INTRODUCTION

Sleep disorders are conditions that disrupt the normal quality and pattern of sleep for patients and are very common in the general population. Using a good history, physical exam and selected diagnostic testing, sleep disorders can also be well managed by the family physician. Sleep disorders account for a significant number of outpatient visits, with any sleep disturbance accounting for over 12.1 million visits in 2010 according to NHANES 1999-2010. Similarly visits related to sleep apnea and sleep related breathing disorders rose 400% in the same survey, accounting for 5.8 million visits. The direct and indirect costs associated with sleep disorders are substantial, with the direct costs of the treatment increasing sleep can often lead to accurate diagnosis without excessive diagnostic testing. The American Academy of Sleep Medicine joint consensus statement on sleep duration, recommends that the average adult should get between 7-9 hours of sleep per night. Ideally this would be continuous uninterrupted sleep, although the historical record would indicate that uninterrupted sleep at night is a relatively new phenomenon and that sleep at night need not be continuous to be considered adequate. Sleep duration less than 6 hours is associated with several deleterious health effects, and interestingly, sleep duration of more than 9 hours has similar, but less clear association with poor health outcomes. The clinician should ask about duration, quality and pattern of sleep. History from the patient can be augmented with information from the bed partner, as this can also provide important clues regarding sleep. Care should be taken to differentiate primary sleep disorders and sleep complaints secondary to another disorder. For instance, diagnostic criteria for several psychiatric illnesses including Attention Deficit Hyperactivity Disorder (ADHD), anxiety, depression, and bipolar disorder include sleep disturbance as part of the criteria but these would obviously be incorrectly categorized as a sleep disorder and should be treated with appropriate modalities. Sleep disturbance is also comorbid with several chronic health conditions such as Chronic Obstructive Pulmonary Disease (COPD), Alzheimer’s dementia, asthma, fibromyalgia, and other chronic.

Historically, society has developed a culture that historically would indicate that uninterrupted sleep at night is a relatively new phenomenon and that sleep at night need not be continuous to be considered adequate. Ideally this would be continuous uninterrupted sleep, although the historical record would indicate that uninterrupted sleep at night is a relatively new phenomenon and that sleep at night need not be continuous to be considered adequate. Sleep duration less than 6 hours is associated with several deleterious health effects, and interestingly, sleep duration of more than 9 hours has similar, but less clear association with poor health outcomes. The clinician should ask about duration, quality and pattern of sleep. History from the patient can be augmented with information from the bed partner, as this can also provide important clues regarding sleep. Care should be taken to differentiate primary sleep disorders and sleep complaints secondary to another disorder. For instance, diagnostic criteria for several psychiatric illnesses including Attention Deficit Hyperactivity Disorder (ADHD), anxiety, depression, and bipolar disorder include sleep disturbance as part of the criteria but these would obviously be incorrectly categorized as a sleep disorder and should be treated with appropriate modalities. Sleep disturbance is also comorbid with several chronic health conditions such as Chronic Obstructive Pulmonary Disease (COPD), Alzheimer’s dementia, asthma, fibromyalgia, and other chronic.areas, insomnia, circadian rhythm disorders, sleep related breathing disorders, sleep movement disorders, sleep behavior disorders and daytime sleepiness disorders.

HISTORY & PHYSICAL EXAMINATION

As with any disorder, evaluation starts with a good history and physical examination. The importance of a reliable history regarding sleep can often lead to accurate diagnosis without excessive diagnostic testing. The American Academy of Sleep Medicine joint consensus statement on sleep duration, recommends that the average adult should get between 7-9 hours of sleep per night. Ideally this would be continuous uninterrupted sleep, although the historical record would indicate that uninterrupted sleep at night is a relatively new phenomenon and that sleep at night need not be continuous to be considered adequate. Sleep duration less than 6 hours is associated with several deleterious health effects, and interestingly, sleep duration of more than 9 hours has similar, but less clear association with poor health outcomes. The clinician should ask about duration, quality and pattern of sleep. History from the patient can be augmented with information from the bed partner, as this can also provide important clues regarding sleep. Care should be taken to differentiate primary sleep disorders and sleep complaints secondary to another disorder. For instance, diagnostic criteria for several psychiatric illnesses including Attention Deficit Hyperactivity Disorder (ADHD), anxiety, depression, and bipolar disorder include sleep disturbance as part of the criteria but these would obviously be incorrectly categorized as a sleep disorder and should be treated with appropriate modalities. Sleep disturbance is also comorbid with several chronic health conditions such as Chronic Obstructive Pulmonary Disease (COPD), Alzheimer’s dementia, asthma, fibromyalgia, and other chronic.
pain syndromes. Medications, including supplements, should be reviewed in any patient complaining of sleep disorder. Alcohol, food, and caffeine consumption, including chocolate, should also be reviewed. Some key historical features suggestive of a specific diagnosis include cataplexy with narcolepsy, limb movements after falling asleep in sleep movement disorders and limb movement preventing sleep initiation in restless leg syndrome. One of the more useful tools in the history in evaluating sleep disorders is a patient completed sleep diary. This diary should also include activities and behaviors just before bedtime as well, such as exercise and smartphone, tablet, laptop or television "screen time." While sleep diaries are useful, patients typically over-report sleep duration when compared to objective measures.20 Multiple night wrist actigraphy can provide objective measures of sleep duration and has been well validated to correlate with actual sleep duration.21,22

Diagnosis of sleep disorders can often be made by history alone, but in some cases diagnostic testing is indicated. Testing strategies include in home polysomnography or full formal polysomnography in a sleep lab. Most patients do not require advanced testing, but it should be considered if sleep apnea or a sleep movement disorder is suspected, or if the interventions for a sleep disorder fail. In home sleep studies have been shown to accurately diagnose obstructive sleep apnea but cannot differentiate between central and obstructive sleep apnea.23-25 The multiple sleep latency test is needed for the diagnosis of narcolepsy, and is useful for monitoring response to treatment. Physical exam is typically benign in most sleep disorders, but care should be taken to assess for craniofacial abnormalities, tonsilar hypertrophy and neck circumference. Clinicians can consider an easy screen for sleep disorders by adding the question "Have you had any difficulty with sleep in the past week" to the review of systems in a regular office visit.26

INSOMNIA

Insomnia is the most common sleep disorder. Between 6-10% of the population meets diagnostic criteria for insomnia and up to one third of the population report at least some symptom of insomnia at any given time.26 There are two main diagnostic rubrics for the diagnosis of insomnia that can be used, either from the Diagnostic and Statistical Manual edition 5 (DSM-5) published by the American Psychiatric Association or from the International Classification of Sleep Disorders version 2 (ICSD-2) published by the American Academy of Sleep Medicine (AASM).27,28 Both are accurate in the diagnosis of insomnia, with the ICSD-2 further subdividing insomnia into 12 further specific insomnia disorders. The diagnostic criteria for both are presented in Table 1. Clinicians should feel free to use either scheme as they both are able to support a primary diagnosis of insomnia remembering that the diagnosis rests mainly in the clinical interview. The duration of insomnia is important as the symptoms often wax and wane, and the most common form of insomnia is a secondary insomnia triggered by acute psychosocial distress. Sleep diaries, including peri-bedtime behaviors provide valuable information but should be used in conjunction with clinical interview as the patient with insomnia often over-estimates the time needed to fall asleep and underestimates the total time spent sleeping.29 History should also include the timing of insomnia, difficulty with initiation of sleep, waking in the middle of the night or waking too early.30 Interesting clues can be discovered if the patient is asked about their perception of the cause of insomnia, the subjective amount of sleep a patient feels is necessary as well as if the patient is taking daytime naps.

Numerous treatment modalities exist and while numerous pharmacologic agents exist, medications should be considered among the final options for management. Polling suggests that 4 out of 10 patients with chronic insomnia self-medicate with either over the counter sleep aids, usually anti-histamines, or alcohol.31 Several effective non-pharmacologic approaches are available all easily

### Table 1:

<table>
<thead>
<tr>
<th>DSM 5 diagnostic criteria for insomnia</th>
<th>ICSD diagnostic criteria for insomnia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A.</strong> Predominant complaint of dissatisfaction in sleep quantity of quality, with one or more of the following</td>
<td><strong>A.</strong> A complaint of difficulty initiating sleep, maintaining sleep, waking up too early of chronically non-restorative or poor quality sleep</td>
</tr>
<tr>
<td>1. Difficulty initiating sleep</td>
<td><strong>B.</strong> The sleep difficulty occurs despite the adequate opportunity an circumstances for sleep</td>
</tr>
<tr>
<td>2. Difficulty maintain sleep</td>
<td><strong>C.</strong> At least one of the following daytime impairment</td>
</tr>
<tr>
<td>3. Early morning awakening</td>
<td>1. Fatigue of malaise</td>
</tr>
<tr>
<td><strong>B.</strong> The sleep disturbance causes impairment in important areas of function (social, work, school, behavior)</td>
<td>2. Attention, concentration or memory impairment</td>
</tr>
<tr>
<td><strong>C.</strong> Occurs at least 3 night a week</td>
<td>3. Poor social, vocational or school performance</td>
</tr>
<tr>
<td><strong>D.</strong> The difficulty is present for at least 3 months</td>
<td>4. Mood disturbance or irritability</td>
</tr>
<tr>
<td><strong>E.</strong> Occurs despite the adequate opportunity for sleep</td>
<td>5. Daytime sleepiness</td>
</tr>
<tr>
<td><strong>F.</strong> The insomnia is not better explained by another sleep disorder</td>
<td>6. Motivation, energy or initiative reduction</td>
</tr>
<tr>
<td><strong>G.</strong> The insomnia is not due to the effects of a substance</td>
<td>7. Proneness to errors at work or driving</td>
</tr>
<tr>
<td><strong>H.</strong> Co-morbid medical or psychiatric disorders do not adequately explain the insomnia</td>
<td>8. Tension, headaches or GI symptoms in response to sleep loss</td>
</tr>
<tr>
<td></td>
<td><strong>9.</strong> Concerns or worries about sleep</td>
</tr>
</tbody>
</table>

Adapted from DSM 5 and ICSD-2.27,28
discussed by the Family Physician in the office setting. The first step is to address sleep hygiene, the actual environment in which the patient sleeps and pre bedtime behaviors. Nicotine, large meals, caffeine, vigorous exercise, and alcohol should be avoided for several hours before bedtime. The bedroom should be cool, dark, well ventilated and the bed should be comfortable. Reading, watching television and computers, basic stimulus control, should be avoided while in the bedroom. While sleep hygiene is important, evidence suggest that sleep hygiene recommendations alone are not effective in the treatment of insomnia. Sleep restriction therapy has been shown to increase total time spent sleeping and decreasing sleep latency. In general sleep restriction therapy involves going to bed about 15 minutes before adequate time for sleep and gradually increasing this time to a full night. Naps are avoided and a routine wake time is established as well. One study noted that simply reducing the total time spent in bed increased the amount of sleep, a very simple intervention. Cognitive Behavioral Therapy (CBT) is another treatment known to be effective and should be considered first line therapy after sleep hygiene, but is use is limited in practice by the need for trained therapists to administer this modality. CBT involves a combination of sleep restriction, stimulus control, and cognitive measures to challenge the patient perceptions of insomnia. Cognitive therapy can involve writing worries about sleep in a journal, writing distressing thoughts to help clear the patients mind prior to bed and discussion of thought patterns that hinder sleep. CBT has been shown to be effective for long term treatment in as little as two sessions. Self-help programs and online resources are also available at relatively low cost and given the efficacy of CBT should be dispensed as often as medications.

Several pharmacologic agents are available for insomnia each with relative advantages and disadvantages (Table 2). As the ideal pharmacologic agent with a short half-life, no risk of dependence, and no next day sedation that works for both sleep initiation and maintenance does not exist, clinicians should weigh benefits of the choice of pharmacologic therapy for each patient. In general, medications can be divided into three categories, benzodiazepines, non-benzodiazepines, and other agents. Benzodiazepine sleep agent use is limited by tolerance and dependence, and their use has been supplanted by the non-benzodiazepines, the so-called “z-drugs” such as zolpidem, eszopiclone, and zaleplon. Other agents include medications that are FDA approved for insomnia and work outside of the benzodiazepine receptor model. Some physicians may choose to prescribe trazodone or mirtazapine for sleep, but there is little evidence to suggest these work for sleep outside of insomnia associated with depression. While sedating, anti-psychotic agents should be avoided as sleep aids given the significant potential for adverse effects. There is significant debate on the long term nightly use of sleep aid medications. Agents should be used as sparingly as possible and for the shortest time needed. Zolpidem has been used nightly for up to one year without dose escalation or rebound insomnia, but about one third of patients gradually discontinued use of benzodiazepine sleep agents reverted back to nightly use by two years.

CIRCADIAN RHYTHM DISORDERS

Circadian rhythm sleep disorders are caused by a misalignment of the natural internal clock of the human body and the 24-hour external environment. The human body has a natural sleep-wake cycle determined by a complex interaction of the central circadian pacemaker located in the suprachiasmatic nucleus, endogenous melatonin production, and core body temperature as well as external cues such as light/dark cycles. Studies have demonstrated that the internal circadian clock in the absence of external cues is about 24.2 hours. Several circadian sleep disorders are recognized, including advanced or delayed sleep phase disorders, shift worker disorder and jet lag syndrome. Diagnosis for all is usually made by history and a sleep diary, but actigraphy can provide objective information on sleep wake cycles.

As the names imply, advanced or delayed sleep phase syndromes are the timing of sleep onset outside of socially accepted norms. Patients with advanced sleep phase difficulties will generally report an involuntary and significant urge to fall asleep from 6-9 PM, while delayed sleep phase syndrome patients will report an inability to fall asleep until 2-6 AM. Care should be taken to differentiate behavioral references for different bedtimes and sleep phase disorders. Patients with sleep phase disorders will have increasing difficulty adhering to societal conventions as time passes. Objective information with 7 nights of actigraphy or of a shift in core body temperature nadir, which is naturally lowest in the morning after a full night sleep, can aid in diagnosis. The therapy of choice for sleep phase disorders is chronotherapy, bright light therapy and melatonin. Chronotherapy is the delay of sleep by 3 hours every 2 days in delayed sleep phase, and the advancement of sleep by 3 hours every 2 days until the desired bedtime if reached. This technique requires significant time and strict adherence. Timed bright light for 2 hours, either in the morning from 7-9 for delayed sleep phase, or in the evening from 7-9 in advanced sleep phase. Melatonin, up to 3mg, given 5 hours before the desired bedtime in delayed sleep phase syndrome also appears effective, although most recommendations are based on expert opinion given a paucity of controlled trials.

Shift work disorder is the result of having to sleep during non-standard hours. Estimates are that 20% of the workforce in industrialized countries work nonstandard hours and of those patients up to 10% have shift work disorder. Patients usually complain of non-refreshing sleep and excessive sleepiness that varied with work schedule. Treatment options include bright light exposure during the night, morning melatonin before sleep and adherence to sleep hygiene measures. Bright light and melatonin are used to help reset the circadian clock. Workers with rapidly varying schedules should likely not try to change circadian clock with bright light or melatonin. Stimulants such as caffeine, 200-400 mg at the start of a shift, or prescription modafinil 200 mg at the start of the shift. Modafinil is FDA approved for shift work disorder, but caution is advised as the stimulants improve sleepiness, but do not appear to improve alertness.

Jet lag is the rapid desynchronization of an established circadian rhythm to a new rhythm, made possible by modern air travel. Symptoms are directly related to the number of time zones traversed and the main symptoms are insomnia and daytime sleepiness. Treatment usually lasts only for 3-4 days, with melatonin administered between 10-12 pm at the destination preceded by 3 nights of melatonin around 6 PM prior to leaving. Eastward travelers can be advised to avoid bright light in the morning and seek bright light in the evening and westward travelers can be advised to seek the opposite.
TABLE 2: Medications for insomnia

<table>
<thead>
<tr>
<th>Name</th>
<th>Cost/Generic Available</th>
<th>Half Life (Hours)</th>
<th>Controlled Substance / FDA Approved</th>
<th>Other Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonbenzodiazepine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zaleplon (Sonata)</td>
<td>$15 - $30 / Yes</td>
<td>1</td>
<td>Yes / Yes</td>
<td>Ultra-short duration of action, rapid onset</td>
</tr>
<tr>
<td>Zolpidem (Ambien)</td>
<td>$6 - $12 / Yes</td>
<td>2 - 3</td>
<td>Yes / Yes</td>
<td>Controlled release formulas available, risk of abnormal sleep behaviors, max dose different for men and women</td>
</tr>
<tr>
<td>Eszopiclone (Lunesta)</td>
<td>$20 - $70 / Yes</td>
<td>6</td>
<td>Yes / Yes</td>
<td>1mg starting dose as 3mg can cause excess sedation for over 11 hours, unpleasant aftertaste</td>
</tr>
<tr>
<td>Benzodiazepine</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Triazolam (Halcion)</td>
<td>$9 - $30 / Yes</td>
<td>1.5 - 5.5</td>
<td>Yes / Yes</td>
<td>Rapid onset, risk of complex sleep related behaviors, aggression, caution when used with opiate analgesics</td>
</tr>
<tr>
<td>Temazepam (Restoril)</td>
<td>$8 - $12 / Yes</td>
<td>8.8</td>
<td>Yes / Yes</td>
<td>Intermediate onset, caution if used with opiate analgesics</td>
</tr>
<tr>
<td>Melatonin Receptor Agonist</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ramelteon (Rozerem)</td>
<td>$300 - $350 / No</td>
<td>2 - 5</td>
<td>No / Yes</td>
<td>Can worsen depression, suicidal ideation</td>
</tr>
<tr>
<td>Orexin Receptor Agonist</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Suvorexant (Belsomra)</td>
<td>$290 - $300 / No</td>
<td>12</td>
<td>Yes / Yes</td>
<td>Long half-life can lead to next day sedation, give with caution to patients with respiratory problems, rarely associated with cataplexy</td>
</tr>
<tr>
<td>Antidepressants</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doxepin (Silenor)</td>
<td>$330 - $340 / No</td>
<td>15</td>
<td>No / Yes</td>
<td>Generic 10mg Doxepin is generic and much less costly, Anticholinergic side effects, next day somnolence</td>
</tr>
<tr>
<td>Mirtazapine (Remeron)</td>
<td>$4 - $12 / Yes</td>
<td>20 - 40</td>
<td>No / No</td>
<td>Edema, increased hunger, weight gain, suicidality in patients under 24 with depression</td>
</tr>
<tr>
<td>Trazodone</td>
<td>$4 - $12 / Yes</td>
<td>3 - 6</td>
<td>No / No</td>
<td>Anticholinergic side effects, sexual dysfunction, next day somnolence suicidality in patients under 24 with depression</td>
</tr>
<tr>
<td>Antihistamines</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doxylamine</td>
<td>$4 - $12 / Yes</td>
<td>10</td>
<td>No / Yes</td>
<td>Available over the counter, CNS depression, tolerance can develop quickly</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>$4 - $12 / Yes</td>
<td>3 - 9</td>
<td>No / Yes</td>
<td>Available over the counter, CNS depression, tolerance can develop quickly</td>
</tr>
</tbody>
</table>

Prices from GoodRx.com, the best available price for 30 day supply, with coupon if freely available, reflecting prices in the authors’ hometown and the nearest 2 metropolitan areas.
SLEEP RELATED BREATHING DISORDERS

Obstructive sleep apnea (OSA) has long been recognized, likely first characterized as Pickwickian syndrome in the 19th century, but with the advent of effective treatment obstructive sleep apnea has become an important target for detection and management. OSA is caused by lack of airflow through the upper airway, with several risk factors, including craniofacial abnormalities, narrow upper airway, tonsillar hypertrophy or laxity in the musculature of the upper airway leading to collapse during breathing. The classic symptoms of OSA include snoring, daytime hypersomnia and larger body habitus, but other symptoms such as morning headaches, nocturia and erectile dysfunction can also be symptomatic of OSA.\(^\text{43}\) Nocturnal gasping or choking are the most reliable indicators of OSA.\(^\text{44}\) Untreated OSA has important clinical consequences such as difficult to control hypertension, cardiac arrhythmias and congestive heart failure.\(^\text{45}\)

Several screening tools have been developed for us in primary care to help identify patients at risk for obstructive sleep apnea, with the STOP-Bang and Berlin questionnaire as the most sensitive tool for finding patients with moderate to severe OSA.\(^\text{46}\)\(^\text{47}\) There is not a recommendation for the primary care physician to screen for sleep apnea in the general population from primary care professional societies. Diagnosis of sleep apnea requires a sleep study, either in home or in a sleep lab. Traditionally, full in lab polysomnography was thought to be required for diagnosis but in home testing has been shown to be as effective in identifying patients with OSA, regardless of pretest probability.\(^\text{48}\)\(^\text{49}\) Sleep testing looks for episodes of stopping breathing of shallow breathing, with or without hypoxia. These episodes are translated into the apnea-hypopnea index, the AHI, essentially the number of times per hour a patient has a significant respiratory disturbance. OSA does not have a universally accepted definition, but the AASM defines mild OSA as an AHI of 5-15, moderate as 15-30 and severe as 30 or more, and daytime sleepiness must be present.\(^\text{50}\)

Treatment of OSA usually involves continuous positive airway pressure (CPAP) devices. CPAP provides constant upper airway support, alleviating the collapse the upper airway. Several other airway support devices such as bi-level positive airway pressure (BIPAP), adaptive servovventilation (SV) and volume assured pressure support (VAPS) are also available, but are limited to very specific clinical situations. Adherence to CPAP is notoriously poor but educational and behavioral interventions have been shown to increase adherence.\(^\text{51}\) Oral appliances can be used for sleep apnea, but are more appropriate for mild OSA or for patients intolerant of CPAP. One study demonstrated that oral appliances were as effective as CPAP, but the study conclusions are limited by short duration, one month, and low adherence rate to CPAP.\(^\text{52}\) Oral appliances do not appear to improve daytime sleepiness symptoms but do decrease snoring, the clinical significance of which is not clear.\(^\text{53}\) Surgical resection of the upper airway to improve patency has limited outcomes at this time and should only be considered as a last resort for treatment of OSA.

SLEEP BEHAVIOR DISORDER

Commonly known as parasomnias, sleep behavior disorders involve complex movement and behaviors during sleep. Patients may seem to move or behave with purpose in a goal directed fashion, but by definition of the disorder, the patient is asleep. Diagnosis is usually clinical, based on history alone but overnight video polysomnography can be obtained is the diagnosis is not clear. Collateral informants are key to the history and a validated questionnaire, The Mayo Sleep Behavior Questionnaire, is also available to aid in diagnosis.\(^\text{54}\) Sleep behavior disorder can be very distressing for both the patient and bed partner and have some of the most unusual symptoms of all of the sleep disorders. In describing sleep behavior disorders, it is useful to categorize the disorder as occurring during REM sleep or not during REM sleep. REM sleep is the phase of sleep when dreams occur and is accompanied by muscle paralysis.

Common non-REM sleep disorders include, sleep walking, sleep talking, and sleep terrors. While these are very different events, they do share some common clinical features. Patients do not remember the events, have minimal cognitive function and often appear awake, patients may even have their eyes open during the events.\(^\text{55}\) Sleep terrors should not be confused with a nightmare, as the patient is not having a dream, as they are not in REM sleep. Patients in a sleep terror can sound extremely distressed, but bed partners should be assured that the episode is not harmful. A specific sub-type of sleepwalking includes sleep related eating disorder, when patients will have amnestic episodes of eating during sleep.\(^\text{56}\) The underlying etiology of these events are not clear but they do seem to be related to acute psychosocial stress. Treatment is rarely needed, usually education about the transient and benign nature of the events. Medications that are associated with the events include serotonin modifying antidepressants and short acting hypnotics and should be stopped if clinically warranted. Paradoxically, sleep related eating disorder first line treatment includes selective serotonin reuptake inhibitors (SSRIs), while topiramate is a reasonable second option.\(^\text{57}\) If the events do become more common, a safe sleep environment should be ensured.

REM associated sleep behavior disorders include nightmare disorder and REM sleep behavior disorder. Nightmare disorder sounds as if it should be similar to sleep terror, but there are several important distinguishing features. Nightmare disorder usually involves intense and vivid dreams that the patient will remember. Patients will move very little during a nightmare and will behave relatively normally upon wakening, whereas sleep terrors can involve intense movements and patients are typically very confused upon wakening. Treatment is usually supportive if needed. If a patient is particularly active, violent or are enacting very complex activities, REM sleep behavior disorder (RBD) should be considered as an alternate diagnosis. RBD usually corresponds to the dream state of patients, typically a dream that involves the patient being attacked, or the patient being placed in an unpleasant situation, although this presumption is still under debate.\(^\text{58}\)\(^\text{59}\) RBD occurs because of pathological loss of the normal muscle paralysis with REM sleep. Assault of bed partners or expletive laden vocalizations can occur. RBD tends to respond very well to either melatonin or clonazepam.\(^\text{58}\) Patients and bed partners should seek to maximize safety, such as removing sharp objects, firearms etc. RBD is associated with neurodegenerative diseases such as Parkinson’s disease and Lewy body dementia.\(^\text{59}\) RBD may precede the onset of these conditions by decades but if the disorder presents in younger patients, medication side effects are more often the etiology.
DAYTIME SLEEPINESS DISORDERS

Narcolepsy and idiopathic hypersomnia are the most common disorders falling under this term. Both involve excessive daytime sleepiness with the cardinal distinguishing feature of narcolepsy being cataplexy, sudden loss of muscle tone triggered by emotions.\(^6\)\(^,\)\(^7\) Loss of muscle tone can be very subtle, such as a head bob or loss of jaw tone, or can be very profound such as general loss of tone resulting in collapse, the “sleep attack.” Both require daytime sleepiness, but patients with idiopathic hypersomnia typically do not find daytime naps to be refreshing. The Epworth Sleepiness Scale, presented in Table 3, can help identify patients with significant daytime sleepiness and correlates well with sleep latency, the time needed to fall asleep, as measured on a multiple sleep latency test. The diagnosis of narcolepsy typically includes an overnight sleep study followed by a multiple sleep latency test, to prove the markedly reduced time needed to fall asleep. Narcolepsy diagnosis can also be made clinically based on daytime sleepiness with cataplexy. A decrease in CSF hypocretin level in the presence of daytime sleepiness and cataplexy is diagnostic of narcolepsy and can be considered in lieu of a multiple sleep latency test.\(^6\)\(^2\)

Treatment of narcolepsy and idiopathic hyper somnolence is very similar, typically consisting of stimulants. Modafinil, 200-400 mg daily, reduces daytime somnolence and has the FDA indication for narcolepsy.\(^6\)\(^3\) Other stimulants such as methylphenidate and amphetamines are tempting to use but sympathomimetic side effects tend to limit use, and should be considered second line treatment. Armodafinil is a long acting isomer of modafinil and has similar effects, but does not carry the FDA indication for narcolepsy and evidence is lacking regarding superiority. Cataplexy associated with narcolepsy traditionally was treated with either fluoxetine or clomipramine, but there is no evidence of efficacy of this treatment.\(^6\)\(^4\) Cataplexy can be treated with sodium oxybate with a goal dose of 6-9 grams per night.\(^6\)\(^5\) Titration to goal dose can take several weeks and optimal response usually takes 8-12 weeks.

**TABLE 3:**

<table>
<thead>
<tr>
<th>Epworth Sleepiness Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>How likely are you to fall asleep or doze off in the following situations?</td>
</tr>
<tr>
<td>0 = No chance of dozing</td>
</tr>
<tr>
<td>- Sitting and reading</td>
</tr>
<tr>
<td>- Watching TV</td>
</tr>
<tr>
<td>- Sitting inactive in a public place (theater, meeting)</td>
</tr>
<tr>
<td>- As a passenger in a car for an hour without a break</td>
</tr>
</tbody>
</table>

**TOTAL SCORE INTERPRETATION**

0 - 7: Unlikely abnormally sleepy
8 - 9: Average amount of daytime sleepiness
10 - 15: May be excessively sleepy depending on the situation, consider medical advice
16 - 24: Excessively sleepy, consider medical attention


SLEEP MOVEMENT DISORDER

Common sleep movement disorders include restless leg syndrome (RLS), Periodic Limb Movement Disorder (PLMD) and sleep bruxism. While RLS and PLMD sound very similar it should be noted that they are in fact distinct diagnoses. Likely more common than previously appreciated, these disorders are an important cause of poor sleep for patients and bed partners.

RLS, recently also referred to as Willis Ekbom disease, has certainly grown in public awareness as a neurological disorder. Though unpleasant and uncomfortable, it is often not “painful.” Typically, symptoms are distal to the knee and deep, not in the skin, but can also involve arms. Common descriptive terms patients may offer in the history include feelings of crawling, creeping, pulling, itching, burning, twitching, aching and restlessness.\(^6\)\(^6\)\(^,\)\(^6\)\(^7\) The diagnosis of RLS/WED can usually be made on clinical grounds, while the polysomnograph may be useful with the periodic limb movement assessment. Diagnostic criteria are the urge to move the legs, usually accompanied by the unpleasant sensations and the urge or sensations begin or worsen during periods of inactivity, such as lying, sitting, or going to bed. The patient experiences partial or total relief by movement or activity, such as walking or stretching for the duration of the activity and the symptoms are worse in the evening or night than during the day or that they occur only at night. RLS/WED is idiopathic or primary in most patients; but comorbid associations, especially with iron deficiencies, RLS/WED may be the initial presentation of an iron deficiency. Checking a serum ferritin level is often useful, with a target ferritin level of greater than 50 ng/mL.\(^6\)\(^8\) The exact pathophysiology of RLS is not understood, but given the responses to iron supplementation and dopaminergic medications the role of central nervous system stores of iron and dopamine seems central. Targeting dopaminergic pathways, commonly used agents to date have included short acting dopamine antagonists dosed typically much lower than the indication of Parkinson Disease. Targeting dopaminergic pathways, commonly used agents to date have included short acting dopamine agonists dosed typically much lower than the indication of Parkinson Disease (PD).\(^6\)\(^9\) The downside of the dopaminergic agents include normal...
dopamine agonist side effects and the phenomenon of augmentation, in which symptoms tend to migrate to become more severe during the day.\textsuperscript{69} These include cardiolp-levodopa, pramipexole and ropinerole with similar typical titration approaches up to a maximum for the RLS/WED indication. GABA analogs gabapentin and pregabalin have been used with success and in some studies a lower augmentation rate than the Parkinsonian agents.\textsuperscript{71,72} Exercise has been shown to decrease symptoms and abstinence from caffeine, nicotine, alcohol and antihistamines should be considered.\textsuperscript{73}

PLMD is repetitive, highly stereotypical movements during sleep that is associated with many sleep and neurological disorders including RLS, OSA, REM sleep behavior disorder, narcolepsy, and PD.\textsuperscript{74} The relationship with symptoms such as insomnia and daytime somnolence is inconsistent and therefore controversial. PLMD is diagnosed based on history and observation of limb movements during a sleep study. One should take the view PLMD may accompany another disorder but assignment of a diagnosis of PLMD should be reserved for when unaccompanied by another disorder and the limb movements themselves are suspected to be the causation of excessive sleepiness or insomnia. PLMD is thought to be a distinct and mutually exclusive diagnosis from RLS/WED. Many patients with RLS have PLMD but the converse is not necessarily true. Management of such has rather limited data and most approaches are indeed derived from managing the related disorders, for example, the GABA analogues and dopaminergic agonist agents all seem to affect a reduction in the periodic limb movement index, similar to RLS.\textsuperscript{74} For PLMD without RLS symptoms treatment should focus on the primary disorder with independent treatment of PLMD rarely needed.

Sleep bruxism is teeth grinding and rhythmic masticatory muscle activity (RMMA) occurring during sleep, keeping in mind that up to 60% of normal adults demonstrate RMMA without teeth grinding. Risk factors include obstructive sleep apnea syndrome and other sleep breathing disorders, loud snoring, moderate daytime sleepiness, heavy alcohol use, caffeine consumption, smokers, anxiety and being subjected to a highly stressful life.\textsuperscript{76} Complications and consequences include destruction of tooth structure, periodontal and other dental problems, damage to the TMJ, myofascial pain, muscle contracture and other muscular problems.\textsuperscript{77} There are significant implications for iatrogenic emergence of RLS and bruxism with the use of SSRIs and venlafaxine intended to treat any combination of anxiety, depression and insomnia. Dental splinting device is a common non-pharmacological approach to arrest progression of dental and periodontal complications and should be considered the first line therapy. Referral to dentistry should be sought early in the disease for fitting of an oral appliance. Mandibular advancement devices decrease bruxing but device discomfort is the greatest barrier to consistent use and occlusal devices protect the teeth from further damage but do not decrease bruxing.\textsuperscript{78,79} Pharmacologic interventions are second line therapy, effective agents including clonidine and clonazepam, with therapy considered only when pain affects quality of life or in patients at risk for significant tooth damage.\textsuperscript{78,80} Other medications such as levodopa, propranolol, amitriptyline and bromocriptine have been shown to be ineffective and should be avoided.\textsuperscript{78}

CONCLUSION

Sleep disorders are a common complaint that will be encountered by the family physician. Management can easily be initiated based on history and physical exam. Full polysomnography is not needed for all sleep complaints. Patient centered therapy and education are critical for long term successful treatment.

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