1. Define: apnea, hypopnea, RDI, obstructive sleep apnea, central sleep apnea and upper airway resistance syndrome. BG

2. What are the criteria for mild, moderate and severe obstructive sleep apnea in adults? SW

3. What is a polysomnogram and how is it performed? What are the findings in obstructive sleep apnea? BG

4. What is a multiple sleep latency test? AL

**Multiple Sleep Latency and Maintenance of Wakefulness Tests**

The measurement of sleepiness and alertness remains controversial (ie, the multiple sleep latency test [MSLT] for objectively measuring sleepiness and the maintenance of wakefulness test [MWT] for measuring alertness).[140]

The MSLT may follow PSG. It is considered an objective measurement of excessive daytime sleepiness (EDS). The MSLT consists of 4-5 naps of 20-minute duration every 2 hours during the day. The latency to sleep onset for each nap is averaged to determine the daytime sleep latency. Normal daytime sleep latency is greater than 10-15 minutes. OSAHS is generally associated with latencies of less than 10 minutes. It is not uncommon for the MSLT to demonstrate profound daytime sleepiness in OSA patients; mean sleep latency cannot discriminate between patients with OSA and patients with narcolepsy.

Routine use of the MSLT in the evaluation of OSA has significantly decreased because sleep physicians generally treat OSA on basis of the subjective symptoms reported by the patient. The MSLT is generally used to confirm the diagnosis of narcolepsy in patients in whom narcolepsy is a consideration. As opposed to people without narcolepsy, narcoleptic patients have rapid eye movement sleep on at least 2 of the 4-5 naps during the day.

Whether the MWT is a good enough test to measure treatment efficacy is debated. The low correlation between self-reported sleepiness, as typically measured by the Epworth Sleepiness Score (ESS), and objective measures of sleepiness, as measured by the MSLT, continues to present a
problem to clinicians and researchers in the determination of how to use these disparate measures in clinical practice and in research.

5. Is there a role for a home sleep study? SW

6. Describe the normal stages of sleep, normal ratios/duration of each stage and alterations seen in central and obstructive sleep apnea. AL

**Young Adult Normal Sleep Architecture:**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Arousal Threshold</th>
<th>EEG Pattern</th>
<th>Sleep Distribution %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Low</td>
<td>Theta waves</td>
<td>2-5</td>
</tr>
<tr>
<td>2</td>
<td>High</td>
<td>K complexes or sleep spindles</td>
<td>45-55</td>
</tr>
<tr>
<td>3</td>
<td>Higher</td>
<td>Delta waves</td>
<td>3-8</td>
</tr>
<tr>
<td>4</td>
<td>Highest</td>
<td>Delta waves</td>
<td>10-15</td>
</tr>
<tr>
<td>REM Sleep</td>
<td>Variable</td>
<td>Sawtooth waves</td>
<td>20-25</td>
</tr>
</tbody>
</table>

Sleep has been divided into two distinct states: nonrapid eye movement (NREM) and rapid eye movement (REM). These two states differ based on a constellation of physiologic parameters associated with each state. NREM sleep is characterized by a steady, slow heart rate and respiratory rate as well as low blood pressure. NREM is the “quiet” stage of sleep. REM sleep, in contrast, is characterized by bursts of rapid conjugate eye movement, increased autonomic activity, and dreaming. During REM sleep, there are large fluctuations in the blood pressure, heart rate, and respiratory rate. This stimulated state is combined with a decrease in muscular activity. REM can be defined as “a highly activated brain in a paralyzed body.”

The pattern of sleep in the young, healthy adult remains fairly constant from night to night. There appear to be minimal differences between the sleep of men and women in this age group. Sleep onset begins in stage 1 NREM, a short stage that lasts only a few minutes. The arousal threshold for this stage is very low; that is, a small noise can arouse someone from this
Stage 2 NREM occurs next. This stage is characterized by sleep spindles or K complexes on the electroencephalogram (EEG). This stage lasts anywhere from 10 to 25 minutes and is considered a deeper stage of sleep compared with stage 1. Stage 3 NREM sleep begins with high-voltage slow-wave activity being seen on the EEG. This is a short stage, lasting only a few minutes, and then stage 4 NREM sleep begins and usually lasts 20 to 40 minutes. The combination of stage 3 and 4 NREM sleep, which have similar EEG activity, make up deep sleep, or delta sleep. Finally, sleep begins to lighten, and stage 2 is entered, followed by either stage 1 or REM sleep. The initial REM sleep period is short, often only a few minutes, but as the night progresses, REM sleep time increases. In the young adult, NREM sleep occupies about 80% of the night, and REM occupies the other 20% (Table 46.1).

Infants/Children: higher % of REM sleep and stage 4 sleep
Elderly: same % REM sleep as adult, stage 4 NREM sleep diminishes dramatically

7. What are the symptoms of adult sleep apnea? Tell us about the Epworth Sleepiness Scale. BG

8. Discuss physical exam findings in obstructive sleep apnea. What is a Mueller’s maneuver? SW

9. Describe the Fujita classification of obstructive sleep apnea. Are there other classifications systems? CB


11. What are the complications of untreated sleep apnea? AL

Consequences of Untreated Obstructive Sleep Apnea

A number of negative health effects have been attributed to untreated OSA including increased mortality, an increase in cardiovascular disease, and neurocognitive difficulties. In a retrospective study, He and colleagues found that untreated OSA patients with an apnea index (AI) greater than 20 had a statistically significant increase in mortality com-
pared with patients with an apnea index less than 20.20 He also found that untreated patients with an AI greater than 20 had a 63% probability of surviving 8 years compared with 96% in those with an AI less than 20.20 Additionally, untreated OSA is reported to increase the risk of fatal and nonfatal motor vehicle accidents by 2.5-fold.21 A significant proportion of the mortality and morbidity related to OSA occurs through its effect on the cardiovascular system, including hypertension, coronary heart disease, congestive heart failure, arrhythmias, sudden death, pulmonary hypertension, and stroke. Untreated moderate and severe OSA has been reported to result in a threefold increase in fatal and nonfatal cardiovascular events, when compared with both healthy men without OSA and men with CPAP-treated OSA.22 Treatment of OSA with CPAP has also been reported to lower blood pressure by 10 mm Hg.23

Untreated OSA has been demonstrated to be an independent risk factor for insulin resistance.24 Recently, it has been suggested that OSA may contribute to the development of diabetes and metabolic syndrome, the term used to describe the commonly occurring conditions of obesity, insulin resistance, hypertension, and dyslipidemia. However, further research is needed to determine whether there is an independent link between OSA and metabolic abnormalities.

The prevalence of gastroesophageal reflux disease (GERD) in OSA patients is significantly higher than in the general population.25-27 Even though these disorders commonly occur together, no temporal or causal relationship has ever been demonstrated between the two. This may reflect the fact that they share similar risk factors. Treatment of OSA with CPAP has been demonstrated to decrease the occurrence of GERD.28

Aside from the obvious physical effects of OSA, such as excessive daytime sleepiness and impaired mood, neurocognitive deficits have also been associated with OSA. Untreated OSA has been documented to cause problems with attention, working memory, and executive function, all of which are improved with CPAP treatment.29 Bed partner dissatisfaction is also a common complaint among OSA patients and treatment has been shown to improve the quality of life in both the treated individuals and their bed partners.30 Thus the benefits of treating OSA are substantial and well documented.

12. Discuss non-surgical management of obstructive sleep apnea. What is the efficacy of each?--give us data. CB

13. Discuss the surgical management of obstructive sleep apnea. Describe the Powell and Riley protocol. Give us some data on the efficacy of each of these procedures. TT

14. List UPPP complications and how to prevent them. TT

15. What is the proper post-operative management of obstructive sleep apnea? TT
16. What are the non-surgical and surgical treatments for snoring? Will insurance cover any treatments? HH

17. Compare the results of radiofrequency ablation of the palate with injection snoreplasty in the treatment of snoring. HH