The Assessment and Differential Diagnosis of Insomnia in People With Developmental Disabilities

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Sleep is a complex neurobiological state that is essential for health and cognition. Insomnia is a disturbance in sleep initiation and maintenance that results in significant daytime complications. Insomnia in individuals with intellectual disabilities is a common problem that is often overlooked as a cause of disruptive or aggressive behavior. In most clinical circumstances, sleep data collection is limited to recording the quantity of sleep, offering little information about quality. This paper provides a model of data collection that helps to correct these shortcomings and provide basic data for appropriate referral for more extensive sleep studies.

Keywords: developmental disability, insomnia, intellectual disability, mental retardation, obstructive sleep apnea, psychiatric disorder, sleep arousals, sleep disorders, sleep phase

“Sleep, that knits up the ravell’d sleeve of care,
The death of each day’s life, sore labour’s bath,
Balm of hurt minds, great nature’s second course,
Chief nourisher in life’s feast.”
Shakespeare, Macbeth Act II

Sleep is fundamental to health maintenance, as well as psychological and cognitive function. Even though sleep is natural, we are constantly battling to match the sleep needs of a diurnal primate with the demands of our 21st century industrialized world. In addition to problems with “how long,” we also must learn “when” to sleep. This process is called entrainment and occurs very early in infancy. Over our lifetime, we are constantly redefining this process. As infants we master day-night patterns of sleep. During development, these patterns of nocturnal sleep change in response to changing school, lifestyle, and work schedules, not to mention jet lag and other assaults on our sleep wake cycle. 1,13,31

Sleep follows another developmental course as our patterns of sleep change throughout our lifetime. There are substantial differences between the sleep patterns of infants when compared with their grandparents. In addition, illness, degenerative disorders, psychiatric disorders, and pain affect the nightly rhythms of sleep. Insomnia is the inability to initiate and/or maintain sleep—an inability to fall asleep, frequent or prolonged awakenings during the night, or early morning arising before one’s customary rising time. Each pattern of sleep disturbance tends to adversely affect daytime functioning (severity), by producing daytime fatigue sleepiness, deficits in attention or alertness, and productivity. 1,8,9

Transient insomnia is extremely common in industrial societies and may be attributed to psychosocial stress, occupational demands, and possibly electric lighting. Chronic insomnia presents a more substantial problem for between 10-15% of the general population. Lost productivity, potential for accidents, risk for psychiatric and other health problems, and impairments in attention, memory, and higher cortical functions are unfortunate outcomes of chronic insomnia. 1,9,31 Although there are many exceptions, aging adversely affects total sleep time, as well as the efficiency, quality, and stability of sleep. With respect to gender differences, females are also more likely to develop chronic insomnia than males. A wide variety of neurobiological, hormonal, and psychological factors contribute to these gender and age differences. 19,30

In addition to daytime complaints of drowsiness, poor concentration, and deficits in higher cortical functions, chronic insomnia also contributes to significant psychiatric and medical morbidity, and mortality risk. 8,9 For example, insomnia is a vulnerability factor, trigger, clinical...
symptom, and predictor of treatment response for individuals with mood disorders. Chronic insomnia is also associated with neurodegenerative disorders and other brain disorders. But the relationship is a complex one. Sleep deprivation or insomnia is common in individuals with epilepsy while at the same time insomnia can worsen seizures, and adversely affect the longitudinal course or treatment outcome.

Insomnia is widespread among people across the spectrum of intellectual disabilities. Among these individuals, the prevalence and typology of insomnia and other sleep disorders is strongly influenced by the severity of intellectual disability (ID), underlying brain dysfunction, behavioral phenotype, comorbid neurological conditions, living environment, iatrogenic causes, as well as higher rates of mental disorders. Teasing out the effects of these multiple factors requires an investigation of each factor as well as a good working knowledge of sleep physiology and pathology.

This paper outlines a protocol to aid in the differential diagnosis and monitoring of treatment response. In most clinical settings, sleep disorders are monitored by observational data and sleep records. By itself, this method of data collection may lack the sensitivity to assist in the differential diagnosis of many sleep disorders. To deal with these potential shortcomings, we need to expand the traditional sleep charting methodologies to include data that can be helpful in the initial assessment of insomnia. These modifications will hopefully provide more complete data for differentiating the relationship between insomnia and other sleep disorders. This expanded data set should help in the decision process about referral for more formal sleep studies.

**Sleep and Homeostasis**

Adequate sleep is crucial to normal daytime functioning. Unfortunately, most people in industrialized societies sleep far less than our ancestors did 100 years ago. As a result of these social changes, many of us get insufficient sleep. The causes of this society-wide sleep deprivation include problems with high stress levels, work or academic demands, and a range of other psychosocial or cultural factors. Although we stress productivity and creativity in the workplace, cutting down our total sleep time may not be the best solution. Inadequate sleep has obvious consequences for cognition, motor performance, attention, memory, and most complex executive skills. We compensate for this shortfall by trying to overcome daytime sleepiness with caffeine and other stimulant drugs. In this sense we are burning our candle at both ends and ignoring the importance of sleep as a critical factor in restoring our physiological balance or homeostasis.

Why do we get sleepy? While awake, the brain produces a wide variety of sleep inducing neurotransmitters and modulators (e.g., adenosine and sleep related peptides). When the levels of these compounds rise sufficiently, we feel drowsy or fatigued and are prone to fall asleep. Under normal circumstances the urge to sleep fits into our circadian sleep-wake cycles. When we disrupt this balance, there are negative consequences for healthy sleep. Without sufficient sleep we feel tired, lethargic, irritable, drowsy, or become increasingly inattentive. Even minor fluctuations in our usual sleep time or efficiency can have an adverse effect on a wide range of higher cognitive functions. Sleep deprivation can lead to serious consequences including impairments of complex decision-making processes and executive functions as well as an increased risk for serious accidents.

Even though we are a predominantly diurnal species, we must entrain our sleep-wake cycles in order to concentrate sleep during the nighttime hours. A breakdown in the complex process of entrainment contributes to erratic sleep patterns and disturbs the balance between these homeostatic needs and circadian rhythms. This imbalance can result in significant disturbances in the transition from wakefulness and sleep-difficulty initiating sleep. Most people consider the inability to initiate sleep as insomnia. Clinically, insomnia is a more complex problem. It includes not only difficulty initiating (falling asleep), but also affects sleep maintenance (staying asleep or nocturnal awakenings), the quality of sleep (restful and restorative sleep) and the presence of troublesome, daytime lethargy or drowsiness.

For most of us, a rebound in REM and Stage IV sleep compensate for transient insomnia. Under these circumstances, sleep rebound increases sleep efficiency the following evening. But there are limits to the efficacy of this compensatory mechanism. Conditions that lead to chronic insomnia override these attempts at
compensation and undermine the restorative power of sleep. Over time chronic insomnia disrupts normal alertness and arousal and produces an intrusive and often irresistible urge to sleep (drowsiness) as well as micro-sleep events (catnaps) and sleep automatisms (similar to road hypnosis). Since chronic insomnia affects nearly 10% of the general population, this sleep disorder has not only enormous economic impact, but also adversely affects health, increases the risk accidents, and increases the likelihood of developing new psychiatric disorders.

Some individuals with chronic insomnia have difficulty with high levels of arousal that prevent disengaging from the waking world. There are many factors that contribute to this problem with sleep onset. Stressful events are probably the most common cause of acute insomnia. When dealing with chronic insomnia, however, the list of causes expands to include not only extrinsic factors (e.g., stress, trauma, or drug abuse) but also includes disturbances in the intrinsic processes crucial for healthy sleep. These intrinsic factors include many disturbances in sleep initiation and regulation, circadian rhythms, and underlying brain disorders. Many of these factors are common in individuals with severe ID and comorbid neuropsychiatric disorders.

It seems obvious that chronic insomnia can result in significant sleep deprivation. There is growing evidence for the negative effects of sleep deprivation on normal growth, cognitive development, behavior and general health of children. The adverse effects of sleep deprivation differ symptomatically in children. In contrast to adults, where mental slowing, poor concentration, lethargy, fatigue, and increased daytime somnolence are prominent, children with chronic sleep disturbances more frequently present with hyperactivity, irritability, and impaired learning.

People with developmental disabilities share these differences, but take this developmental trend a step further. Adults with ID may respond to sleep loss with increases in irritability, hyperactivity, aggression, and self-injurious behavior, but may not compensate by increasing daytime sleepiness. Because of these developmental responses to sleep deprivation, the clinician confronted with excessive daytime sleepiness may wish to pursue additional causes for excessive diurnal somnolence—sedation from medications, nocturnal seizures, or other primary sleep disorders.

People with ID appear to have a wide variety of extrinsic factors that influence sleep onset. As a result, the clinician needs to investigate not only the sleep environment, but also other potential sources of protracted arousal. It is also necessary to gather an appropriate sleep history—usual bedtime and time of awakening, average length of sleep, sleep environment, medication history, daytime sleepiness and napping. The purpose of this information is to establish usual sleep patterns and to provide a background for understanding current changes in sleep patterns. In most circumstances, disturbances in sleep onset are related to extrinsic factors and interventions are designed to include proper exercise, evening activities, room temperature and sleep environmental issues, and bedtime routines or rituals. Psychophysiological and conditioned sleep disturbances need to be addressed in sleep hygiene interventions. But these are not the only causes of problems with sleep initiation and maintenance. The persistence of insomnia in spite of adequate sleep hygiene measure requires more investigative work.

Circadian and Ultradian Sleep Rhythms

Sleep is also a rhythmic phenomenon. Daily sleep wake cycles (circadian rhythm) are the most noticeable. Humans are readily entrained to day-night cycles. Light has a major regulating effect on when we sleep (zeitgebers). The anterior hypothalamic, supra-chiasmic (SCN), periventricular nuclei are actively involved in setting circadian rhythms of sleep. Melatonin signals the SCN to day-night fluctuations in light (light suppresses melatonin) but also plays an important role in hormonal and temperature rhythms. Disturbances in the complex interaction between light, melatonin synthesis and excretion, and regulation of sleep onset are major contributors to some forms of insomnia.

One example of a disruption in this process is Delayed Sleep Phase Disorder. This syndrome presents with apparent difficulty falling asleep but except for conflicts with work or school schedule, there are minimal fundamental shifts in total sleep time or quality of sleep. The shift towards later sleep onset and later daytime awakenings is common among teens and early adults.
### Table 1. Diagnostic Data Required to Work Up or Assess Sleep Disorders Before Referral to a Sleep Lab

<table>
<thead>
<tr>
<th>A good sleep record should include:</th>
</tr>
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<tbody>
<tr>
<td>■ Traditional sleep chart data</td>
</tr>
<tr>
<td>■ Age, gender</td>
</tr>
<tr>
<td>■ Caffeine, nicotine, level of physical activity, late evening feeding habits</td>
</tr>
<tr>
<td>■ Psychiatric or neurological diagnoses, including seizure type, severity and frequency</td>
</tr>
<tr>
<td>■ Medical diagnosis (e.g., reflux disease, anemia, renal insufficiency)</td>
</tr>
<tr>
<td>■ Current medications—compare any changes in medicines to time line of the sleep disturbance</td>
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<tr>
<td>■ Onset of sleep problem</td>
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<tr>
<td>■ Duration of sleep problem</td>
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<tr>
<td>■ Snoring or excessive motor activity during sleep</td>
</tr>
<tr>
<td>■ Family history of sleep disorder</td>
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<tr>
<td>■ Past interventions</td>
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<tr>
<td>■ Daytime activities</td>
</tr>
<tr>
<td>■ Bedtime</td>
</tr>
<tr>
<td>■ Time individual falls asleep—sleep latency</td>
</tr>
<tr>
<td>■ Customary rituals prior to bedtime</td>
</tr>
<tr>
<td>■ Environment for sleeping</td>
</tr>
<tr>
<td>■ Total sleep time</td>
</tr>
<tr>
<td>■ Behaviors on awakening—level of consciousness, organization, and complexity of behavior</td>
</tr>
<tr>
<td>■ Ease of awakening in A.M.</td>
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<tr>
<td>■ Daytime naps—timing, duration, and observed behavior, behavior upon awakening from a nap</td>
</tr>
<tr>
<td>■ Staring, reduced eye blinking, automatism (micro-sleeps)</td>
</tr>
<tr>
<td>■ Irresistible sleep attacks—falling asleep at work or during preferred activities</td>
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<tr>
<td>■ Periods of muscle weakness or persistent head nodding</td>
</tr>
<tr>
<td>■ Changes in daytime target behaviors</td>
</tr>
<tr>
<td>■ Treatment plan—behavioral, sleep hygiene, light therapy, melatonin, and psychotropics</td>
</tr>
</tbody>
</table>

Unfortunately, work and school schedules require waking up after inadequate sleep and tend to disconnect the normal nocturnal temperature and hormonal cycles. These “night owls” may spend much of the morning struggling to maintain alertness, get other biorhythms in sync, or remain “half asleep” until later in the morning—their “normal” wake-up time.

Individuals with Advanced Sleep Phase Disorder face a different problem. For example, many elderly people experience an earlier sleep onset, most frequent nocturnal awakenings (middle insomnia) or early morning awakening, but aside from naps, may not have daytime complaints. A careful review of this pattern of sleep reveals that other circadian rhythms are out of phase. These “early birds” or “larks” may get adequate sleep but it occurs earlier in the evening so that a normal 6-8 hours of sleep starts at 8:00 PM and ends around 3 or 4:00 AM.

The Sleep Phase Disorders may require chronotherapy, light therapies, in conjunction with interventions focusing on improving sleep hygiene. For individuals with phase delays, resetting their sleep onset may require maximizing the environments required for sleep onset, delaying sleep for three hours, and phase advancing by the use of light therapy in the morning (simulated early dawn). For individuals with Advanced Sleep Phase Disorders, light therapy is most effective in the evening (simulating later sunset) by creating sleep onset by suppressing the early onset of melatonin release. Seasonal affective disorder may present with excessive sleep, non-restorative sleep, hyperphagia, daytime fatigue and the reversed diurnal variability in lethargy. The timing of effective light treatment suggests that seasonal affective disorder is similar to Delayed Sleep Phase Disorder and generally responds best to early morning light therapy.
Some individuals with major affective disorder appear similar to individuals with Advanced Sleep Phase Disorder—reduced REM latency and disrupted diurnal shifts in cortisol excretion.\textsuperscript{1,2,16} There is a subgroup of patients with major depression who may transiently improve with overnight sleep deprivation. Sleep deprivation can also trigger mania in people with bipolar disorder.\textsuperscript{1,16,30} Both phase advancement and onset of mania can occur during corticosteroids treatment, suggesting a role for the Hypothalamic Pituitary Adrenal axis in sleep and primary psychiatric disorders and during treatment of medical/neurological illness.\textsuperscript{1,3,19,25,31} Cytokines and Interleukins (inflammatory agents) also influence changes in sleep physiology and "sickness behaviors." These compounds are also elevated in individuals with mood disorders. These examples suggest a complex relationship between some psychiatric and neurological disorders and sleep phase disorders.\textsuperscript{1,3,31}

Sleep also occurs in stages. There are recurring cycles throughout the duration of nocturnal sleep. The stages represent a series of phasic shifts in EEG rhythm, cardiovascular, cerebrovascular, and neuroendocrine and muscle activity. As such, we can define the phases of sleep through a progression of 90-120 cycles.\textsuperscript{1,13,31} In general, sleep can be characterized as NREM (nonREM) and REM sleep. NREM sleep begins with the initial phases of drowsiness and drifting off to sleep (Stage I), but then quickly progresses to Stage II sleep (thalamic activation), then progressive slowing of the EEG into Stage III and IV or Slow Wave Sleep (SWS). SWS occurs predominantly early in the evening and is the most restorative stage of sleep. SWS dominate the early sleep cycles but declines as morning approaches. REM sleep or dream sleep represents a significant increase in brain activity, autonomic arousal, and paradoxical paralysis of most voluntary musculature. REM sleep becomes more frequent and intense in the later stages of the nighttime sleep cycle.\textsuperscript{1,13,31,32}

The organization of sleep architecture changes during development. Infants display high levels of active sleep and short latency for REM activity. After about six months of age, there is a gradual increase in SWS, reduction in total REM sleep time, and reduction in total sleep time.\textsuperscript{13,33} By adulthood, most individuals face declines in SWS, but relatively less drop off in REM sleep. By the time most reach their 60's, SWS is disappearing, and REM and other sleep changes predominate.\textsuperscript{1,2,3}

Disturbances in early sleep stages reflect changes in the balance between homeostatic and circadian sleep rhythms. Difficulty falling asleep, maintaining sleep, experiencing sleep as restorative and interference with daytime activities may result from the phasic disturbances.\textsuperscript{1,30} Specific sleep disorders linked to problems with these sleep cycles:

1. Stage I represents the gradual disengagement, some rhythmic sleep movements, sleep starts, problems with Restless Legs Syndrome,\textsuperscript{24} improper sleep environment, over stimulation, medication or drug effects,\textsuperscript{23} poor sleep hygiene, and psychiatric disorders such as anxiety disorders which can affect sleep onset.\textsuperscript{16} Most people will complain of insomnia.

2. Stage II presents with shifts in thalamic activity that result in characteristic EEG changes. During this phase of sleep individuals are still reactive to external events. Individuals with periodic limb movement causing disruptions in Stage II sleep may also complain about insomnia. Bed partners are the most likely to experience significant distress. RLS and periodic limb movements of sleep (PLMS) are often co-existing conditions.\textsuperscript{24}

3. Stage III-IV, or SWS, are essential to normal cognitive functioning. Disturbances in SWS can occur secondary to sleep deprivation, alcohol and other drugs,\textsuperscript{21} aging and some degenerative disorders.\textsuperscript{2,5,6,20} During childhood the most common problems with SWS involve breakdowns in the integrity and stability of stage IV sleep. Paronamias (night terrors and somnambulism) occur out of SWS.\textsuperscript{11,17} Pain from fibromyalgia and other disorders may also disrupt the integrity of SWS (alpha intrusions).\textsuperscript{26} Seizures are also more likely to occur out of this sleep phase.\textsuperscript{4,7} Aging, mood disorders, and dementias have the greatest effects on stage IV (restorative). Disruptions in SWS leave the individual tired, "sleepy" and result in significant cognitive and emotional changes.\textsuperscript{5,9}

4. REM or Paradoxical Sleep occurs out of SWS. It is associated with multiple physiological changes and is the source of dreaming and nightmares.\textsuperscript{31} Sleep apneas, especially obstructive sleep apnea, tend to occur during
REM when the upper airway collapses during periods of muscle paralysis. Unstable REM is noted in disorders such as narcolepsy. This disorder is associated with sleep attacks, variable levels of cataplexy (muscle weakness while awake), sleep paralysis, and hallucinations. REM related behavior disorder represents dreaming without paralysis. In some circumstances violent behavior can occur. REM related behavior disorder is associated with many neurodegenerative disorders that affect the brainstem and midbrain. Many medications suppress REM sleep, and may result in significant REM rebound when they are discontinued.

Each of these disorders may present with at least some features of insomnia. It is important to consider these sleep disorders before embarking on any treatment plan. Many can be eliminated by history and a sleep diary, but some require more extensive studies. Polysomnography, Multi-Sleep latency test, and sleep EEG with telemetry may be required to clarify the specific syndrome.

**Sleep and Developmental Disorders**

Sleep changes during development, and so it follows that sleep is also affected by problems that emerge during brain development. Many individuals with intellectual disabilities have sleep difficulties, but few routinely are referred for sleep studies. The clinical focus is often directed at sleep environments and faulty sleep hygiene. Unfortunately, there is more limited emphasis on the effects of medical conditions such as hypothyroidism, cardiopulmonary disease, chronic renal insufficiency or under treated pain,iatrogenic sleep problems secondary to medications, the impact of visual impairment, primary sleep disorders, and secondary sleep disorders due to comorbid neuropsychiatric disorders. Studies focusing on sleep physiology among individuals with ID note disruption in both homeostatic and circadian-ultradian phases of sleep. Sleep for many individuals with ID resembles that seen in Idiopathic Insomnia; Klein Levin syndrome, sleep phase disorders, and intrinsic hypothalamic-thalamic disorders. Each of these issues underlines the relative dearth of controlled studies of even “normal” sleep among people with ID. The organization of homeostatic and circadian influences is dependent on the integrity of the central nervous system. As a result, we should anticipate that people with severe ID will experience a significantly greater prevalence of sleep problems based on higher rates of neurological complications, structural and functional brain abnormalities, visual impairment, and underlying genetic disorders. For example, epilepsy is associated with significant disruptions in normal sleep. This physiological vulnerability can be accentuated by the use of multiple anticonvulsant drugs that may also affect sleep by excessive daytime sedation, decreased motor activity, or by direct effects of specific drugs on sleep. Visual impairment, disruptions in the pathways from the retina to the hypothalamus, hypothalamic dysfunction, and disorganized melatonin production contribute to disturbances in both the timing of sleep onset, as well as the integrity of sleep architecture. Many individuals with severe visual impairment may be unable to entrain to normal night-day zeitgebers. One problem for individuals living in out-of-home placements is the conflict between highly individualized lifelong sleep patterns and “rules” regarding bedtime and duration of sleep. In these settings, the considerable differences in sleep rituals, customary bedtimes, total sleep time, phase advances and phase delays are not always considered. Depending on temperamental style, many will have trouble adjusting and adapting (entraining) to the new sleep schedule. For these individuals, differences between lifelong sleep patterns and those expected in the new environment may disrupt sleep onset. In this regard, changes in the sleep environment influence the prevalence, or recognition of insomnia.

Even taking these environmental factors into consideration, there still exists considerable heterogeneity among individuals with insomnia. For example, insomnia may be psychophysiological (behavioral or conditioned sleep onset difficulties); programmatic (overzealous awakening schedules to prevent toileting accidents or enuresis); distinguishing daytime naps versus excessive somnolence or sedation; arbitrary bed times that disregard sleep phase needs; RLS/PLMS; pain; GERD; obesity and obstructive sleep apnea; undiagnosed psychiatric disorders and their treatments. Many individuals who are lifelong
### Table 2. Table of Sleep Difficulties

<table>
<thead>
<tr>
<th><strong>Primary Psychiatric Disorders</strong></th>
<th><strong>Difficulty Falling Asleep</strong></th>
<th><strong>Awakening-Middle Insomnia</strong></th>
<th><strong>Early Morning Awakening</strong></th>
<th><strong>Daytime Sleepiness</strong></th>
</tr>
</thead>
</table>
| Depression can be linked to difficulty falling asleep (DFA), early morning awakening (EMA), middle insomnia, daytime sleepiness | **Past pattern of sleep:**  
- Usual bedtime  
- Duration of sleep  
- Time of awakening  
- Evidence of sleep phase delay  
- Risk factors for restless legs  
- Pain | **Past sleep patterns:**  
- Time of bedtime  
- Time and duration of awakening  
- Documented sleep, nocturnal panic, mood, or seizure disorder  
- Behaviors associated with awakening  
- Associated medical conditions | **Past pattern of sleep:**  
- Timing of awakening  
- Duration of sleep prior to awakening  
- Behaviors associated with awakening  
- Efficacy of sleep-short cycle or phase advance may not experience fatigue or continued sleepiness | **Past history of daytime somnolence:**  
- Obesity, OSAS, behavioral phenotype like Prader-Willi  
- REM instability- other symptoms of narcolepsy  
- Most recent seizure of AED regimen  
- Level of environmental stimulation  
- Medical status |
| Mood Disorders | **Phase advance-short REM latency** | **Associated with more severe forms of depression-melancholia** | Melancholia | Atypical, Seasonal, and Bipolar depressed illness; depression may be the underlying cause of daytime fatigue in individuals with sleep related breathing disorders |
| Anxiety Disorders |  
- Increased arousal state  
- Comorbidity with bipolar disorder | Less common, although parasomnias may recur during times of distress | Nightmares may occur, especially severe in post traumatic stress disorder |  
- Over sedation,  
- Alcohol use/abuse  
- Bipolar disorder, depressed phase |
| Seizure Disorder |  
- Fear of seizures during sleep  
- Anticonvulsant effects  
- Comorbid psychiatric disorder |  
- Nocturnal seizures during slow wave sleep  
- Arousal disorders |  
- Comorbid depression  
- Early morning seizures-myoclonus followed by tonic clonic generalized seizures |  
- Atypical Absence  
- Post-ictal state  
- Medication side effects  
- Comorbid primary sleep disorder |
| Degenerative Disorders |  
- Sun downing, nocturnal agitation  
- Medication side effects;  
- Lack of light exposure and daytime activities |  
- Phase advance  
- Frequent awakenings  
- Severe phase advance  
- Comorbid mood disorder |  
- REM related behavior disorders  
- Phase advance  
- Severe mood disorder |  
- Sleep deprivation  
- Boredom  
- Thyroid and other endocrine changes |
short cycle sleepers or those with idiopathic insomnia are referred for medication management while those with primary sleep disorders may go unrecognized.21

**DATA COLLECTION SYSTEMS AND SLEEP DISORDERS**

In most clinical settings, sleep data is condensed into either a sleep chart or graph of the total nocturnal sleep time. Although seriously limited by the accuracy and reliability of the staff data, there are some useful approaches to gleaning information that can be useful for the initial phases of the differential diagnosis of sleep disorders.10,11,29

1. Timing of bedtime:

   a. When is the individual’s normal bedtime? This data should include parental observations, previous reports from other caregivers, and current staff. This data would represent the equivalent of a sleep diary and go towards establishing if this is an artificial result based on arbitrary bedtimes or morning wake-up.

   b. By itself, this data provides little information about why the individual has trouble sleeping. For example, what if bedtime is 8:00 P.M.? An individual with an advanced sleep phase would awaken after eight hours of sleep at 4:00 A.M. and have no problems staying alert and awake during the day. This sleep pattern contrasts with those who have a delayed sleep phase syndrome. These individuals (or “night owls”) have significant delays in sleep onset time, and can be mistaken for those with difficulty initiating sleep. The trouble would come when staff tried to awaken the individual and engage in programming while he was out of sync with respect to the normal sleep cycle. A short cycle sleeper (one who sleeps four hours per night since childhood) might be considered to have early morning awakening, and depending on staff responses, be disruptive or depressed, with efforts then made to “medically” prolong sleep.21,25

   c. Individuals with difficulty falling asleep may emerge under adverse environmental conditions, lack of daytime activity, physical discomfort, restless legs, chronic medical disorders, or a range of intrinsic difficulties with the balance between homeostatic and circadian sleep drives.2,13,25 Daytime somnolence may reflect sleep disruptions such as pathological arousals from sleep,17 sleep obstructive apnea,12,15 nocturnal seizures,4 panic attacks or even bad dreams associated with PTSD27 or other psychiatric disorders.2,16,29 A data system that records only if sleep is interrupted needs to also reflect what happens during this period of awakening or arousal.

d. Difficulties with sleep maintenance may also be seen in chronic insomnia, but these difficulties are also related to arousal problems, nightmares, advanced sleep phase, and many neuropsychiatric disorders.6,30 As before, it is important to describe what happens during the period of awakening. In most situations, night terrors or other parasomnias may appear early in the sleep period. Nightmares may disrupt sleep during the latter part of the sleep cycle.1,13 REM behavior disorders overlap periods of increased REM sleep and follow a similar pattern of nocturnal distribution of nightmares.6 Mood disorders are associated with middle and early morning insomnia, but these symptoms should emerge in tandem with other indicators of mood disorder.2,16,29

e. Daytime sleepiness is usually associated with either sleep deprivation or disruption due to sleep apneas,12,15 or in conjunction with sleep aberrances seen with narcolepsy.32 Obesity, snoring, morning headaches and apneic events are linked to excessive daytime somnolence.12,15 Narcolepsy is associated with an irresistible urge to sleep and sleep attacks. Cataplexy, sleep paralysis and hypnagogic/hypnopompic hallucinations may be seen.32 One of the most common causes of daytime sedation is excessive medication.23 The time, duration, relationship to medication doses, postictal states, boredom and lack of activity may contribute.4,7 Fatigue, hypersomnia, and anhedonia may be associated with mood disorders.16,30 In many children and
individuals with severe ID, sleep deprivation may not contribute directly to daytime sleepiness, but may be associated with increased irritability, aggression, attention deficits, and explosive behaviors.\(^\text{8,9,11}\)

In order to capture these events, the sleep data should include not only a sleep chart, but also a record and description of daytime sleep episodes, duration, response to naps, and behaviors upon awakening. In general, other sleep related issues could be classified based on standardized criteria. In addition to insomnia, other sleep disorders are noted but infrequently diagnosed in individuals with ID. These include:

1. The presence of Disorders of Excessive Somnolence. These may also be overlooked in most sleep data, unless sleep during the day is recorded. Although rare, narcolepsy is probably grossly under-diagnosed in people with ID. Sleep attacks may not be recognized or be confused with post-ictal states; cataplexy may be misconstrued as atonic seizures, syncope, conversion; sleep paralysis never recognized; and hypnagogic/ hypnopompic hallucinations considered as symptoms of psychosis.\(^\text{32}\)

2. Sleep Related Breathing Disorders are among the most common sleep disorders. Obesity, oropharyngeal anatomical abnormalities, and sensitivity of receptors in the brainstem to rising carbon dioxide and declining oxygen levels are the most common underlying causes.\(^\text{12}\) Specific developmental disorders (Down syndrome), obesity secondary to Prader-Willi and Angelman’s syndrome are examples of genetic disorders that increase the frequency of sleep-related breathing disorders.\(^\text{12,15}\) Depending on frequency and duration, these syndromes can be lethal, or result in daytime sleepiness, irritability, morning headaches, and significant cognitive-behavioral decline. Snoring is a reasonable screen, but is not the most effective method of diagnosis—sleep polysomnography is the definitive procedure.\(^\text{12}\)

3. Sleep related movement disorders: Restless legs, periodic limb movements during sleep (disrupted sleep),\(^\text{24,29}\) REM behavior disorder (acting on dream material),\(^\text{6}\) paroxysmal dystonias (odd movements without evidence of epilepsy).\(^\text{4,7}\)

4. Sleep related arousal disorders: Parasomnias generally occur during transitions in and out of stage IV sleep, and can range in severity of autonomic and behavioral arousals.\(^\text{13,27}\)

5. Epilepsy has an adverse effect on sleep, especially SWS (stages III-IV). Many seizures (especially frontal lobe seizures) occur nocturnally and are often missed. Early morning tonic-clonic generalized seizures are associated with some forms of myoclonic epilepsy. Anticonvulsants, underlying effects of seizures on hypothalamic function, and focal EEG changes adversely affect both quality and quantity of sleep.\(^\text{3,4,6,23}\)

**SUMMARY AND CONCLUSIONS**

Data collection for sleep is commonly limited to records of 30-minute intervals of sleep. Unfortunately, such a simplistic approach overlooks the complexity of insomnias, parasomnias, and other sleep disorders. Much of the literature for individuals with ID is limited to problems with sleep hygiene (conditioned sleep onset disorders), or in rare instances, a focus on circadian rhythm disturbances. Blindness, free-running sleep cycles and faulty melatonin production are also well described.\(^\text{10,14,21}\)

Put simply, recording time asleep, even if accurate, does not provide sufficient information to make rational treatment decisions about sleep related disorders. We need qualitative data, i.e., observations of behavior during sleep, sleep related breathing changes and daytime somnolence, just to name a few. Daytime sleep data needs to include the setting (where, when, why, how), duration, and even behavior as the individual is falling asleep or upon awakening to be of much value. Without this data, we are left with a number of hours or intervals that provide little information beyond quantifying nocturnal sleep, or acknowledging daytime drowsiness or napping.\(^\text{10,28,30,33}\)

Unfortunately, many clinicians working with individuals with intellectual and other developmental disabilities have limited general knowledge of sleep medicine. As a result, there is frequently a great deal of confusion regarding sleep data. Another problem emerges from a bias in behavioral data collection towards measurable daytime behaviors. For example, chronic sleep
disturbances adversely affect daytime behaviors. Many clinicians are aware that excessive daytime sleepiness is the result of sleep disruptions, but may not consider hyperactivity, irritability, or increases in disruptive, aggressive, or self-injurious behaviors as symptoms of sleep deprivation.\(^{2,3,9,11}\) Regardless of the reason for disrupted sleep, we need to find methods of data collection and history gathering that allow us to also focus on the qualitative aspects of sleep disorders. This data is invaluable especially if we plan referrals to a sleep laboratory for more specific studies.\(^{14,29}\)

**References**


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