With Age Comes Wisdom, but Also Insomnia

BY KERRI WACHTER
Senior Writer

SAN JUAN, P.R. — Sleep disorders become more common with increasing age, but effective behavioral and pharmacologic therapies are available, sleep experts said at the annual meeting of the American Association for Geriatric Psychiatry.

"Most older adults are not being treated for their insomnia, and most older adults won't get a diagnosis of insomnia," despite the high prevalence of the disorder among this age group, said Dr. Phyllis C. Zee, medical director of the Sleep Disorders Center at Northwestern Memorial Hospital in Chicago.

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There are several age-related changes in sleep architecture. The number of awakenings during sleep time increases, especially in the early morning. The amount of light sleep is increased, and the amount of deep sleep is decreased. There also is a decrease in REM sleep, said Dr. Zee, who also serves as a professor of neurology, neurobiology, and physiology at Northwestern University, Chicago.

Two major mechanisms regulate sleep in humans: the homeostatic drive and the circadian drive. Control of the circadian system resides in the suprachiasmatic nucleus, which provides timing information for physiologic, hormonal, and behavioral rhythms.

Several changes in circadian sleep rhythms come with age. The amplitude of circadian rhythms decreases, while the variability of circadian rhythms increases. "There's also a very noticeable advance in [the] phase of circadian rhythms," Dr. Zee said. Severe disruptions of the sleep/wake cycle often occur among older adults with dementia and in those in nursing homes.

The homeostatic drive for sleep depends on accumulating enough hours of wakefulness to trigger sleep, and this drive is reset during sleep. It's thought that the homeostatic drive is regulated by the ventrolateral preoptic area of the hypothalamus.

It's important to understand these sleep mechanisms when treating sleep disorders. "There is not a thing you can do to make yourself go to sleep. … What you can do is arrange the circumstances and timing of your wakefulness in a way that makes the involuntary process of sleep more likely," said Dr. Daniel J. Buysse, medical director of the sleep evaluation center at the Western Psychiatric Institute and Clinic of the University of Pittsburgh

"Sleep hygiene education is without a doubt the most widely employed and … the least efficacious" of the behavioral treatments for insomnia, Dr. Buysse said. Most patients are already aware of many of the suggestions for good sleep hygiene, such as avoiding caffeine before bed.

To understand the patient's sleep habits, behavioral therapists start by asking about average time in bed, average rise time, total time in bed, time to fall asleep, amount of wakefulness during the night, and total wake time.

Using this information, they calculate the average amount of total sleep (total time in bed minus total wake time). For most individuals with insomnia, there is a discrepancy between the total amount of sleep that they get and the total amount of time they spend in bed, said Dr. Buysse, also a professor of psychiatry at the University of Pittsburgh.

Several common elements are involved in behavioral treatments for insomnia: monitoring sleep-wake patterns, reinforcing associations between bed and sleep, limiting awake time in bed, establishing a regular sleep-wake schedule, and using voluntary behavior to influence the involuntary physiologic process of sleep.

To help patients minimize insomnia, he advised doing the following:

- ► Restrict time in bed.
- ► Establish a regular wake time.
- ► Go to bed only when sleepy.
- ► Stay in bed only when asleep.

By putting these suggestions into practice, a patient's total time in bed should be equal to the total sleep time, plus about 30 minutes, Dr. Buysse said. He and his colleagues have tested the effect of these changes in sleep behavior on sleep quality in a small study of 13 patients who made these changes, compared with 12 subjects who received basic sleep information. Those in the active treatment group showed a significant improvement in sleep quality, while those in the control group showed no change.

In addition, those in the active treatment group also showed improvement in sleep latency—how long it takes to fall asleep—and waking after sleep onset, while controls did not.

Pharmacologic management of acute and chronic insomnia includes benzodiazepine receptor agonists, melatonin, melatonin receptor agonists, and antidepressants.

In 2005, a National Institutes of Health state-of-the-science panel noted that hypnotics are efficacious in the short-term treatment of insomnia. However, with the exception of eszopiclone, the benefits of these agents have not been studied for long-term use.

Zaleplon (Sonata), zolpidem (Ambien), and eszopiclone (Lunesta)—benzodiazepine receptor agonists—are all indicated for the treatment of insomnia.

"These drugs differ [from benzodiazepines] mainly in terms of their pharmacokinetics," said Dr. Buysse. Otherwise, these drugs are quite similar to benzodiazepines. One note of caution, however: Benzodiazepines and related drugs have been shown to be a risk factor for falls.

Ramelteon, a melatonin receptor agonist, "takes advantage of the circadian system that secretes melatonin at night," Dr. Buysse said. Ramelteon is short acting and has an active metabolite. Caution should be used with this drug when prescribed for patients also taking fluvoxamine, which inhibits some of the enzymes that degrade ramelteon.

Ramelteon has been shown to reduce sleep latency and increase total sleep time in both younger and older adults. The drug appears to be less effective on wakefulness after sleep onset.

Ramelteon "has fewer side effects of the sort that characterize benzodiazepine receptor agonists," Dr. Buysse said. In addition, the drug is unscheduled.

Trazodone, an antidepressant, seems to improve sleep continuity. "When it's been assessed in insomnia, there have been variable results. Typically, it decreases wakefulness during the night but doesn't have as much effect on the time to fall asleep," Dr. Buysse said.

He recommends starting pharmacotherapy with a short-acting benzodiazepine receptor agonist or ramelteon. If that doesn't work, he recommends using a low-dose (20-50 mg) antidepressant such as trazodone, amitriptyline, or doxepin. As a last resort, he suggests combining a benzodiazepine receptor agonist with an antidepressant. ■

Phobic Anxiety Found to Promote Coronary Artery Disease Mortality

BY HEIDI SPLETE
Senior Writer

DENVER — Phobic anxiety was significantly associated with both ventricular arrhythmia and mortality in coronary artery disease patients during a median 3-year follow-up, said Lana Watkins, Ph.D., at the annual meeting of the American Psychosomatic Society.

The relationship between sudden cardiac death and phobic anxiety in particular has not been well studied, Dr. Watkins noted. She and her colleagues at Duke University Medical Center in Durham, N.C., evaluated 941 adult patients who were being treated for coronary artery disease. A majority of these patients were white males. About a third of the patients did not have high school diplomas, and smoking and obesity were common among patients in the study.

The highest number of arrhythmias occurred among those patients with the highest levels of phobic anxiety based on the Crown-Crisp index, which rates eight types of phobias, including fear of heights, crowds, and closed spaces.

Overall, the highest tertile of phobic anxiety scores had twice as many females as males, and a higher level of phobic anxiety was significantly associated with female sex, minority status, increased body mass index, and younger age.

Despite the finding of an association between phobic anxiety and ventricular arrhythmias, no significant relationship was found between phobic anxiety and sudden cardiac death, Dr. Watkins noted. During a follow-up period, 134 patients died, and 46 of these met the criteria for sudden cardiac death. Sudden cardiac death was defined as death within 72 hours of collapse, in order to account for deaths of patients who lived alone.

However, mortality was highest among patients with high levels of phobic anxiety, Dr. Watkins said. The predictability of phobic anxiety for both mortality and an increased risk of ventricular arrhythmias was maintained in a regression analysis after other predictors of mortality including age, gender, education level, and comorbidities were adjusted for. ■

Study Links Cognitive Decline With End-Stage Liver Disease

DENVER — End-stage liver disease was associated with significant deficits in memory, abstract thought, sustained attention, and executive function in a study of 104 adult patients, Tina Meyer, Ph.D., reported in a poster presented at the annual meeting of the American Psychosomatic Society.

"We want to enlighten the surgeons and primary care doctors that cognitive decline can indicate serious liver problems," Dr. Meyer said in an interview. She and her colleagues in the Transplant Institute at the Henry Ford Health System in Detroit enrolled liver disease patients who met medical and psychosocial criteria for a transplant. About half (51%) were male, 74% were white, and the average age was 54 years. The patients' mean score on the model for end-stage liver disease (MELD) was 11.3.

The participants completed a cognitive assessment including the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), which estimates brain function in five different domains: immediate memory, delayed memory, visuospatial abilities, language, and attention.

Overall, scores on the RBANS were below average on the subtests of immediate memory, visuospatial abilities, and attention. After the investigators controlled for education levels, higher MELD scores were significantly associated with lower scores on the immediate memory and delayed memory subtests of RBANS, as well as with lower scores on the Mini-Mental State Exam, the Shipley Institute of Living Scale, and the Trail-Making Test, parts A and B.

—Heidi Splete

Pages 70a—70d