

# When teenagers are out of sync. Case report teaching us what we should know about adolescents and Delayed Sleep Phase Disorder

Viki Vanderstraeten<sup>a</sup>, Nigitha van Doorn<sup>b</sup>, Sonia De Weerd<sup>c</sup>, Anna Jansen<sup>a</sup>

<sup>a</sup> Pediatric Neurology Unit, Department of Pediatrics, UZ Brussel, Brussels, Belgium

<sup>b</sup> Department of Clinical Psychology, UZ Brussel, Brussels, Belgium

<sup>c</sup> Department of Pneumology, UZ Brussels, Brussels, Belgium

Viki.vanderstraeten@uzbrussel.be

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## Abstract

We present 2 cases of delayed sleep phase disorder (DSPD) in adolescents. DSPD is the most frequent circadian rhythm sleep disorder, which often starts in teenage years. However, it frequently goes unrecognized or is misdiagnosed as attention deficit disorder or mood disorder. Incorrect diagnoses lead to inappropriate therapy and persistence of symptoms. DSPD in adolescents often results in a wide range of secondary problems, including school problems (poor school results, concentration problems, absenteeism), behavioral problems, mood swings, and substance abuse.

With this case report, we intend to increase awareness and recognition of this disorder.

## Introduction

The sleep-wake rhythm is regulated by the internal biological clock, which is located in the suprachiasmatic nuclei. In circadian rhythm sleep disorders (CRSDs), the sleep-wake rhythm is disturbed. Delayed sleep phase disorder (DSPD) is the most frequent CRSD (1-3). Adolescents appear to be particularly prone to DSPD. Approximately 90% of DSPD patients report onset of symptoms during adolescence. Prevalence of DSPD in adults is estimated around 1%. Studies in adolescents show a prevalence 3 to 4 times higher (4,5).

In DSPD, the sleep-wake rhythm is delayed, and no longer adapted to the daily school or work routine. DSPD is characterized by difficulty falling asleep at a preferred or socially acceptable time in the evening, and difficulty getting up in the morning. The delay is usually more than 2 hours. Because these patients fall asleep late, but still have to get up early in the morning to go to school, they develop a sleep deficit throughout the week. The short- and long-term consequences of sleep deprivation, both on physical as on mental health, are well known, the main concerns being poor scholastic performance and behavioral problems (6,7). Therefore, correct and early diagnosis is important. With the possibility of a DSPD in mind, a few targeted questions in the patient's history can help ensure that diagnosis is not unnecessarily delayed.

## Case Reports

### Case 1

A 16-year-old male presented with headaches, sleeping problems, and increased daytime fatigue.

The complaints of fatigue started 4 years ago after an infection with Epstein-Barr virus. Sleep itself was not disturbed at that time, but there was increased sleepiness during the day. Since then, fatigue complaints persisted.

Four months prior to consultation, the complaints worsened. Initiating sleep was especially difficult and took up to 3 hours. In addition, he developed sleep maintenance problems. In the morning, he did not feel rested and he felt depressed. He complained of headaches and a pronounced daytime sleepiness that required frequent naps. The complaints led to arguments with his parents, behavioral problems and school difficulties leading to important school absenteeism. There were

no other systemic complaints.

The family doctor and the infectious disease specialist carried out a medical work-up with blood analysis, which did not reveal any abnormalities. He was treated with ibuprofen for headaches and valerian for sleeping problems, with unsatisfactory results.

He was referred to the sleep clinic consultation for further examination and treatment.

Further enquiries about his sleeping habits revealed the following concerns. On weekdays, he went to bed around 10pm. He often lay awake for 2 to 3 hours before falling asleep. He slept very lightly and woke up 2 to 5 times per night. He had to get up at 6am for school, but had difficulty waking and did not feel rested in the morning. He got an average of 3 to 6 hours of sleep per night. During the day he was very tired, especially in the morning. As the days progressed, he felt better and had more energy. During school holidays, he went to sleep around 2-3am. He usually fell asleep within 10 minutes and slept until 12-13 in the afternoon. He felt better rested and had no headaches or other complaints.

Physical examination was normal. Mental neurological examination showed a cooperative young man, well oriented in time and space, without neurological deficit.

PSG showed a sleep onset latency of 30 minutes, an interrupted sleep (Wake After Sleep Onset (WASO) 247 minutes), a strongly decreased sleep efficiency (46%), and total sleep time which was too short (236 minutes). There were no arguments for other sleep pathology such as obstructive sleep apnea syndrome (OSAS) or periodic limb movement disorder.

The boy was diagnosed with DSPD with co-morbid insomnia.

Individual cognitive behavioral therapy for insomnia (CBT-I) with psychoeducation and restructuring of the sleep-wake rhythm was started. To support this, exogenous melatonin was started (Circadin® 2 mg), taken two hours before bedtime.

Over several weeks, his sleep phase was gradually and successfully shifted forward to a sleep-wake rhythm adapted to school and he became free of complaints. The data obtained from his sleep log also showed a clear improvement in sleep parameters (table 1).

**Table 1:** data from sleep log after start of therapy with exogenous melatonin

	Week 2	Week 3	Week 4	Week 5
Time in bed (min)	531	590	560	580
Total sleep time (min)	341	554	551	556
Sleep latency (min)	180	5.6	6.4	13
WASO (min)	10	58	2	11
Sleep efficiency (%)	64	94	98.5	96

**Case 2**

A 14-year-old female presented with difficulty initiating sleep, which started 5 years ago and gradually worsened. At time of consultation, it took about 2 hours to fall asleep. She subsequently developed sleep maintenance problems. At night, she often lay awake for an additional hour. She slept about 6 hours per night and did not feel rested in the morning. Throughout the day, she suffered from a high degree of fatigue, but around 8pm she had a peak of energy. On school days, she went to sleep around 9.30pm and got up at 6.45am. If she were to choose her own sleep schedule, she would go to sleep at 11pm and get up at 10.30am. During periods when she could maintain this rhythm, she did not experience any difficulty with sleep initiation, sleep maintenance, or next-day functioning. The girl had no other complaints apart from a chronic tendency to constipation treated with a laxative.

A polysomnography showed problems with sleep initiation and maintenance. Sleep structure was disturbed with a lack of REM sleep and persistence of Slow Wave Sleep towards the morning (fig 1). There were no arguments for other sleep pathology.

Based on her history, sleep logs and polysomnography, she was diagnosed with DSPD.

Individual CBT-I with psychoeducation was started. It was also advised to postpone bedtime with 30' to reduce sleep onset latency. Simultaneously, light therapy was started. After waking in the morning, a light intensity of 1000 lux was offered for 30 minutes with Luminette® glasses. After a few days the patient already felt more refreshed and less tired during the day. At bedtime she fell asleep within 15 minutes. The patient spontaneously reported that she no longer suffered from constipation.

**Discussion**

Both teenagers represent clear examples of DSPD. Nevertheless, diagnosis was delayed. Failure to recognize DSPD can lead to chronic insomnia and to a range of secondary pathologies, which can be prevented when diagnosis is made in a timely manner.

During consultation, sleeping habits should be questioned in detail, and a few specific questions in the patient's history can quickly orient the physician in the right direction.

When an adolescent has difficulties initiating sleep, it is important to not only ask about his or her sleep schedule on school days, but also to enquire about sleeping habits during school holidays. If there are no complaints when circumstances permit to sleep at the teenager's self-selected sleep schedule, the patient might have DSPD.

Another clue can be found by looking at the evolution of complaints throughout

the day. Most individuals with DSPD complain of excessive sleepiness mainly in the morning, and begin to feel more awake and energetic as the day progresses.

In addition to a targeted patient history, the sleep schedule should be mapped using a sleep log or an actigraphy. Although there is agreement that sleep logs can be very useful in the assessment of suspected CRSDs, the lack of widely accepted standardized sleep logs, makes their use less reliable and less valuable. An actigraphy records a sleep schedule in a more objective manner, but might not always be available in all settings.

A targeted patient history, supplemented with a sleep log and/or an actigraphy, is often sufficient for a diagnosis of DSPD. Although PSG is not routinely indicated, it can be useful when other sleep pathology is suspected, e.g. to exclude OSAS in case of snoring.

Sleep timing can mostly be shifted forward with phototherapy and/or exogenous melatonin. Both treatments are safe and, in most cases, successful in the short term (3,4,8).

Studies have shown that relatively small doses of melatonin (from 0,2mg to a maximum of 5mg for adolescents) and advance administration (3 to 4 hours prior to bedtime) are more effective for DSPD than larger doses or those given closer to sleep onset (10). Phototherapy may be of particular benefit to patients with DSPD who suffer from pronounced morning sleepiness, or in patients who are somewhat reluctant to take medication.

In more severe cases of DSPD, chronotherapy with time shifting in a delay direction can be necessary.

Long-term success is often co-determined by circumstances and requires a sustained strict sleep structure.

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