Sleep and Sleep Disorders in Later Life

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Educational Objectives

1. Describe basic changes in sleep that occur as we age.
2. Review several important sleep disorders common in later life
3. Discuss treatment modalities for these sleep disorders and potential benefits of therapy.

Background

Many of us may have little understanding of sleep and the changes that occur as we age. Sleep is not a unitary process but rather subdivides into non-REM (rapid eye movement) sleep and REM or dreaming sleep. Non-REM sleep is then broken up into Stage 1, Stage 2, and Stage 3 sleep. One enters sleep through the portal of light Stage 1 sleep, which normally takes up only about 5% of the night. The next stage of light sleep is Stage 2 sleep, which generally encompasses roughly 50% of the night. Sleep spindles and K complexes on the electroencephalogram (EEG) mark Stage 2 sleep. Particularly when young, we tend to have about 20-25% of the night comprised of Stage 3 or deep sleep. Stage 3 sleep, the final portion of non-REM sleep, is defined by the presence of low frequency and high voltage EEG waves called delta or slow waves. As we grow old, we tend to lose this deep sleep and this attenuation in deep sleep is often more marked in males than females. We tend to be least easily awakened from Stage 3 sleep.

REM sleep differs greatly from non-REM sleep and is defined by rapid eye movements, loss of chin muscle tone, and a low voltage and mixed frequency EEG. Unlike deep sleep, the percentage of REM sleep does not change much over time and generally involves 20-25% of the night. Interestingly, with the exception of our eye muscles and our diaphragm (the principal muscle of respiration), we are paralyzed during REM sleep.

In general, it takes about 15 minutes to enter Stage 2 sleep. This period from wakefulness to Stage 2 sleep is called the sleep latency to Stage 2. It then takes about 90-110 minutes from the onset of sleep until we enter our first period of REM sleep. Thereafter, we cycle through non-REM and REM sleep through the night, generally about three to five REM periods a night for adults. Most of our deep sleep is localized to the first half of the night and most of our REM sleep occurs during the second half of the night. REM periods generally become longer during the night. A long first REM period can clue a sleep specialist to the possibility of a depression.

How long should we sleep? This question elicits some controversy and depends greatly on one’s age. For example, high school students seem to require 9.25 hours of sleep a night. Adults (including the aged) tend to require less sleep than teens, with many specialists recommending about seven to eight hours of sleep a night. Studies suggest that the lowest mortality in adults is associated with approximately seven hours of sleep a night. Both significantly less sleep and greater sleep amounts are associated with reports of greater mortality, for reasons that remain unclear. However,
restricted sleep has been associated with such important sleep disorders as hypertension, diabetes mellitus, and obesity (e.g., Vorona et al. 2005). Whether prolonging sleep duration will lower blood pressure, lower blood sugar, or help reduce weight remains to be established.

**Older Adults**

One consequence of aging is a reduction in deep or slow wave sleep. However, a number of other changes in sleep occur with aging. We tend to spend more time in bed awake. Sleep efficiency is defined as the proportion of time one is asleep while in the bed. In healthy sleepers, sleep efficiency is approximately 90%. However, in the elderly sleep efficiency might drop to as low as 70%. Older adults tend to “phase advance” with their internal clocks encouraging an earlier bed time and earlier wake time. Such a phase advance should not be problematic unless extreme, impairing one’s ability to live an active social life. Further, older adults tend to spend more time in lighter stages of sleep, have more arousals during sleep (mini-awakenings), and to nap more during the day than younger adults.

Several important sleep disorders afflict older adults. We will highlight obstructive sleep apnea syndrome (OSAS), Restless Legs Syndrome/Periodic limb movements of sleep (RLS/PLMS), REM sleep behavior disorder (RBD), and chronic insomnia.

OSAS is the repetitive complete or near complete occlusion of the upper airway during sleep. This syndrome is common and becomes increasingly so, probably plateauing in prevalence after age 65 (Mehra et al, 2007). Symptoms of OSAS include snoring, nocturnal choking, gasping and pauses, as well as non-restorative sleep and daytime sleepiness. Patients might also note morning headaches, nocturia, mood disturbances, and enuresis (wetting the bed).

OSAS looks to be a common secondary cause of hypertension and has been associated with an increased risk of congestive heart failure, irregular heart rhythms, and stroke (Bradley & Floras, 2009). Moderate to severe and severe OSAS have also been linked to increases in “all-cause mortality” (Marshall et al., 2008). For two decades data have demonstrated that patients with OSAS are significantly more likely to have car crashes (Findley et al., 1988).

The diagnosis of OSAS is optimally established at an American Academy of Sleep Medicine accredited sleep disorders center or accredited sleep disordered breathing laboratory. After a history and physical examination, the patient undergoes a polysomnogram (sleep study) at the sleep center, during which the patient typically spends a full eight hours in a special bedroom. Such sleep studies measure a number of physiologic signals that include brain waves, eye movements, nasal and oral air flow, electrocardiogram, chest and abdominal wall movements, oxygen level, and leg movements. While the patient sleeps, sleep technologists or technicians monitor the signals, troubleshoot, and care for the patient’s needs. Technicians subsequently score the raw data; a sleep specialist then reviews their scoring and raw data, generates a report, and establishes a diagnosis. OSAS is defined by the presence of at least five partial (hypopneas) or complete pauses (apneas) in breathing per hour of sleep, described as the AHI or apnea-hypopnea index. Although far from a perfect metric, sleep specialists assume that greater number of apneas or hypopneas demonstrate more severe sleep apnea.

A number of treatment options are available for OSAS subjects. Generic treatments can include weight loss and reduction or avoidance of alcohol. One study suggests that a 10% decline in BMI leads to a 26% reduction in AHI. Patients should be instructed not to drive if sleepy and to adopt countermeasures to sleepy driving that include caffeine and napping.

Beyond these general recommendations, positive airway pressure (PAP), dental appliances, and surgery offer effective OSAS treatments. PAP applies a positive pressure to the OSAS patient’s upper airway, thus splinting open the airway and preventing the recurrent partial or complete upper airway occlusions of OSAS. PAP has been demonstrated to improve quality of life, lessen daytime sleepiness, reduce car crash rate to baseline rates, and improve mood (George, 2001; Kawahara et al., 2005). PAP may also lower blood pressure, reduce blood sugar in diabetes mellitus, improve insulin resistance, and improve ventricular dysfunction in congestive heart failure (Dorkova et al., 2008; Mansfield et al., 2004). Some early data even suggest that over the long term PAP
may reduce cardiovascular mortality. Thus, it is gratifying that an increasing database demonstrates the utility of treating OSAS.

Dental appliances for OSAS are worn over the teeth during sleep (almost akin to a football player’s mouthguard) and can be effective in mild to moderate sleep apnea. Most believe that patients must have eight to ten teeth both above and below to anchor such devices. Dental specialists should fashion these devices. A number of surgical procedures, usually performed by Ear Nose and Throat surgeons and/or Oral Maxillofacial surgeons, can treat OSAS. With older adults who may have co-morbidities, surgery should be reserved for significant OSAS that has not responded to the application of less invasive maneuvers.

Restless legs syndrome (RLS) afflicts roughly 10% of the population in the United States. As defined by the International Restless Legs Syndrome Study Group (IRLSSG), four basic criteria must be satisfied to diagnose RLS: 1) The patient describes an urge to move and almost always with accompanying paresthesias (an antsy or creepy-crawly sensation); 2) The urge to move occurs at rest; 3) A clear circadian rhythm exists with symptoms being most problematic in the evening/night; and 4) Movement rapidly alleviates symptoms, although symptoms can occur with resumption of rest.

Approximately 60% of those with RLS are females and most are ages 50 or older. Patients with RLS often complain about an inability to initiate or maintain sleep and not infrequently manifest mood disorders. The clinician evaluating RLS must remember that, although many cases of RLS are idiopathic, a number of secondary causes can underpin RLS. For example, iron deficiency, end stage renal disease, and multiple sclerosis have all been associated with RLS (e.g., Hening et al., 2007).

Physicians and ancillary clinicians can diagnose RLS through history and physical examination. A polysomnogram is superfluous if the clinician does not suspect another sleep disorder. Some 80% of RLS patients demonstrate periodic limb movements of sleep (PLMS) on sleep studies. PLMS are repetitive and stereotypic leg jerks. RLS treatment should ensue if symptoms are frequent enough and onerous enough to prevent adequate sleep. Patients should be urged to minimize caffeine and alcohol and to avoid anti-histamines. Some will gain relief through warm baths, massage or exercise. Medications to include dopamine agonists (e.g., ropinirole and pramipexole), gabapentin, and opiates all can be effective. A recent study also suggests that intermittent pneumatic compression devices can offer relief to RLS sufferers.

REM sleep disorder. As noted above, normal REM sleep leaves the sleeper paralyzed with the exception of diaphragm and oculomotor function. REM sleep behavior disorder (RBD) is a fascinating disorder in which patients act out dreams that often are comprised of violent content. Patients can be noted by their bed partner to holler and/or punch or kick in their sleep. Patients with RBD may abruptly leave the bed, and are apt to injure themselves and more likely to injure their bed partner. RBD most commonly afflicts older men. Evaluation should be performed by a sleep specialist who may order specialized polysomnography to include video, extra limb electromyographic leads, and more involved electroencephalographic leads (to interrogate for seizures). A paradoxical loss of REM atonia in the chin, arm and/or leg EMG leads strongly supports RBD.

Treatment of RBD should first involve making the bed area safe for both patient and the bed partner. Clonazepam effectively attenuates RBD symptoms in roughly 90% of patients (Olsen et al., 2000). Case reports suggest that caffeine can exacerbate RBD, so a reduction of intake could be worthwhile. Clinicians should be alert to the use of selective serotonin reuptake inhibitors (certain antidepressants) in patients who present with RBD. Finally, RBD not infrequently presents with or presages degenerative neurological diseases such as Parkinson’s disease and Lewy Body dementia (Olsen et al., 2000). A full neurological evaluation in patients with RBD is worthwhile.

Chronic insomnia. One can define insomnia as the inability to initiate sleep or maintain sleep, the presence of early morning awakenings or the belief that sleep quality is poor. These complaints should occur in the presence of an adequate opportunity to sleep and with daytime impairment. Insomnia more commonly troubles women than men and occurs more often with increasing age. Clinicians can most effectively address insomnia
by remembering to take an organized approach that evaluates and treats the following possible cause(s): 1) Environmental issues like a noisy or hot bedroom; 2) Mood disorders, as there likely is a bidirectional relationship between depression and insomnia; 3) Medical disorders such as asthma/COPD, arthritis, reflux or prostatism; 4) Intrinsic sleep disorders such as OSAS (50% of OSAS subjects have insomnia complaints) or RLS; 5) Sleep hygiene abnormalities, e.g., chaotic bed and wake times, excessive alcohol and caffeine or using the bed to watch TV or work; 6) Circadian rhythm disorders such as Advanced Sleep Phase syndrome; and 7) Psychophysiological insomnia with hyperarousal in the bedroom and learned maladaptive behaviors.

Perhaps the most contentious issue in the treatment of chronic insomnia involves the use of behavioral interventions versus sleeping pills. Most sleep specialists recommend the use of behavioral interventions such as stimulus control, sleep restriction or cognitive behavioral interventions as opposed to long term sleeping pill use. However, a small but burgeoning literature supports the long term use of sleeping pills for insomnia.

Case Study #1

Mr. S. arrived at the physician’s office aghast that he struck his wife last night during his sleep. This 65 year old patient relates that his aggrieved wife awakened him immediately and that he remembered punching at an attacker in his dream. A phone call to his wife alerts the physician to the fact that Mr. S. has a long history of modest snoring but no history of nocturnal gasping or pauses in respiration. He has been yelling and moving in his sleep for some time. He admits to no history of depression and takes only a diuretic for his hypertension. No family history of sleep disorders exists and his review of systems reveals only the recent increase in drooling.

On physical exam the physician noted a subtle shuffling of gait, as well as a resting tremor and some cogwheel rigidity. The physician, suspecting RBD, scheduled Mr. S. for a sleep evaluation and probable expanded polysomnogram. The subsequent sleep study revealed no sleep apnea (thus no pseudo-RBD) but demonstrated a marked increase in muscle tone in REM sleep. The physician instituted clonazepam with resolution of RBD symptoms, and in follow-up a neurological consultant confirmed the diagnosis of Parkinson’s disease.

Case Study #2

Ms. R. reported a long history of both problems falling asleep and staying asleep. She believes that she obtains but six hours of sleep a night and finds herself increasingly irritable and unable to concentrate. She admits to a long history of depression that has been well controlled through the use of bupropion. She notes that two previous physicians have treated her insomnia with zolpidem and more recently temazepam with but modest success. She fervently denies snoring or witnessed pauses in respiration. When asked to explain specifically why she cannot sleep, she admits to an urge at rest (and at night) to move her legs associated with a feeling that she simply cannot describe. The physician then elicits that walking quickly reduces her symptoms but only transiently. Ms. R. notes that she drinks seven to eight glasses of sweet tea daily and that her late mother had the “fidgets”. The physician recommends marked reduction of caffeine and checks a serum ferritin level, which returns normal. Initiation of a dopamine agonist relieves her RLS and her insomnia.

Conclusion

Clinicians, older adults, and others should be alert to the possibility of sleep disorders in later life. Sleep disorders in older adults are common, may have substantive consequences, and often can be treated effectively.

Study Questions

1. What changes occur in sleep architecture as we age?
2. What are some of the major consequences of untreated obstructive sleep apnea syndrome?
3. Do most sleep specialists prefer the use of behavioral interventions or long term sleeping pills to treat chronic insomnia?

References


General Recommended Reading or Sources:

A good source for the public: [www.sleepfoundation.org](http://www.sleepfoundation.org)

Information about specific sleep problems: [www.sleepeducation.com](http://www.sleepeducation.com)

General information from NIH: [www.nhlbi.nih.gov/health/public/sleep/healthy_sleep.htm](http://www.nhlbi.nih.gov/health/public/sleep/healthy_sleep.htm)

About the Author

Robert Daniel Vorona obtained his M.D. degree from the University of Virginia, completed his internal medicine residency at the University of Michigan in Ann Arbor, and his pulmonary fellowship at the University of North Carolina at Chapel Hill. He is board certified in Internal Medicine, Pulmonary Medicine, and Sleep Disorders Medicine (American Board of Sleep Medicine). Dr. Vorona currently serves as the medical director of the Eastern Virginia Medical School/Sentara Norfolk General Hospital Sleep Disorders Center and as an Associate Professor in the Division of Sleep Medicine at EVMS. He serves as the first president of the Virginia Academy of Sleep Medicine ([www.vasleepmedicine.org](http://www.vasleepmedicine.org)).