



## Sleep in the elderly What is normal?

Steven H. Feinsilver, MD

*Division of Pulmonary and Critical Care, North Shore University Hospital, 300 Community Drive,  
Manhasset, NY 11030, USA*

### **Normal sleep**

Physicians have known for decades that sleep is much more than decreased consciousness. Sleep is divided into two distinct types: REM and non-REM, and these two stages of sleep differ from each other as much as they differ from wakefulness. Within non-REM sleep four distinct stages are seen, which are discussed in this article.

### **Monitoring sleep**

Polysomnography refers to the simultaneous recording of multiple physiologic parameters during sleep. Sleep is staged by monitoring brain waves via electroencephalography (EEG, generally at least two channels); muscle activity, typically the chin muscles, via electromyography (EMG); and eye movements via electrooculography (EOG). These electrical signals are measured by attaching pairs of electrodes to a patient: one pair is a recording electrode, the other a reference electrode. The electrical signals become inputs into a differential amplifier whose output is proportional to the voltage difference between the two inputs. Thus, identical in-phase signals are rejected, eliminating some sources of interference such as 60 Hz electrical noise.

Sleep EEG differs from clinical neurology EEG monitoring in several important ways. Typical clinical EEG requires placement of multiple electrodes that record potential differences between relatively small areas of the scalp. For polysomnography, although the same “International 10–20” system for electrode placement is used, only a few electrodes are placed. A recording electrode on one side of the scalp is typically referenced to the region of the opposite ear. This allows a more global view of the electrical activity of the brain, rather than

---

*E-mail address:* [stevenf@nshs.edu](mailto:stevenf@nshs.edu)

attempting to localize small areas of brain function. In addition, for largely practical purposes because long periods of time are being monitored, the typical recording is viewed at 10 mm per second, rather than the much faster paper speeds typical of clinical EEG. Both of these differences may make seizure activity more difficult to observe on polysomnograms. When seizure activity is specifically being looked for, faster recording speeds or additional electrodes may be required.

By convention electrodes applied to the left side of the head are given odd numbers and the right side even; these electrodes are labeled C for central, O for occipital F for frontal, A for auricular, and so forth. Thus, a typical recording channel C3-A2 refers to a central left-sided electrode referenced to the right ear. Decisions about sleep stages are based primarily on central electrodes.

### Staging sleep

Because of the foresight of some of the founders of sleep medicine, the rules for staging sleep have not significantly changed since the original description by Rechtschaffen and Kales in 1968 [1]. Their description was the result of an ad hoc committee meeting of 12 experts in the staging of human sleep.

In this system, sleep is staged largely by determining the predominant pattern in 30-second “epochs” of EEG, muscle, and eye movement activity. This is often a difficult and highly subjective assessment. The EEG is assessed for wave frequency and some specific events and patterns.

EEG activity can be described as alpha, beta, delta, and theta frequencies (Table 1). Alpha represents 8- to 13-Hz activity and is seen in relaxed, but awake, men and women with eyes closed. Beta activity is greater than 13 Hz, and is seen in awake, active subjects. Delta waves, also known as slow waves, are 0.5- to 2-Hz waves, at least 75  $\mu$ V in amplitude, representing the deepest stages of sleep. Theta activity is 3 to 7 Hz, and is seen in light sleep (non-REM stage 1); a variant of theta with notched “saw tooth” waves may be seen in REM sleep.

Various EEG events are also used to stage sleep (Table 2). Sleep spindles are greater than 0.5-second episodes of 12- to 14-Hz activity that may have a characteristic waxing and waning (spindle-shaped) appearance. K complexes are biphasic, large waves with an initial negative deflection (up on EEG tracing) followed by a positive component (down), lasting at least 0.5 second. Both

Table 1  
EEG frequencies

Type	Characteristic	Significance
Alpha	8–13 Hz	Normal, quiet wakefulness with eyes closed
Beta	>13 Hz	Awake, active subject
Delta	0.5–2 Hz, at least 75 $\mu$ V	Deep non-REM sleep
Theta	3–7 Hz	Light sleep, REM

*Abbreviations:* EEG, electroencephalogram; REM, rapid eye movement.

Table 2  
EEG events

Type	Characteristic	Significance
Sleep spindles	12–14 Hz, > 0.5 s, wax and wane	Stage 2 sleep
K complexes	Sharp negative (up), slower positive, > 0.5 s	Stage 2 sleep
Vertex sharp waves	Sharp negative deflections	Stage 1 sleep

*Abbreviation:* EEG, electroencephalogram.

K complexes and sleep spindles are characteristic of stage 2 non-REM sleep. Vertex waves are sharp negative deflections, usually in theta-range frequency, seen particularly in stage 1 sleep.

### *Stages of normal sleep*

Stage 1 non-REM sleep represents very light sleep from which a person easily can be aroused. The transition from wakefulness to stage 1 is marked by the cessation of alpha activity on the EEG and often accompanied by slow “rolling” eye movements on EOG. The predominant pattern is low-voltage mixed-frequency activity. Theta activity is common and vertex sharp waves may be seen. Spindles or K complexes may not be present; their occurrence requires stage 2 sleep.

Stage 2 non-REM is defined by the appearance of sleep spindles and/or K complexes. These events may occur at varying intervals. If an interval lasts 3 minutes or longer without a K complex or spindle, it is scored as stage 1 (“3-minute rule”). Most of a typical night’s sleep is spent in stage 2.

Stage 3 and 4 are often referred to collectively as delta sleep or slow wave sleep. In stage 3, at least 20%, but not more than 50%, of the epoch consists of delta waves. Stage 4 requires more than 50% of delta wave activity. Slow wave sleep is the deepest sleep with the highest arousal threshold. Children spend more of their night in slow wave sleep than adults. The absence of slow wave sleep is not unusual in elderly individuals.

REM sleep consists of relatively low-voltage, mixed-frequency EEG activity somewhat similar to stage 1 or wakefulness, with the appearance of episodic REMs. Alpha activity may be present, often at a slower frequency than waking alpha. One of the prime characteristics of REM sleep is a very low level of muscle tone. The EMG during REM must be at the lowest level of the recording. Spindles or K complexes should not be present; if two K complexes or spindles are seen within a 3-minute period the interval between is scored as stage 2 if there are no REMs during the interval (essentially the same 3-minute rule stated previously).

Because REM sleep is characterized by a somewhat awake-appearing EEG and very decreased muscle tone, it should not be surprising that this is when dreaming occurs. The loss of muscle tone protects against acting out the plot of a dream. Any dream with developed content is thought to occur during REM sleep. REM sleep is also the period of greatest respiratory and cardiac instability during sleep.

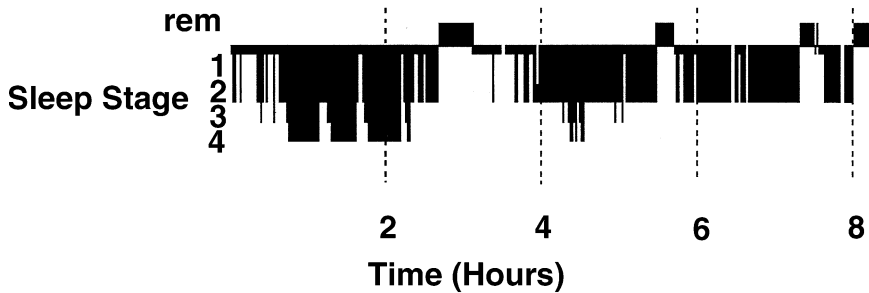


Fig. 1. Normal sleep histogram (hypnogram).

### *Sleep architecture*

The progression of sleep stages across the night is called “sleep architecture,” which is displayed as a sleep histogram or “hypnogram.” (Fig. 1). Typically, subjects pass through stages of non-REM sleep with a REM period every 90 to 120 minutes throughout the night. More stage 3 and 4 sleep is generally observed in the first half of the sleep period, and more REM sleep during the second half. Brief awakenings (at least one 30-second epoch of wake) punctuate normal sleep in essentially everyone, although only the longest of these will be remembered on waking. Arousals, defined as a change in EEG frequency lasting at least 3 seconds, also are a feature of normal sleep, becoming more frequent as sleep quality worsens. Awakenings and arousals become more frequent with aging.

The time to the first REM period is known as REM latency. Normal REM latency is greater than about 90 minutes, although there is wide variability. A short REM latency may be seen in depressed or elderly individuals. A very short REM latency suggests narcolepsy. A prolonged REM latency is commonly seen during the first night in a laboratory setting.

### **Normal sleep in the elderly population**

Changes in sleep timing and quality occur with normal aging. With aging, it becomes both more difficult to stay asleep at night and more difficult to stay awake during the day. The total sleep time in a 24-hour period appears to decline with advancing age, but a decrease in nocturnal sleep time is partially offset by increased daytime napping. It is not clear whether older people need less sleep but it appears that at least nocturnal sleep time declines with age.

An expected feature of geriatric sleep is a decline in sleep efficiency: the ratio of time asleep to time in bed. This is mostly the result of more awakenings during the night, some of which can be prolonged. Multiple awakenings are a frequent complaint among elderly individuals. The time needed to fall asleep (sleep latency) is not generally problematic.

Table 3  
Sleep in healthy elderly individuals

Parameter	Characteristic
Total sleep time	Reduced
Sleep efficiency	Reduced
Wake after sleep onset	Increased
REM latency	Slightly reduced
Delta sleep	Reduced
Sleep latency (MSLT)	Reduced

*Abbreviations:* MSLT, Multiple Sleep Latency Test; REM, rapid eye movement.

Sleep architecture changes with advancing age. The amount of REM sleep stays reasonably constant throughout adult life but REM latency shortens slightly with advancing age. This latency also shortens with depression, which is commonly seen in this age group, and may color this observation. A striking finding of sleep in elderly persons is a reduction in slow wave or delta sleep (stages 3 and 4). This appears to result more from a reduction in EEG amplitude rather than from a decrease in slow wave–frequency activity. This may be an artifact of measurement because EEG amplitude depends on the electrical resistance of the skull and scalp, which increases with age. Thus, if the requirement of a 75- $\mu$ V amplitude for delta waves is eliminated, slow wave sleep may appear more normal in elderly persons [2].

Daytime functioning remains the only measure of the significance of these sleep changes in elderly persons. The best validated measure of daytime sleepiness is the Multiple Sleep Latency Test (MSLT), which measures the ability of a subject to fall asleep during four or five 20-minute periods during the day in a laboratory setting. This test has been criticized for testing the ability to fall asleep, rather than the ability to maintain wakefulness, which might be more relevant to daytime function. Sleep latency measured in this fashion is significantly reduced in elderly persons suggesting they are more somnolent than the general population. In the Cardiovascular Health Study, which reported on 4578 adults older than 65 years, 20% of participants reported being “usually sleepy in the daytime.” Sleepiness was more common in those with depression, loud snoring, medication use for heart failure, and sedentary lifestyle, among other factors [3].

A summary of changes in sleep in healthy elderly individuals is shown in Table 3.

## Insomnia

Insomnia can be defined as the inability to fall asleep, the inability to stay asleep, or the subjective complaint of poor sleep quality. Elderly persons frequently complain of poor sleep and are likely the largest consumers of hypnotics. In one recent population study, 52.7% of prescriptions for hypnotics were for patients aged 65 or older [4]. In the Berlin Aging Study, a random

Table 4  
Causes of poor sleep in elderly individuals

Problem	Examples
Behavioral	Retirement reducing need for regular wake time Napping
Medical illness	Chronic cardiac or pulmonary disease Any disease causing chronic pain (arthritis) Chronic renal failure
Medications	Diuretics (nocturnal wakes)
Psychiatric illness	Depression
Primary sleep diseases	Sleep-disordered breathing Periodic limb movements of sleep

sample of persons aged 70 to 100 years, 19.1% of the participants were taking some form of sleep medication [5].

Some of the more common causes of poor sleep in elderly men and women are shown in Table 4. Many of these causes are behavioral. Poor sleep hygiene, dietary habits, and excessive daytime napping may be detrimental to nocturnal sleep. A predictable, constant wake schedule is beneficial for good sleep. In many patients retirement releases patients from this requirement and it is not uncommon for sleep complaints to follow this lifestyle change. In addition, many medical illnesses make sleep difficult, including chronic cardiac or pulmonary disease, and any condition associated with chronic discomfort. Many medications affect sleep; these effects vary from patient to patient and are incompletely understood.

Evaluation of insomnia in elderly individuals should begin with a medical evaluation for diseases and medications that may be causing the insomnia. Psychiatric consultation may be indicated, especially for depression, which is a frequently occurring disease in elderly persons and a frequent cause of sleep complaints. Specific diseases of sleep, including sleep apnea and periodic limb movements, are discussed later. Finally, it is important to distinguish between treatable complaints and the normal changes in sleep with aging, especially frequent wakes, that should not prompt treatment other than explanation and reassurance. Elderly patients should be taught that it is normal to awaken several times during the night, and that this will not seriously interfere with their next-day functioning as long as they can get back to sleep in a reasonable time.

Most of the treatment for insomnia in elderly patients should be behavioral. Patients should be instructed to choose a constant wake time to maximize the efficiency of their “body clock.” The technique of sleep restriction therapy, in which patients are instructed to limit their time in bed, helps associate going to bed with going to sleep and is relatively easy to teach [6,7].

Because elderly patients have trouble staying asleep during the night and awake during the day, napping during midafternoon may be a reasonable strategy. In one study of nine healthy 74- to 87-year-old men and women, a 90-minute nap schedule improved evening performance measures and produced only a trivial decrease in nocturnal sleep efficiency [8]. A study of 455 70-year-old men and women in Israel, however, showed that those who reported habitual napping had

a significantly higher mortality at 6.5 years of follow-up (20% versus 11%) [9]. It is difficult to attribute causality to this observation.

When hypnotic medication is believed to be necessary, use of one of the newer nonbenzodiazepine agents may be advantageous. Both zolpidem and zaleplon have short half-lives, to avoid morning drowsiness, and have a more specific action on benzodiazepine receptors, causing sedation with much less muscle relaxation than benzodiazepine hypnotics. This may be a particular advantage in elderly patients who may be at risk for accidental falls during nighttime wakes while affected by muscle relaxants. Use of exogenous melatonin has generally been disappointing as a sleep aid. One study has shown that elderly patients with disturbed sleep do not have lower melatonin levels than elderly subjects with normal sleep [10].

### **Sleep-disordered breathing**

Sleep-disordered breathing is a broad term for disturbances of the breathing pattern occurring only, or at least primarily, during sleep. This can be thought of as a spectrum ranging from snoring (the sound made by partial upper airway obstruction during sleep), through hypopnea (a decrease in amplitude of airflow associated with an arousal and or oxygen desaturation), to apnea (complete cessation of airflow). Other disturbances of the breathing pattern include periodic breathing and central alveolar hypoventilation.

Sleep-disordered breathing should be considered in any patient with a history of snoring and daytime somnolence. In those with a reasonable number of hours of sleep, sleep-disordered breathing is the most common reason for daytime somnolence. Obstructive sleep apnea refers to repetitive episodes of upper airway obstruction during sleep and is recognized as cessation of airflow with continued movement of the chest and abdominal wall. Apnea generally produces both oxygen desaturation and arousal from sleep. Patients with apnea are described as loud and obnoxious snorers by bed partners. Most patients exhibit some level of increased daytime somnolence; a smaller number complain more of frequent awakenings and insomnia. Cardiopulmonary consequences may include hypertension, but arrhythmias are surprisingly uncommon despite severe hypoxemia in some patients. Severity of this condition as seen using polysomnography is often expressed as an apnea index (AI)—that is, the average number of apneas during an hour of sleep—or an apnea hypopnea index (AHI) or respiratory disturbance index (RDI)—that is, apneas and hypopneas per hour of sleep. An AI of less than 10 may be normal; a typical severely symptomatic patient might have an AI greater than 50.

Central sleep apnea alone is much less common than obstructive sleep apnea. The recording in central apnea reveals a complete cessation of both airflow and effort implying a failure in the regulation of breathing during sleep. This may be seen as part of a pattern of periodic breathing in which tidal volume rhythmically waxes and wanes. Central sleep apnea is often associated with cerebrovascular or

cardiac disease, such as Cheyne-Stokes respiration seen in congestive heart failure. Patients may be asymptomatic or may less frequently complain of difficulty maintaining sleep.

Most patients with sleep apnea are reported to be severe snorers and many are overweight or have crowding of the upper airway on physical examination. The disease is widely prevalent. In the study by Young et al [11], 4% of middle-aged men and 2% of women had an AHI greater than 5 and daytime somnolence. It remains difficult to diagnose sleep-disordered breathing without formal sleep polysomnography [12].

Treatment of sleep-disordered breathing could be directed at increasing respiratory drive or relieving upper airway obstruction. Pharmacologic efforts to increase respiratory drive with agents such as progesterone or theophylline have been disappointing. It is more important to avoid reducing ventilatory drive with any sedative or hypnotic drug. Alcohol has dramatic effects on worsening apnea and patients should be cautioned that even a small amount may have a significant effect [13].

Surgical approaches in the past have included tracheotomy, which is rapidly effective but rarely performed now that less-invasive forms of therapy are available. Surgical techniques to improve the upper airway, including uvulopalatoplasty, radiofrequency ablation of the tongue or soft palate, and injection to stiffen the palate, have been used but are most successful at eliminating the noise of snoring rather than the obstructive apneas. Currently, the most common approach to treatment is the use of a tight-fitting nasal or facial mask to deliver positive pressure as a “pneumatic splint” to keep the airway patent [14]. In patients who are obese weight loss is also recommended and can be successful.

A much less common disorder is central alveolar hypoventilation, in which chronic hypoventilation results in hypoxemia and hypercapnia, mostly or entirely during sleep, in patients with normal or near normal lung function. Most of these patients are morbidly obese but the condition can also be secondary to a neurologic disorder depressing the respiratory drive. Even in the morbidly obese, obstructive sleep apnea is far more common than central hypoventilation.

### *Sleep-disordered breathing in elderly patients*

Snoring is extremely common in elderly men and women. The significance of snoring in the absence of any other findings is unclear, but most studies suggest it is not an independent risk factor for other disease or morbidity.

Studies of prevalence of sleep apnea in the elderly population are difficult to interpret. The prevalence has been estimated at anywhere from 27% to 75% in elderly men. These data depend on the definition of sleep apnea (eg, AI, RDI, symptoms) which remains poorly standardized. In 1990, Hoch et al [15] looked at the prevalence of sleep apnea in healthy 60-, 70-, and 80-year-old men and women within 20% of ideal body weight. Using a criterion of AHI greater than or equal to 5, they found sleep apnea in 2.9% of 60-year-old participants, 33.3% of 70-year-old participants, and 39.5% of 80-year-old participants. More than one

third of 80-year-old participants desaturated to less than 85% with sleep, but this was mostly related to diseases other than sleep apnea. This study implies a striking increase in apnea with age, but the threshold for diagnosis is low and the clinical significance of these findings is unclear. Other studies do not support an increase in AI with increasing age [16]. It is possible that the prevalence of apnea declines with age because the most severely apneic patients have decreased life expectancy.

Using an AI of 10 as a cutoff another study reported a sleep apnea prevalence of 10% in independently living elderly persons, 21% in medical ward patients, and 26% in nursing home patients [17]. This higher prevalence of sleep apnea in sicker patients does not necessarily imply cause and effect.

The true significance of sleep-disordered breathing in elderly persons can only be defined based on morbidity (somnia, cognitive function) or mortality. In one study 27 healthy patients older than 64 years, unselected for sleep complaints, had overnight polysomnography [18]. Sleep apnea, defined as AI greater than 10, was measured in 10 patients. The apneic patients had a significantly shorter MSLT latency but no significant change in neuropsychologic testing. This sample may have been too small to find subtle cognitive changes. Berry et al [19] used polysomnography on 28 healthy elderly patients. There was a low rate of sleep-disordered breathing in this group. Follow-up at 1 year showed a correlation between hypertension, snoring, and irregular heartbeat with measures of sleep-disordered breathing, but no correlation with neuropsychologic measures. Phillips et al [20] studied healthy elderly subjects and found an AHI greater than 5 in 15%. There was no correlation in this sample of healthy individuals between sleep-disordered breathing indices and daytime functioning. The authors concluded that finding sleep-disordered breathing in otherwise healthy elderly subjects is not likely a cause for immediate concern.

Other studies have shown correlations between sleep apnea and cognitive deficits [21]. There appears to be a relationship between hypoxemia caused by sleep apnea and cognitive impairment, but this does not necessarily imply cause and effect. In elderly individuals both sleep-disordered breathing and dementia are common. It is at least likely that sleep-disordered breathing could worsen dementia.

It is difficult to prove that sleep-disordered breathing leads to increased mortality in elderly individuals. Bliwise et al [22] followed up 196 patients, with a mean age of 66 years, after polysomnography. Sleep apnea was defined as an AHI greater than 10. Mortality was increased by a factor of 2.7 for those with sleep apnea, but gender and body mass index were also independent variables, and the effect of age alone was greater than any other predictor. He et al [23] found a significantly lower survival rate for those patients with an AI greater than 20 if they were older than 50 years. Several other studies fail to show apnea as an independent risk factor for mortality. The presence of cardiovascular disease, hypertension, pulmonary disease, and the effect of age alone generally overshadow the effect of apnea [24,25].

## **Periodic limb movements**

Periodic limb movements are another common finding in the sleep of elderly patients. The disorder of periodic limb movements of sleep (PLMS) is defined as periodic episodes of repetitive and highly stereotypic limb movements during sleep. Movements may be associated with arousals and contribute to difficulty initiating or maintaining sleep or may be asymptomatic and found incidentally on polysomnography. The related syndrome of restless legs is characterized by unpleasant leg sensations, particularly at sleep onset, causing an almost irresistible desire to move the legs. Most patients with the restless legs syndrome (RLS) have PLMS on polysomnography, but many patients with PLMS are asymptomatic.

Several studies have shown a high prevalence of PLMS in elderly individuals, which may be as high as 60% in asymptomatic, healthy elderly individuals [17]. In one study of healthy elderly subjects within 20% of their ideal body weight, the prevalence of PLMS was given as 60% in 60-year-old subjects, 84% in 70-year-old subjects, and 92% in 80-year-old subjects. Overall, 54% had more than five limb movements per hour [15]. In a more recent population-based survey of 369 men and women aged 65 to 83 years the prevalence of symptoms of RLS was 9.8%, with a significantly higher percentage of women having symptoms than men (13.9% versus 6.1%) [26]. It would appear from these prevalence estimates that most elderly subjects with PLMS have no symptoms of RLS. In addition, most elderly subjects with PLMS have no sleep symptoms.

The significance of PLMS on polysomnography is therefore uncertain in elderly patients. In some patients with insomnia sleep is clearly fragmented by limb movements. Only movements associated with arousals or awakenings are likely to be significant, but arousals remain the most subtle finding on polysomnography. It can be difficult to determine, even in these patients, whether arousals and awakenings that accompany limb movements are the cause or consequence of insomnia.

Many medications and illnesses are associated with PLMS. Both tricyclic and serotonin synthesis and reuptake inhibitor antidepressants can worsen PLMS and the syndrome is found in association with peripheral neuropathies, many rheumatologic conditions, iron deficiency anemia, and chronic renal failure.

Treatment for PLMS can include dopamine agonists, benzodiazepines, and opiates. Benzodiazepines do not appear to reduce the number of movements, but rather the resulting arousals or awakenings [27]. Dopamine agonists (including DOPA/carbidopa, pramipexole, and others) may be more effective to reduce movements.

## **REM sleep-behavior disorder**

In REM sleep-behavior disorder the normal inhibition of motor tone during REM sleep is not present so that patients may act out their dreams [28]. Given the unpredictable content of dreams this can lead to extremely violent behavior.

Unlike usual sleep walking, which occurs mostly in slow wave (very deep) sleep, patients with REM sleep-behavior disorder often remember vivid dream content and can be fairly easily awakened. The disease is seen predominantly in older men, with a mean age of presentation in the mid-50s.

The true prevalence of this disease is unknown, but it is probably greatly underdiagnosed. In a survey of 1034 elderly subjects in Hong Kong 0.8% reported sleep-related injury and the prevalence of REM sleep-behavior disorder was estimated at 0.38% [29]. About 60% of cases appear idiopathic; in the remainder, neurologic lesions are identified. In some patients, there is an association with dementia [30].

The diagnosis is suggested by a classic history of violent behavior during sleep associated with remembered dreaming content. Sleep studies may confirm periods of REM without muscle atonia. Most patients should have an imaging study of the brain to rule out underlying pathology. Treatment is successful in about 90% of patients with clonazepam administered at bedtime.

## References

- [1] Rechtschaffen A, Kales A. A manual of standardized terminology, techniques and scoring system for the sleep stages of human subjects. Los Angeles: Brain Research Institute, University of California; 1968.
- [2] Webb WB, Dreblow LM. A modified method for scoring slow wave sleep of older subjects. *Sleep* 1982;5:195–9.
- [3] Whitney CW, Enright PL, Newman AB, et al. Correlates of daytime sleepiness in 4578 elderly persons: the Cardiovascular Health Study. *Sleep* 1998;21:27–36.
- [4] Straand J, Rokstad K. General practitioners' prescribing patterns of benzodiazepine hypnotics: are elderly patients at particular risk for overprescribing? A report from the More and Romsdal Prescription Study. *Scand J Prim Health Care* 1997;15:16–21.
- [5] Englert S, Linden M. Differences in self-reported sleep complaints in elderly persons living in the community who do or do not take sleep medication. *J Clin Psychiatr* 1998;59:137–44.
- [6] Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. *Sleep* 1987;10:45–56.
- [7] Rubinstein ML, Rothenberg SA, Maheswaran S, et al. Modified sleep restriction therapy in middle-aged and elderly chronic insomniacs. *Sleep Res* 1990;19:276.
- [8] Monk TH, Buysse DJ, Carrier J, et al. Effects of afternoon “siesta” naps on sleep, alertness, performance, and circadian rhythms in the elderly. *Sleep* 2001;24:680–7.
- [9] Bursztyrn M, Ginsberg G, Hammerman-Rozenberg R, Stessman J. The siesta in the elderly: risk factor for mortality? *Arch Int Med* 1999;159:1582–9.
- [10] Baskett JJ, Wood PC, Broad JB, et al. Melatonin in older people with age-related sleep maintenance problems: a comparison with age matched normal sleepers. *Sleep* 2001;24:418–24.
- [11] Young T, Palta M, Dempsey J, et al. The occurrence of sleep-disordered breathing among middle-aged adults. *N Engl J Med* 1993;328:1230–5.
- [12] Chalfin DB, Feinsilver SH, Fein AM. Neural network analysis for the diagnosis of sleep disordered breathing. *Am Rev Respir Dis* 1992;145:A172.
- [13] Issa FG, Sullivan CE. Alcohol, snoring and sleep apnea. *J Neurol Neurosurg Psychiatry* 1982;45:353–9.
- [14] Sullivan CE, Issa FG, Berthon-Jones M, et al. Home treatment of obstructive sleep apnea with continuous positive airway pressure applied through a nose mask. *Bull Eur Physiopathol Respir* 1984;20:49–54.

- [15] Hoch CC, Reynolds III CF, Monk TH, et al. Comparison of sleep-disordered breathing among healthy elderly in the seventh, eighth, and ninth decade of life. *Sleep* 1990;13:502–11.
- [16] Ancoli-Israel S, Kripke DF, Klauber MR, et al. Sleep-disordered breathing in community-dwelling elderly. *Sleep* 1991;14:486–93.
- [17] Ancoli-Israel S. Epidemiology of sleep disorders. *Clin Geriatr Med* 1989;5:347–62.
- [18] Knight H, Millman RP, Gur RC, et al. Clinical significance of sleep apnea in the elderly. *Am Rev Respir Dis* 1987;136:845–50.
- [19] Berry DTR, Phillips BA, Cook YR, et al. Sleep disordered breathing in healthy aged persons: one year follow-up of daytime sequelae. *Sleep* 1989;12:211–5.
- [20] Phillips BA, Berry DTR, Schmitt FA, et al. Sleep disordered breathing in the elderly, clinically significant? *Chest* 1992;101:345–9.
- [21] Yesavage J, Bliwise D, Guilleminault C, et al. Preliminary communication: intellectual deficit and sleep-related respiratory disturbance in the elderly. *Sleep* 1985;8:30–3.
- [22] Bliwise DL, Bliwise NG, Partinen M, et al. Sleep apnea and mortality in an aged cohort. *Am J Public Health* 1988;78:544–7.
- [23] He J, Kryger MH, Zorick FJ, et al. Mortality and apnea index in obstructive sleep apnea. *Chest* 1988;94:9–14.
- [24] Ancoli-Israel S, Dripke DF, Klauber MR, et al. Morbidity, mortality and sleep-disordered breathing in community dwelling elderly. *Sleep* 1996;19:277–82.
- [25] Mant A, King M, Saunders NA, et al. Four-year follow-up of mortality and sleep-related breathing disturbance in non-demented seniors. *Sleep* 1995;18:433–8.
- [26] Rothdach AJ, Trenkwalder C, Habersack J, et al. Prevalence and risk factors of RLS in an elderly population: the MEMO study. *Neurology* 2000;54:1064–8.
- [27] Mitler MM, Browman CP, Menn SJ, et al. The treatment of restless legs syndrome with clonazepam: a prospective controlled study. *Can J Neurol Sci* 1986;13:245–7.
- [28] Schenck CH, Bundlie SR, Patterson AL, et al. Rapid eye movement sleep behavior disorder: a treatable disorder affecting older males. *JAMA* 1987;257:1786–9.
- [29] Chiu HFK, Wing YK, Lam LCW, et al. Sleep-related injury in the elderly—an epidemiologic study in Hong Kong. *Sleep* 2000;23:513–7.
- [30] Ferman TJ, Boeve BF, Smith GE, et al. REM sleep behavior disorder and dementia: cognitive differences when compared with Alzheimer's dementia. *Neurology* 1999;52:951–7.