase, and further undesired phase delays can occur if phototherapy is administered before the core body temperature minimum (Tmin).[24] Also, use caution if prescribing phototherapy to patients taking photosensitizing drugs and/or those with ocular or retinal pathology.[20]

Evening light avoidance. Whether or not you prescribe morning phototherapy, recommending that DSPD patients avoid evening light is essential to avoid further induction or exacerbation of phase delays. Protective eyewear is warranted in instances where these advisory precautions are insufficient (see *Related Resources, page*

65). Such an intervention has been shown effective in decreasing light exposure and undesired phase advances in studies involving subjects exposed to simulated shift work.[25]

Oral melatonin. Abundant evidence supports melatonin use in achieving phase advances in individuals with DSPD.[26,27] A synergistic effect can be obtained when melatonin is combined with phototherapy.[28]

Proper timing of melatonin to achieve a maximal phase advance can be estimated based on the individual's dim light melatonin onset (DLMO), which occurs approximately 14 hours after the habitual (unrestricted) wake time.[29] Maximal phase advances appear to occur when melatonin is given approximately 6 hours before the DLMO.[26] Thus, a rational practice is to recommend that patients take melatonin 8 hours after their natural wake time. Doses of ≤0.5 mg appear to achieve the maximal chronobiotic effect while avoiding an undesired hypnotic effect.[30]

Precautions. Verifying the purity of over-the-counter melatonin is difficult. A review by the National Academy of Sciences states that short-term use of melatonin, ≤10 mg/d, appears to be safe in healthy adults but recommends caution in children/adolescents and women of reproductive age. Doses recommended for circadian-based interventions are typically physiologic in nature (i.e., ≤0.5 mg), which may serve to mitigate these concerns.

Adverse effects such as headaches, somnolence, hypotension, hypertension, gastrointestinal upset, and exacerbation of alopecia areata have been reported at higher melatonin doses in healthy adults and at lower doses in persons with preexisting central nervous system, cardiovascular, gastrointestinal, or dermatologic conditions.[31]

CASE CONTINUED

Under the bright lights

Jason starts phototherapy treatment during his winter break, administering bright light daily upon natural awakening using a 10,000 lux light box for at least 30 minutes. As instructed, he gradually advances the time of administration by approximately 30 minutes every other day, striving for a nocturnal sleep period of 11 PM to 7 AM. He also wears protective eyewear to reduce light exposure during evening hours to avoid further delays in sleep phase. To further promote a phase advance, he takes oral melatonin, 0.5 mg/d at approximately 8 PM, as determined by his self-report and results of actigraphic recording.

Other options

Hypnotics. Little evidence supports the use of hypnotics in DSPD,[32] and patients may show resistance to these drugs.[33] Nevertheless, hypnotics can heighten confidence in the ability to initiate sleep in individuals with a concomitant conditioned insomnia.

With chronotherapy, patients are prescribed a sleep schedule that is delayed several hours incrementally until sleep is aligned to a target bedtime. The individual then is advised to rigorously maintain a regular sleep/wake schedule, repeating the process as necessary.

Although case reports have shown positive results with chronotherapy for DSPD,[34] no controlled trials have demonstrated its efficacy or safety. One study reported high relapse rates,[31] and 1 patient with DSPD developed free-running circadian rhythms.[35] Clinical experience suggests chronotherapy is impractical for patients who must adhere to a fixed schedule.

Behavioral approaches

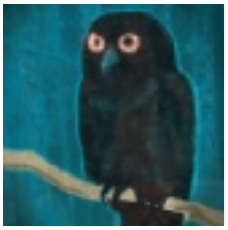
For an adolescent with DSPD, consider asking the school district to allow him or her a later school start-time. This alone often can substantially increase total sleep time and mitigate associated impairments.[36] In all instances pursue and address external contributors to DSPD, such as poor sleep hygiene (including excessive caffeine use) and substance misuse.

Emphasize regular wake times, as arising later on weekends can cause phase delays.[37] DSPD patients may have a concomitant conditioned insomnia that responds to evidence-based behavioral treatments.[38]

Whatever intervention you choose, schedule a follow-up appointment in

Clinical Point

Little evidence supports the use of hypnotics in DSPD, and patients may show resistance to these drugs' effects



Delayed sleep phase disorder

Clinical Point

Emphasize regular wake times, as arising later on weekends can cause sleep phase delays

approximately 2 months to evaluate patients' progress and compliance. Encourage them to contact you with questions or concerns in the interim. Review sleep logs or actigraphy during this visit, and adjust the timing and/or nature of interventions as needed. Adolescents can be particularly noncompliant with clinical interventions, and therapeutic goals cannot be reached without their full investment.

Because no guidelines exist on how long to treat DSPD, stop on a "trial-and-error" basis when symptoms are controlled, and resume if they recur. Another approach is to maintain a desired sleep/wake schedule with bedtime melatonin and encourage continued adherence to other measures.

CASE CONTINUED

Maintenance therapy

Jason returns to the clinic approximately 10 weeks later. After an obviously concerted effort to adhere to treatment, his progress is quite remarkable. He rarely falls asleep later than 11 PM, so he is obtaining 2.5 hours more sleep each night before arising for school at 6:30 AM. Sleepiness at school is rarely problematic, and his mood is more stable.

He nevertheless describes a persistent tendency to retire and arise later and asks to continue melatonin and phototherapy. Because no guidelines exist for long-term therapy of DSPD, he is advised to switch melatonin to bedtime dosing (with a presumed phase-neutral "maintenance" effect), and to continue phototherapy as prescribed.

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Bottom Line

The hallmark of delayed sleep phase disorder is a stable delay in a patient's main sleep period that interferes substantially with social or occupational functioning. For diagnosis, combine a careful history with use of a sleep log or actigraphy for ≥7 days. Phototherapy and oral melatonin have shown greatest benefit in advancing the habitual sleep period.

Related Resources

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Disclosure

Dr. Auger reports no financial relationship with any company whose products are mentioned in this article or with manufacturers of competing products.

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