For patients: *(expands on click)*

Table of Contents: (expands on click)

Chapter : Pediatric Sleep Issues

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For physicians, trainees and other health care providers: *(expands on click)*

Abstract (<250 words, expands on click): Pediatric sleep disorders are quite common and often disturbing to either the patient or the child's family. As the patient matures into adolescence, sleep disorders continue to be common and an important factor in development, both social and cognitive. Sleep disorders can adversely impact physical and mental health. Non-restorative sleep can hamper a child's ability to concentrate and control emotions and behavior. Sleep disorders vary among age groups, but most can occur with varying frequency at any age.

Several disorders are typically seen only during the first few years of life, including colic, excessive nighttime feedings, and sleep onset association disorder. A number of conditions are common during childhood but begin to improve as the child ages. The non-REM sleep parasomnias, including sleepwalking, confusional arousals, and night terrors, are the most common in the pediatric category. Nightmares are also common in childhood but can occur at any age.

Sleep-related breathing disorders including obstructive sleep apnea, central sleep apnea, central alveolar hypoventilation syndrome, and Cheyne-Stokes respirations are not found only in adults but are, in fact, quite common in the pediatric population. While these disorders can occur at any age, treatment options vary substantially by age.

Clinical Outlook:

Summary (<250 words, expands on click):

DISORDERS DURING THE FIRST 3 YEARS OF LIFE

The most common sleep-related problems for children between ages 6 months and 3 years arise because of difficulty initiating or maintaining sleep.1 Numerous factors have been implicated in

the occurrence of repetitive nocturnal waking and inability to fall asleep: infant temperament, nutrition, physical discomfort, mild allergy, and parental marital conflict.2,3

Topic discussion (up to 2 pages, expands on click**):**

Sleep-Onset Association Disorder

Clinical features

Sleep complaints in the infant and young child usually come from the parents, not the child. Nighttime awakenings often become worrisome to parents. However, as might be expected, the problems often reflect certain established patterns of interaction between the parent and the child at time of sleep transition. Nighttime arousals are very common in all ages; however, older children and adults are usually unaware of these disruptions.

Causes/pathogenesis

Parents may incorrectly conclude that nocturnal awakenings are abnormal, and become involved in the sleep transition process. The child may then become accustomed to parental intervention and become unable to make the transition back to sleep alone. This is known as sleep-onset association disorder. The child is then reliant on the parent to help complete the sleep transition regardless of the time of night.

Diagnosis and treatment

Diagnosis is made with a careful history. Children with this disorder usually respond rapidly to simple gradual behavioral interventions, which helps the child learn a new set of sleep- associated habits.4

Difficulties Learning to Sleep Alone

Clinical features

The ability to sleep alone throughout the night without parental intervention is a learned process. Children typically awaken 5 to 8 times per night, at the end of each sleep cycle, but some children are able to put themselves back to sleep without parental awareness or intervention. Most infants are capable of learning this process by about 5 to 7 months of age (1).

Diagnosis and treatment

The key to this process is to gradually withdraw the amount of parental involvement at sleep onset. This same parental behavior response is required for middle-of-the-night awakenings. Consistency is of critical importance if the treatment plan is going to work, especially in conditioning the child to sleep throughout the night. When fear is affecting the progression of this process, it is important to address the child's and/or parent's anxiety effectively. Fear on the part of the child can prevent sleep. In such cases, it is important for the parents to problem-solve about their child's fear and how to best accommodate the behavioral treatment plan. Fear regarding the safety of one's child can alter a planned behavioral intervention. Parents must be aware of their own fears and anxieties so that these do not cause the sleep disturbance in the child.

Excessive Nighttime Feedings

Clinical features

An increase in nighttime awakenings among infants and toddlers may be related to nighttime feedings. Infants fed large quantities at night (8-32 oz) tend to have frequent awakenings, ranging up to eight per night (4-7). Repeated awakenings for feeding disrupt the functioning of circadian-modulated systems, which may cause further deleterious effects on sleep-wake stabilization (4,8,9).

Diagnosis and treatment

Diagnosis relies on a characteristic history: multiple nocturnal awakenings, return to sleep only with feeding, significant fluid intake during the night, and extremely wet diapers. Treatment consists of a gradual decrease in the frequency of feedings during the night (4). Frequent awakenings, three or more per night in a child over 6 months of age, may cause sleep fragmentation that has a deleterious effect on a child. As feedings decrease and associated habits are eliminated over several weeks, sleep consolidation usually promptly occurs (7).

Limit Setting

Clinical features

Inability to set limits at bedtime can also cause sleep deterioration. Typical bedtime struggles may consist of requests for water, stories, use of the bathroom, and adjustment of lights (4,7).

A diagnosis of this sort can be made from the history alone. The parents are typically unable to enforce nighttime rules with enough consistency to keep the child in bed and quiet so that he or she can initiate asleep.

Diagnosis and treatment

Parents have to learn to be firm in their limit setting, enforcing a regular bedtime ritual with sleep onset as the goal. The child should also be kept in his or her bedroom with the use of a gate or closure of the door if necessary. Positive behavior modification using techniques such as a sticker or star chart, as well as other prizes for staying in bed, may elicit a positive response (4).

Fear

Clinical features

Fear and nightmares are also common in early childhood, and represent an element of normal development. A truly anxious child at night should be handled in the same manner whether the child's fears were initially expressed during waking or sleep.

Diagnosis and treatment

Mild fears often respond to supportive firmness and a stable social setting. Positive reinforcement, with rewards for staying in bed, may help motivate the child. Treatment may also consist of sleep schedule correction, progressive relaxation (10), and progressive desensitization (11). Only in rare instances are medications indicated.

Colic

Clinical features

Colic is the most common medical condition affecting the sleep of young infants. It causes an inconsolable fussiness and crying, typically in the late afternoon and evening. Although symptoms usually resolve spontaneously by 3 to 4 months of age, the sleep disturbances often persist, secondary to altered sleep schedules and habitual patterns of the parental responsiveness (7).

Diagnosis and treatment

Colic is diagnosed when there are unexplained spells of crying in healthy infants. Treatment mainly focuses on education and management strategies for helping the parents cope with the stresses of caring for the infant (12).

Summary (<250 words, expands on click):

PARASOMNIAS—NOCTURNAL EVENTS

In the course of clinical practice many unusual nocturnal phenomena may be described by the child or parents. The correct diagnosis can usually be ascertained from the clinical history alone but in some cases video polysomnography may be necessary. Additional EEG leads should be used if a seizure disorder is suspected and additional EMG leads can be useful in patients with movement disorders.

Nocturnal movement disorders are extremely common in the pediatric population. In some cases, these events are so common that they may be considered a normal component of childhood and are usually “outgrown”.

Topic discussion (up to 2 pages, expands on click**):**

Restless Legs Syndrome

Clinical features

Restless legs syndrome is a disorder composed of four principal diagnostic criteria; 1) Intense, irresistible desire to move limbs, usually with uncomfortable feeling in the limbs, 2) Symptoms worsen with decreased activity, 3) Symptoms improve with activity, and 4) Symptoms are typically worse at night. (13) Restless legs syndrome may cause significant sleep onset insomnia.

Adult patients often have difficulty describing the symptoms. Children have even greater difficulty describing the symptoms and are frequently ignored by adults (including physicians). Patients may described the subjective symptoms of RLS in a number of ways including creepy, painful, burning, aching, electrical, crawly, tingly, like worms or bugs crawling under the skin, etc. In children, these symptoms can easily be mistaken for “growing pains”. Children may get into trouble at school or at home because they can have difficulty sitting still. Restless legs syndrome is significantly underdiagnosed or mis-diagnosed because of these factors.

Epidemiology

Restless legs syndrome has an age-adjusted prevalence of up to 10% of adults. While it is less common in children and increases with increasing age, the syndrome is underdiagnosed in children. The symptom severity also typically worsens with increasing age. Primary restless legs syndrome is a genetic disorder with an autosomal dominant pattern. Secondary restless legs syndrome, associated with a precipitating factor, is less common in younger patients. Renal failure, iron deficiency, and diabetes may contribute to the restlessness. In children, growing pains may mimic or cause restless legs.

Evaluation

The laboratory evaluation of RLS includes serum ferritin, screening for uremia and screening for diabetes. Low normal ferritin levels (20-60) may be associated with RLS and frequently respond to treatment with iron. (14) Other vitamin, hormone and mineral derangements can also contribute to the symptoms. Polysomnography is not indicated in the evaluation of RLS, unless there is suspicion of a comorbid sleep disorder.

Treatment

Dopamine agonist therapy is the mainstay of RLS treatment in adults. No agents have been FDA approved for treatment of RLS in children. Fortunately, the use of simple non-pharmacological therapies may be of significant benefit including teaching the child to visualize an activity or simply allowing the child to move/swing the legs. Teachers should be informed of the condition. The fact that it is not a form of attention deficit disorder should be reinforced. Symptoms may be caused by an underlying iron or vitamin deficiency and supplementing with iron, vitamin B_{12}, or folate (as indicated) may be sufficient to relieve symptoms in these specific cases.

Periodic Limb Movement Disorder

Clinical features

Periodic limb movement disorder (PLMD) is “characterized by periodic episodes of repetitive and highly stereotyped limb movements that occur during sleep” (ICSD (15)). While these movements usually occur in the legs they can also occur in the arms. There is typically extension of the toe and flexion of the ankle, and possibly the knee and hip as well. Most patients are unaware of the

movements. The sleep disruption associated with the movements can lead to insomnia or daytime somnolence. There is a repetitive increase in EMG activity (most often measured over the anterior tibialis muscle) lasting 0.5 to 5 seconds. The periodic limb movement index is the total number of periodic limb movements divided by the total hours of sleep. A PLM index over 5 is considered abnormal. Periodic limb movements may be associated with arousals. While many assume that the higher the PLM-arousal index the more like one is to suffer from daytime sleepiness this has not been proven (16).

Epidemiology

Periodic leg movements often accompany restless leg syndrome (RLS), narcolepsy and obstructive sleep apnea. While all patients with PLMD and most patients with RLS have periodic limb movements on a sleep study only the RLS patients have the daytime annoying sensations in their limbs that improve with movement. Use of caffeine, neuroleptics, alcohol, monoamine oxidase inhibitors or tricyclic antidepressants can cause periodic limb movements. Withdrawal of benzodiazepines, barbiturates and certain hypnotics can also cause or aggravate periodic limb movements. Periodic limb movements are reportedly rare in children but increases in prevalence with age.

There are several conditions that mimic periodic limb movements. Sleep starts or hypnic jerks are frequently mentioned by patients. These occur in drowsiness, may be associated with a feeling of falling and do not recur repetitively during sleep. Seizures can cause nighttime kicking movements but may also cause nocturnal enuresis, morning musculoskeletal soreness, or bleeding from oral laceration. An expanded additional 16 lead EEG on the polysomnogram is invaluable in identifying these patients.

Treatment

There are no FDA approved treatments for PLMD in children. Limiting the consumption of caffeine can improve or completely control periodic limb movements. Limiting the use of other aggravating substances and medications is also important. Adequate treatment of underlying sleep disorders including obstructive sleep apnea and RLS can improve the limb movements.

Rhythmic Movement Disorder

Clinical features

Rhythmic movement disorder (RMD) “comprises a group of stereotyped, repetitive movements involving large muscles, usually of the head and neck; the movements typically occur immediately prior to sleep onset and are sustained into light sleep” (ICSD (15)). This can be manifest as repetitive head banging, leg banging or body rolling. The movements typically begin during drowsiness. Movements typically occur with a frequency of 0.5 to 2 times per second. While this is very common in normal infants, it is sometimes associated with a static encephalopathy, autism or psychopathology in older children and adults. It appears to be more common in males. The movements are thought to have a self-soothing effect for some individuals. The noise from the movements can be disturbing to family members. While injuries, even serious injury such as subdural hematoma, are possible, they are not common. It is very important to have the technologist accurately document what was seen at the time this occurs in the sleep laboratory. Continuous video monitoring usually easily confirms the diagnosis.

The differential includes nocturnal seizures, masturbation, bruxism, and periodic limb movement disorder. Bruxism and PLMD are usually easily distinguished on the sleep study. Gasping respirations from sleep apnea can cause rhythmic movements.

Treatment

The primary treatment is to ensure the safety of the patient. The family should be counseled about the diagnosis.

Nocturnal Bruxism

Clinical features

Nocturnal bruxism is “a stereotypical movement disorder characterized by grinding or clenching of the teeth during sleep” (ICSD (15)). This often leads to abnormal destruction of the surface of teeth which may first be noticed by a dentist. It often causes headaches or jaw and facial pain. Its prevalence has been estimated at 5-20 percent or even higher (17). It is relatively common in patients with a static encephalopathy (18). It occurs equally in males and females. Most people with bruxism are of normal intelligence. While a link has been questioned with anxiety and psychosocial stress, psychological problems are not more common in patients with bruxism. There is a familial tendency toward bruxism. Temporal-mandibular joint dysfunction and

malocclusion are sometimes accredited as being an underlying cause or result of bruxism. There is no guarantee that correction of these abnormalities will cure bruxism in a particular individual. It can occur in all stages of sleep and is often disturbing to family members. Rhythmic muscle artifact is usually noted on most electrodes placed on the head during polysomnography.

The only significant differential diagnosis is a seizure disorder. Seizure disorders can cause masticatory movements in some individuals. Usually, there is additional history to lead to this diagnosis.

Treatment

The primary treatment is to protect the teeth with a bite block if necessary.

REM Sleep Behavior Disorder

Clinical features

REM sleep behavior disorder (RBD) is characterized “by the intermittent loss of REM sleep electromyographic atonia and by the appearance of elaborate motor activity associated with dream mentation” (ICSD (15)). The patient physically acts out a dream, leading to a variety of movements and actions and some episodes can be violent. It is more common in males. Although it can be seen at any age it is most prevalent in the sixth and seventh decade, occurring more frequently in patients with Parkinson’s disease. REM sleep behavior disorder is uncommon in childhood but may be seen in patients with narcolepsy.

The polysomnogram shows episodes of sustained increased muscle tone in REM sleep instead of the decreased tone normally seen at this time. The polysomnogram should be preformed with continuous time-locked video. The video may show movements including punching and guttural utterances. If carefully awakened during an episode, the patient may, in some cases, recall the content of the dream and a reason for the movements can sometimes be ascertained. There is often an increase in NREM periodic limb movement index and in the REM density.

A careful general medical and neurological history is necessary. Tricyclic antidepressants and other anti-cholinergic medications may lead to RBD symptoms. There are also reports of transient RBD symptoms following hypnotic or alcohol withdrawal. The differential includes nocturnal

seizures. Concomitant 16 channel EEG can be useful in this situation. Another REM related parasomnia, the nightmare, is sometimes confused with RBD. A nightmare is a frightening dream that often awakens the sleeper. Rarely, striking out can be part of a nightmare. RBD patients tend to be more explosive and usually do not awaken with the frightening aspect so common in a true nightmare. The differential also includes other NREM parasomnias including sleep walking, confusional arousals, and sleep terrors.

Sleep Terrors

Clinical features

Sleep terrors are “characterized by a sudden arousal from slow-wave sleep with a piercing scream or cry, accompanied by autonomic and behavioral manifestations of intense fear” (ICSD (15)). Various autonomic phenomena may occur, including tachycardia, mydriasis, diaphoresis, and flushing. Patients often sit up in bed and scream inconsolably. The patient typically appears fearful and is difficult to awaken. Once awakened, the child often seems confused. While some type of dream may be recalled, it is often simple, fragmented, has no plot and usually makes no sense. The patient is amnestic for the event.

It is usually seen between ages 4 and 12 years of age, occurring in approximately 3% of children. In rare cases, sleep terrors may persist into adulthood. Like most NREM parasomnias, it usually disappears in adolescence. It is more common in males than in females. Other family members often have a history of a NREM parasomnia (19). Sleep terrors begin in slow wave sleep, and are therefore more common in the first third of the night, but can happen anytime during the night.

The differential diagnosis is broad and includes nightmares, confusional arousals, and epileptic seizures. When people awaken from nightmares, they are usually clear of mind and often can remember a dream with some detail. While some children may remember a simple image when awakened from a sleep terror, there is no frightening story such as with a nightmare. Nightmares are more common in the last third of the night where REM sleep is more prevalent. Nightmares typically have less associated autonomic phenomena than do sleep terrors. If there is a partial arousal during slow wave sleep the person often seems stuck in a confused state without the fear seen in sleep terrors. This is called a confusional arousal. These people also do not have the autonomic phenomena represented in sleep terrors. Epileptic seizures can present with a cry and

the patient can be confused afterward. Ictal fear can be seen in certain epileptic syndromes. Most epileptics do not have seizures solely in sleep. Focal dystonic posturing or tonic-clonic activity points to a seizure as the likely diagnosis. In some cases, continuous video-EEG monitoring is needed to distinguish a night terror from a nocturnal seizure.

Treatment

The primary treatment is to ensure the safety of the patient. The family should be counseled about the diagnosis.

Confusional Arousals

Clinical features

Confusional arousals "consist of confusion during and following arousals from sleep, usually from deep sleep in the first part of the night" (ICSD (15)). Patients may not respond at all or when they do, the response is inappropriate and they are usually amnestic for the event. Confusional arousals usually arise in the first third of the night from slow wave sleep (Figure 1). They are sometimes associated with incontinence. Typical of most NREM parasomnias, confusional arousals are common in young children and usually disappear with adolescence. While usually seen in children, it can be seen in adults when there is interference with awakening. Examples include sleep deprivation, metabolic encephalopathies, and use of medications that suppress the central nervous system. It is seen equally in both sexes. There is a familial predisposition to NREM parasomnias in general.

The differential includes sleep terrors, sleepwalking and nocturnal seizures. Sleep terrors are associated with a frightful scream and more autonomic phenomena as described above. Sleepwalking is very similar to confusional arousals except that patients get up and walk with sleep walking. Most epileptics with nocturnal seizures also have diurnal seizures. Video-EEG monitoring may be necessary to distinguish nocturnal seizures from parasomnias.

Treatment

Reassurance for the patient and the patient's family is the most important component of care. In rare cases pharmacotherapy may be necessary. It was recommended that children with confusional arousals and sleeping walking may be safer not using a bunk bed.

Nocturnal Epilepsy

Clinical features

Epilepsy is “a disorder characterized by an intermittent, sudden discharge of cerebral neuronal activity” (ICSD (15)). Almost any seizure type can occur during sleep. (20) In some epileptic syndromes, the seizures occur primarily during sleep (e.g. benign epilepsy with central-temporal spikes or Rolandic epilepsy and nocturnal frontal lobe epilepsy). The manifestation of the seizure depends on its neuroanatomic origin. Generalized tonic-clonic seizures are associated with loss of awareness, tonic flexion and then extension, a forced expiratory “cry”, and then clonic rhythmic jerking of the extremities. Focal (partial) seizures may or may not be associated with alteration of consciousness but are associated with unilateral sensory or motor phenomena. Automatisms consisting of repetitive picking movements or lip smacking may be seen. Some partial seizures can secondarily generalize. Sleep deprivation, noncompliance with anti-epileptic medication, fever and alcohol can contribute to breakthrough seizures. Epilepsy can be idiopathic or symptomatic of an underlying discernable brain lesion. The lesions could be a tumor, stroke, brain dysgenesis, hippocampal sclerosis or due to post-traumatic changes. The EEG may show generalized, bilateral synchronous spike and wave activity or generalized polyspike activity in patients with generalized seizures. The EEG often shows focal, regional epileptiform activity including spikes or sharp waves and focal slowing of background activity in patients with focal (partial) seizures. Focal epileptiform activity is more common in NREM sleep and suppressed in REM sleep. Epileptiform activity is much more common in sleep than wakefulness in children with Rolandic epilepsy. A diurnal EEG may be all that is needed to confirm the diagnosis. An EEG after sleep deprivation or overnight continuous video-EEG may be needed in more complicated cases. Sleep deprivation from other sleep disorders such as sleep apnea has been shown to worsen seizures in some patients.

The differential includes nocturnal paroxysmal dystonia, sleepwalking, rhythmic movement disorder and REM behavior disorder. Nocturnal paroxysmal dystonia occurs in a short form (15-60 seconds) and a longer form (up to 60 minutes). It is characterized by repeated stereotypical dyskinetic episodes of ballismus or choreoathetosis often associated with vocalizations in NREM sleep (21). Sleepwalking, rhythmic movement disorder and REM behavior disorder are not associated with epileptiform activity.

Parasomnias

	Sleep Terror	Nightmare
Prevalence	Uncommon	Common
Sleep stage	SWS	REM
Onset	First 90 minutes of sleep	Second half of night
Features	Intense; vocalization, fear, motor activity	Less intense; vocalization, fear, motor activity
Mental content	Sparse	Elaborate
Violent behavior	Common	None
Injury	More likely	Unlikely
Amnesia	Often	Rare
Ability to arouse	Difficult	Easy
On awakening	Confused	Oriented

	Confusional Arousals	Sleep Walking	REM Sleep Behavior Disorder
Prevalence	Uncommon	Common	0.5%. More common in Parkinson's, MSA
Sleep stage	SWS	SWS	REM
Onset, typically	First 1/3 of the night	First 1/3 of the night	Last 1/3 of the night
Features	Complex behavior. Slow confused	Complex behaviors not limited to walking.	Acting out dreams. May be violent. Increased EMG

	speech.		tone.
Violent behavior	Occasional	Rare	Frequent
Injury	Rare	Rare	Occasional
Treatment	Benzodiazepines	Benzodiazepines. TCA's.	Clonazepam. Carbamazepine.

Tables adapted from "Neurology for the Boards", 3rd Edition Eds. JD Geyer, J Keating, Potts PR Carney, Lippincott Williams & Wilkins, (2006).

Summary (<250 words, expands on click):

Disorders of excessive daytime sleepiness.

Topic discussion (up to 2 pages, expands on click**):**

Narcolepsy

Clinical features

Narcolepsy is a disorder of excessive sleepiness with a loss of control of the boundaries between sleep and wakefulness. The classic tetrad of symptoms defining narcolepsy includes 1) excessive daytime sleepiness, 2) cataplexy, 3) sleep paralysis, and 4) hypnapompic or hypnagogic hallucinations. (15) Most patients will display only some of these symptoms, while a minority of patients, between 10% and 15%, will actually have the entire tetrad of symptoms. Excessive sleepiness is the most common symptom. Sleep attacks, sudden and unpredictable episodes of severe sleepiness or sleep, are less common but can result in serious accidents and injury.

Narcolepsy Diagnostic Criteria

excessive daytime sleepiness

cataplexy

sleep paralysis

hypnapompic or hypnagogic hallucinations

Epidemiology

Narcolepsy occurs in approximately 1 in 2000 persons and peaks in the second decade of life. Subtle symptoms may be present much earlier. Parents often refer the child with narcolepsy as

having been a “sleepy head” as a young child. There is no significant gender difference for narcolepsy but there is a significant ethnic difference with the disorder occurring much more frequently in Japan. Monozygotic twins are discordant for narcolepsy. Eighty-six percent of narcoleptics with definite cataplexy have HLA DQB1-0602 on chromosome 6, but greater than 99% of patients with these haplotypes do not have narcolepsy. Orexin or hypocretin may also be involved in narcolepsy. (22)

Treatment

Several treatment options are available but are not FDA approved for use in the child. Treatment of the excessive sleepiness is vital to improve daytime function and school performance. Modafinil (Provigil/Nuvigil) is a pro-alerting drug that can dramatically improve daytime sleepiness. If modafinil proves ineffective, traditional stimulants such as methylphenidate, and dextroamphetamine may also be of benefit. Xyrem is an option for the sleepiness especially when there is concomitant cataplexy. In an off label use, tricyclic antidepressants may also be quite effective for cataplexy.

Summary (<250 words, expands on click):

Circadian Rhythm Sleep Disorders

For optimal sleep and alertness, desired sleep time and wake times should be synchronized with the timing of the endogenous alertness promoting circadian rhythm. Misalignment between the circadian rhythm and the 24 hour physical environment can result in symptoms of insomnia and/or excessive daytime sleepiness. Circadian rhythm sleep disorders arise when the physical environment is altered relative to the internal circadian timing system, such as in jet lag and shift work, or when the timing of endogenous circadian rhythms are altered, such as in circadian rhythm sleep phase disorders. The latter, is thought to occur predominantly because of chronic alterations in the circadian clock or its entrainment mechanisms. This section focuses on this second group of disorders, which is the most common in childhood.

The essential feature of a Circadian Rhythm Sleep Disorder (CRSD) is that the sleep disturbance is due primarily to alterations of the circadian time-keeping system or a misalignment between the endogenous circadian rhythm and exogenous factors that affect the timing or duration of sleep. The circadian related sleep disruption leads to insomnia or excessive daytime sleepiness

that causes functional impairment or distress. Furthermore, maladaptive behaviors often influence the presentation and clinical course of circadian rhythm sleep disorders.

Topic discussion (up to 2 pages, expands on click**):**

Delayed Sleep Phase Syndrome

Clinical features

Delayed Sleep Phase Syndrome (DSPS) is characterized by bedtimes and wake times that are usually delayed 3-6 hours relative to desired or socially acceptable sleep/wake times. The patient typically cannot fall asleep before 2-6am and has difficulty waking up earlier than 10am-1pm.(23,24) In most cases, attempts to advance the patient's sleep times are unsuccessful. When allowed to follow their preferred schedule, circadian phase of sleep is delayed, but relatively stable and sleep quality is reported to be normal. Patients with DSPS often report feeling most alert in the evening and most sleepy in the early morning. They score as definite "evening" types on the Horne and Ostberg questionnaire of diurnal preference and often are described as "night" people, or "night owls".(25) Most patients seeking treatment do so because of enforced socially acceptable bed times and wake up times result in insomnia, excessive sleepiness and functional impairments, particularly during the morning hours.(23)

Clinical epidemiology

DSPS is probably the most common of the primary circadian rhythm sleep disorders in children.(26) Although the actual prevalence of DSPS in the general population is unknown, it has been reported that among adolescents and young adults, the prevalence is 7-16%.(23,27)

Differential Diagnosis

Delayed Sleep Phase Syndrome must be distinguished from "normal" sleep patterns, particularly in adolescents and young adults who exhibit delayed schedules without impaired functioning. Social and behavioral factors play an important role in the development and maintenance of the delayed sleep patterns. Attempts to fall asleep earlier result in prolonged sleep latency and may promote as well as perpetuate features of conditioned insomnia. Exposure to bright light in the evening may promote the inability to sleep and exacerbate the delayed circadian phase. Furthermore, the role of school avoidance, social maladjustment and family dysfunction must be considered as precipitating and contributing factors, especially in adolescents. Individuals may

use alcohol and excessive caffeine to cope with symptoms of insomnia and excessive sleepiness, which in turn, may exacerbate the underlying circadian rhythm sleep disorder.

A family history may be present in approximately 40% of individuals with DSPS, and the DSPS phenotype has been an autosomal dominant trait.(28)

Diagnostic evaluation

The diagnosis of DSPS depends primarily on the clinical history. However, diagnostic studies such as actigraphy and sleep diaries can be very useful to confirm the delayed sleep phase pattern. Recordings of sleep diaries and actigraphy over a period of at least 2 weeks demonstrate delayed sleep onset and sleep offset, with sleep onsets typically delayed until 2-6 AM and wake up times in the late morning or early afternoon. Daily work or school schedules may result in earlier than desired wake time during weekdays, but a delay in bedtime and wake up time is almost always seen during weekends and while on vacation. Polysomnographic (PSG) parameters of sleep architecture, when performed at the natural delayed sleep times, are essentially normal for age. However, if a conventional bedtime and wake up time is scheduled, PSG recording will show prolonged sleep latency and decreased total sleep time.

Clinical management

Approaches aimed at resetting circadian rhythms, such as chronotherapy, timed bright light and melatonin have been employed for the treatment of DSPS. Chronotherapy is a treatment in which sleep times are progressively delayed by approximately 3 hours per day until the desired earlier bedtime schedule is achieved.(29) Although effective, the length and repeated nature of treatment and need for adherence to restrictive social and professional schedules limit practicality in the clinical setting. However, in adolescents, in which behavioral factors often contribute to the delayed sleep phase, chronotherapy in conjunction with enforcement of regular sleep and wake times are important components of the clinical management.

Exposure to bright light for 1-2 hours in the morning results in an advance of the phase of circadian rhythms, whereas evening light exposure causes phase delays. Therefore, bright light exposure during the early morning hours and avoidance of bright light in the evening have been shown to be effective treatments for DSPS.(30) Following 2 weeks of exposure to 2 hours of

bright light of 2500 lux each morning and restricted evening light, individuals with DSPS showed earlier sleep times and reported improved morning alertness level. However, many patients, particularly those who are severely delayed, find it difficult to awaken earlier for the 1-2 hour of bright light therapy. Despite potential utility of bright light therapy, the timing, intensity and duration of treatment remain to be defined. Exposure to broad spectrum light of 2,000 to 10,000 lux for approximately 1-2 hours is generally recommended for use in clinical practice.

Due to the practical limitations of chronotherapy and phototherapy, melatonin, taken orally in the evening, has been increasingly investigated as a treatment for DSPS. Several studies have demonstrated the potential benefits of melatonin administered in the evening.(31) However, because the timing of administration and dose varied between studies, and the relative lack of large scale controlled clinical trials, clinical guideline for the use of melatonin in the treatment of DSPS is not available. The treatment of DSPS with melatonin is an unapproved use and remains empirical. Treatment success depends on many variables including severity of the delayed sleep phase, co-morbid psychopathology, ability and willingness of the patient to comply with the treatment, school schedule, work obligations, and social pressures.

Summary (<250 words, expands on click):

Topic discussion (up to 2 pages, expands on click**):**

Pediatric Obstructive Sleep Apnea

Epidemiology

Pediatric OSA occurs with a prevalence of 2-4% for children between the ages of 2 and 18.(35, 36) Obstructive apnea is relatively uncommon in normal children. In the past obstructive sleep apnea was primarily seen in patients with significant adenotonsillar hypertrophy or neurological dysfunction.(37) More recently, obstructive sleep apnea has been associated with obesity in children.

The symptoms of obstructive sleep apnea are different in children compared to adults. Although daytime sleepiness and fatigue are reported in children, behavioral problems, hyperactivity and neurocognitive deficits are much more common in children with sleep apnea compared to normal controls or adults with obstructive sleep apnea.(38)

Diagnosis

Pediatric OSA can be confirmed with overnight polysomnography. The severity of OSA has been defined by use of AHI criteria alone. This approach is, however, flawed. This problem becomes more pronounced in the pediatric population. The criteria are different than adults, with an apnea index of > 1/hr considered abnormal.

In children a cessation of airflow for two or more respiratory cycles is considered an apnea when the event is obstructive (35-39). Of note, the respiratory rate in children (20-30/min) is greater than adults (12-15/min). The obstructive apnea hypopnea index (AHI) > 1 is considered abnormal in children as opposed to 5 in adults. There is usually only a mild decrease in the arterial oxygen saturation.

The importance of central apnea in older children is less certain than in infants. Most pediatric sleep specialists do not consider central apneas following sighs (big breaths) to be abnormal. Some central apnea is most likely normal in children especially during REM sleep. In one study, up to 30% of normal children had at least occasional central apneas. Central apneas longer than 20 seconds or those of any length associated with arterial oxygen desaturation below 90% are often considered abnormal although a few such events have been noted in normal children (38). Therefore, in most cases, observation is recommended unless the events are frequent or the arterial oxygen desaturations severe.

There is a shortage of sleep laboratories that can accommodate children appropriately. Other potential screening techniques have not proved successful thus far. Therefore, in a child with behavioral problems, hyperactivity or daytime sleepiness, a polysomnogram should be considered especially if obesity, adenotonsillar hypertrophy or other upper airway anatomic anomalies are present.

Treatment

The treatment of choice for the majority of pediatric OSA cases is adenotonsillectomy. There are some specific groups who are at increased risk for postoperative morbidity: children < 3 years of age; severe OSA; and those with underlying medical disorders. Weight loss and nasal CPAP are also used in pediatric OSA for those cases who do not improve after adenotonsillectomy or are

not surgical candidates. CPAP is used as the primary treatment modality much less frequently in children than in adults. Craniofacial surgeries are also an option in selected children with anatomic abnormalities.

[1-39]

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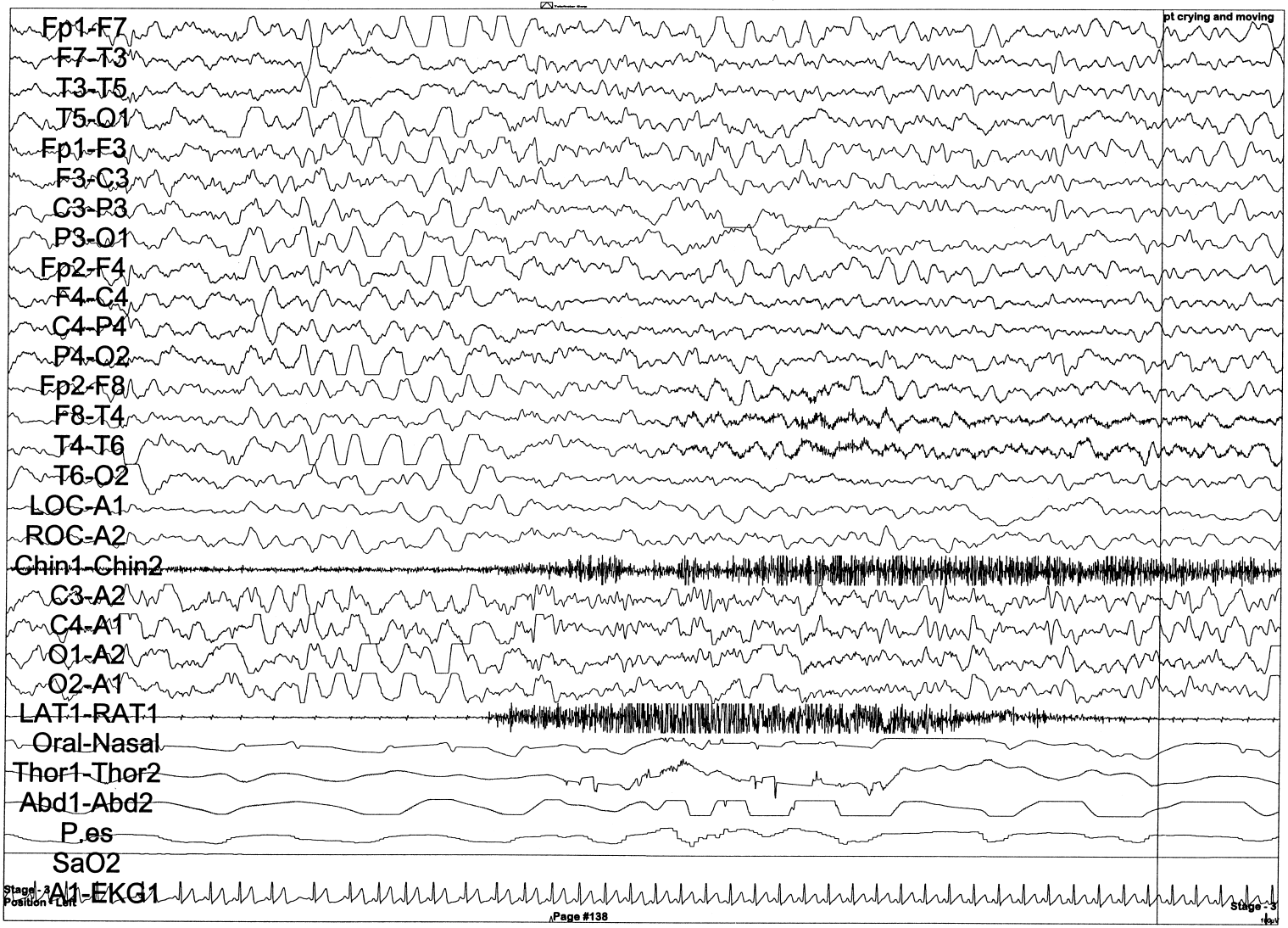
pt crying and moving
Fp1-F7
F7-T3
T3-T5
T5-O1
Fp1-F3
F3-C3
C3-P3
P3-O1
Fp2-F4
F4-C4
C4-P4
P4-O2
Fp2-F8
F8-T4
T4-T6
T6-O2
LOC-A1
ROC-A2
Chin1-Chin2
C3-A2
C4-A1
O1-A2
O2-A1
LAT1-RAT1
Oral-Nasal
Thor1-Thor2
Abd1-Abd2
P.es
SaO2
A1-EKG1
Stage - 3
Position - Left
Page #138
Stage - 3

Figure 1

Polysomnogram: Expanded EEG montage with esophageal manometry pressure monitoring; 30-second page. Eight year old boy with confusional arousals. Staging: Stage 3 to stage 4 sleep. Following the arousal, the EEG shows continued delta activity intermixed with faster frequencies, associated with moving and crying. The observed behavior was typical of a confusional arousal. In the 5 to 6 seconds preceding the arousal, the EEG shows delta activity that is more rhythmic and synchronous than the delta activity with usually occurs in slow-wave sleep. Rhythmic, synchronous delta activity sometimes precedes or accompanies arousals from slow-wave sleep in patients with arousal disorders. (Used by permission, "Atlas of Polysomnography", 2nd Edition Eds. J. Geyer MD, T. Payne MD, P. Carney MD Lippincott Williams & Wilkins, 2010).