

# Introduction to Sleep and Polysomnography

## Overview of Sleep Stages and Cycles

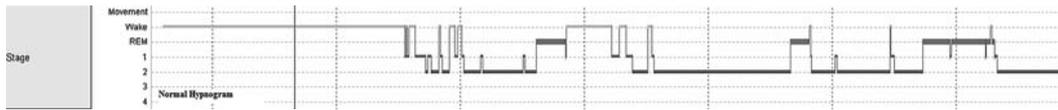
The monitoring of sleep is complex and requires a distinct skill set including a detailed knowledge of EEG, respiratory monitoring, and EKG. Expertise in only one of these areas does not confer the ability to accurately interpret the polysomnogram.

Sleep is not homogeneous and is characterized by sleep stages based on electroencephalographic (EEG) or electrical brain wave activity, electro-oculographic (EOG) or eye movements, and electromyographic (EMG) or muscle electrical activity.<sup>1-3</sup> The basic terminology and methods involved in monitoring each of these types of activity will be discussed below. Sleep is composed of nonrapid eye movement (NREM) and rapid eye movement (REM) sleep. NREM sleep is further divided into stages 1, 2, and 3. Stages 3 and 4 sleep were recently combined into stage 3 sleep. Stages 1 and 2 are called light sleep and stage 3 is called deep or slow-wave sleep. There are usually four or five cycles of sleep, each composed of a segment of NREM sleep followed by REM sleep. Periods of wake may also interrupt sleep during the night. As the night progresses, the length of REM sleep in each cycle usually increases. The hypnogram (Fig. 11-1) is a convenient method of graphically displaying the organization of sleep during the night. Each stage of sleep is characterized by a level on the vertical axis of the graph with time of night on the horizontal axis. REM sleep is often highlighted by a dark bar.

Sleep monitoring was traditionally done by polygraph recording using ink-writing pens which produced tracings on paper. It was convenient to divide the night into epochs of time that correspond to the length of each paper page. The usual paper speed for sleep recording is 10 mm/s; a 30-cm page corresponds to 30 s. Each segment of time represented by one page is called an epoch; sleep is staged in epochs. Today most sleep recording is performed digitally, but the convention of scoring sleep in 30-s epochs or windows is still the standard. If there is a shift in sleep stage during a given epoch, the stage present for the majority of the time names the epoch. When the tracings used to stage sleep are obscured by artifact for more than one-half of an epoch, it is scored as movement time (MT). When an epoch of what would otherwise be considered an MT is surrounded by epochs of wake, the epoch is also scored as wake. Some sleep centers consider MT to be wake and do not tabulate it separately.

## Sleep Architecture Definitions

The term sleep architecture describes the structure of sleep. Common terms used in sleep monitoring are listed in Table 11-1. The total monitoring time or total recording time (TRT) is also called total bedtime (TBT). This is the time duration from lights out (start of recording) to lights on (termination of recording). The total amount of sleep stages 1, 2, 3, REM, and MT



**FIGURE 11-1.** Typical hypnogram: The various stages of sleep are represented by levels on the vertical axis; time of night is shown on the horizontal axis. In this patient, there were several sleep cycles, each composed of a segment of NREM sleep followed by REM sleep. The length of REM sleep increased toward morning.

**TABLE 11-1** Sleep Architecture Definitions

- Lights out—start of sleep recording
- Lights on—end of sleep recording
- TBT (total bedtime)—time from lights out to lights on
- TST (total sleep time) = minutes of stages 1, 2, 3, and REM
- WASO (wake after sleep onset)—minutes of wake after first sleep but before the final awakening
- SPT (sleep period time) = TST + WASO
- Sleep latency—time from lights out until the first epoch of sleep
- REM latency—time from first epoch of sleep to the first epoch of REM sleep
- Sleep efficiency— $(TST \times 100)/TBT$
- Stages 1, 2, 3, and REM as % TST—percentage of TST occupied by each sleep stage
- Stages 1, 2, 3, and REM, WASO as % SPT—percentage of SPT occupied by sleep stages and WASO
- Arousal index

is termed the *total sleep time* (TST). The time from the first sleep until the final awakening is called the *sleep period time* (SPT). SPT encompasses all sleep as well as periods of wake after sleep onset (WASO) and before the final awakening. This wake time is termed the “wake after sleep onset” Therefore,  $SPT = TST + WASO$ . The time from the start of sleep monitoring (or lights out) until the first epoch of sleep is called the *sleep latency*. The time from the first epoch of sleep until the first REM sleep is called the *REM latency*. It is useful not only to determine the total minutes of each sleep stage but also to characterize the relative proportion of time spent in each sleep stage. One can characterize stages 1 to 3 and REM as a percentage of TST (%TST). Another method is to characterize the sleep stages and WASO as a percentage of the SPT (%SPT). Sleep efficiency (in percent) is usually defined as either the  $TST \times 100/SPT$  or  $TST \times 100/TBT$ .

The normal range of the percentage of sleep spent in each sleep stage varies with age<sup>2,3</sup> and is impacted by sleep disorders (Table 11-2). In adults, there is a decrease in stage 3 sleep with increasing age, while the amount of REM sleep remains fairly constant. The amounts of stage 1 sleep and WASO also increase with age. In patients with severe obstructive sleep apnea (OSA), there is often no stage 3 sleep and a reduced amount of REM sleep. Chronic insomnia (difficulty initiating or maintaining sleep) is characterized by a long sleep latency and increased WASO. The amount of stages 3 and REM sleep is commonly decreased as well. The REM latency is also affected by sleep disorders and medications. A short REM latency (usually <70 min) is noted in some cases of sleep apnea, depression, narcolepsy, prior REM sleep deprivation, and the withdrawal of REM suppressant medications. An increased REM latency can be seen with REM suppressants (ethanol and many antidepressants), an unfamiliar or uncomfortable sleep environment, sleep apnea, and any process that disturbs the sleep quality.

**TABLE 11-2** Representative Changes in Sleep Architecture

	20-Year-Old	60-Year-Old	Severe Sleep Apnea <sup>a</sup>
WASO% SPT	5	15	20
Stage 1% SPT	5	5	10
Stage 2% SPT	50	55	60
Stage 3% SPT	20	5	0
REM% SPT	25	20	10

<sup>a</sup>High interpatient variability.

## Introduction to Electroencephalographic Terminology and Monitoring

EEG activity is characterized by the frequency in cycles per second or hertz (Hz), amplitude (voltage), and the direction of major deflection (polarity). The classically described frequency ranges are delta (<4 Hz), theta (4 to 7 Hz), alpha (8 to 13 Hz), and beta (>13 Hz). Alpha waves (8 to 13 Hz) are commonly noted when the patient is in an awake, but relaxed, state with the eyes closed. They are best recorded over the occiput and are attenuated when the eyes are open. Bursts of alpha waves also are seen during brief awakenings from sleep—called arousals. Alpha activity can also be seen during REM sleep. Alpha activity is prominent during drowsy eyes-closed wakefulness. This activity decreases with the onset of stage 1 sleep. Near the transition from stage 1 to stage 2 sleep, vertex *sharp waves*—high-amplitude negative waves (upward deflection on EEG tracings) with a short duration—occur. They are more prominent in central than in occipital EEG tracings. A sharp wave is defined as a deflection of 70 to 200 ms in duration (Table 11-3).

### QUESTION 11.1: In standard sleep staging, delta sleep is defined as...

- <4 Hz
- <2 Hz
- 1 to 3 Hz
- <1 Hz

**ANSWER:** b.

Sleep spindles are oscillations of 12 to 14 Hz with a duration of 0.5 to 1.5 s. They are characteristic of stage 2 sleep. They may persist into stage 3 but usually do not occur in stage REM. The K complex is a high-amplitude, biphasic wave of at least 0.5-s duration. As classically defined, a K complex consists of an initial sharp, negative voltage (by convention, an upward deflection) followed by a positive-deflection (down) slow wave. Spindles frequently are superimposed on K complexes. Sharp waves differ from K complexes in that they are narrower, not biphasic, and usually of lower amplitude.

As sleep deepens, slow (delta) waves appear. These are high-amplitude, broad waves. In contrast to the EEG definition of delta activity as less than 4 Hz, delta slow-wave activity is defined for sleep staging purposes as waves slower than 2 Hz (longer than 0.5-s duration) with a peak-to-peak amplitude of greater than 75  $\mu$ V. The amount of slow-wave activity *as measured in the central EEG derivations* is used to determine if stage 3 is present<sup>1</sup> (see below). Because a K complex resembles slow-wave activity, differentiating the two is sometimes difficult. However, by definition, a K complex should stand out (be distinct) from the lower-amplitude, background EEG activity. Therefore, a continuous series of high-voltage slow (HVS) waves would not be considered to be a series of K complexes.

**TABLE 11-3** Standard Sensitivity and Filter Settings

	Sensitivity	Low Filter	High Filter
EEG	50 $\mu$ V = 1 cm; 100 $\mu$ V = 1 channel width	0.3 <sup>a</sup>	35 <sup>a</sup>
EOG	50 $\mu$ V = 1 cm; 100 $\mu$ V = 1 channel width	0.3	35
EMG	50 $\mu$ V = 1 cm; 100 $\mu$ V = 1 channel width	10	100
EKG		0.1	35
Airflow (thermistor)	Variable	0.1	15
Chest	Variable	0.1	15
Abdomen	Variable	0.1	15
SaO <sub>2</sub> (%)	1 Volt = 0–100 or 50%–100%	DC	15
Nasal pressure machine flow	Variable	DC or AC with low filter setting of 0.01	15 100 (to see snoring)

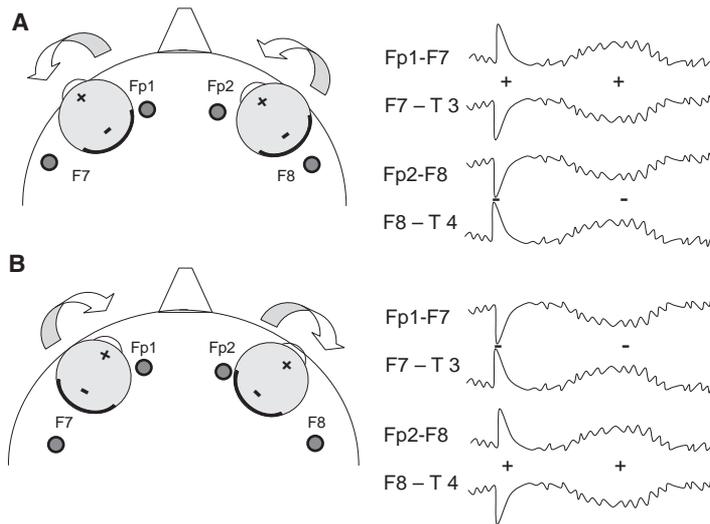
<sup>a</sup>Note that these filter settings are different from traditional EEG monitoring settings.

Sawtooth waves (see Fig. 11-10) are notched-jagged waves of frequency in the theta range (3 to 7 Hz) that may be present during REM sleep. Although they are not part of the criteria for scoring REM sleep, their presence is a clue that REM sleep is present.

## Eye Movement Recording

The main purpose of recording eye movements is to identify REM sleep. EOG (eye movement) electrodes typically are placed at the outer corners of the eyes—at the right outer canthus (ROC) and the left outer canthus (LOC). In a common approach, two eye channels are recorded and the eye electrodes are referenced to the opposite mastoid (ROC-A1 and LOC-A2). However, some sleep centers use the same mastoid electrode as a reference (ROC-A1 and LOC-A1). To detect vertical as well as horizontal eye movements, one electrode is placed slightly above and one slightly below the eyes.<sup>4,5</sup>

Recording of eye movements is possible because a potential difference exists across the eyeball: front positive (+), back negative (–). Eye movements are detected by EOG recording of voltage changes. When the eyes move toward an electrode, a positive voltage is recorded (Fig. 11-2). By standard convention, polygraphs are calibrated so that a negative voltage causes an upward pen deflection (negative polarity up). Thus, eye movement toward an electrode results in a downward deflection.<sup>4,7</sup> Note that movement of the eyes is usually conjugate, with both the eyes moving toward one eye electrode and away from the other. If the eye channels are calibrated with the same polarity settings, eye movements produce *out-of-phase deflections* in the two eye tracings (e.g., one up and one down). Figure 11-3 shows the recorded results of eye movements to the right and left (assuming both amplifier channels have negative polarity up). The same approach can be used to understand the tracings resulting from vertical eye movements. Because ROC is positioned above the eyes (and LOC below), upward eye movements are toward ROC and away from LOC. Thus, upward eye movement results in a downward deflection in the ROC tracing and an upward deflection in the LOC tracing.



**FIGURE 11-2.** The effects of conjugate eye movements on deflections in tracings from ROC (right outer canthus) and LOC (left outer canthus) are illustrated. The front of the globe is positive with respect to the back. **A.** Leftward eye movements. **B.** Rightward eye movements.

There are two common patterns of eye movements (Fig. 11-3). Slow eye movements (SEMs), also called slow-rolling eye movements, are pendular oscillating movements that are seen in drowsy (eyes closed) wakefulness and stage 1 sleep. By stage 2 sleep, SEMs usually have disappeared. REMs are sharper (more narrow deflections), which are typical of eyes-open wake and REM sleep.

In the two-tracing method of eye movement recording, large-amplitude EEG activity or the artifact reflected in the EOG tracings usually causes *in-phase deflections*.

## Electromyographic Recording

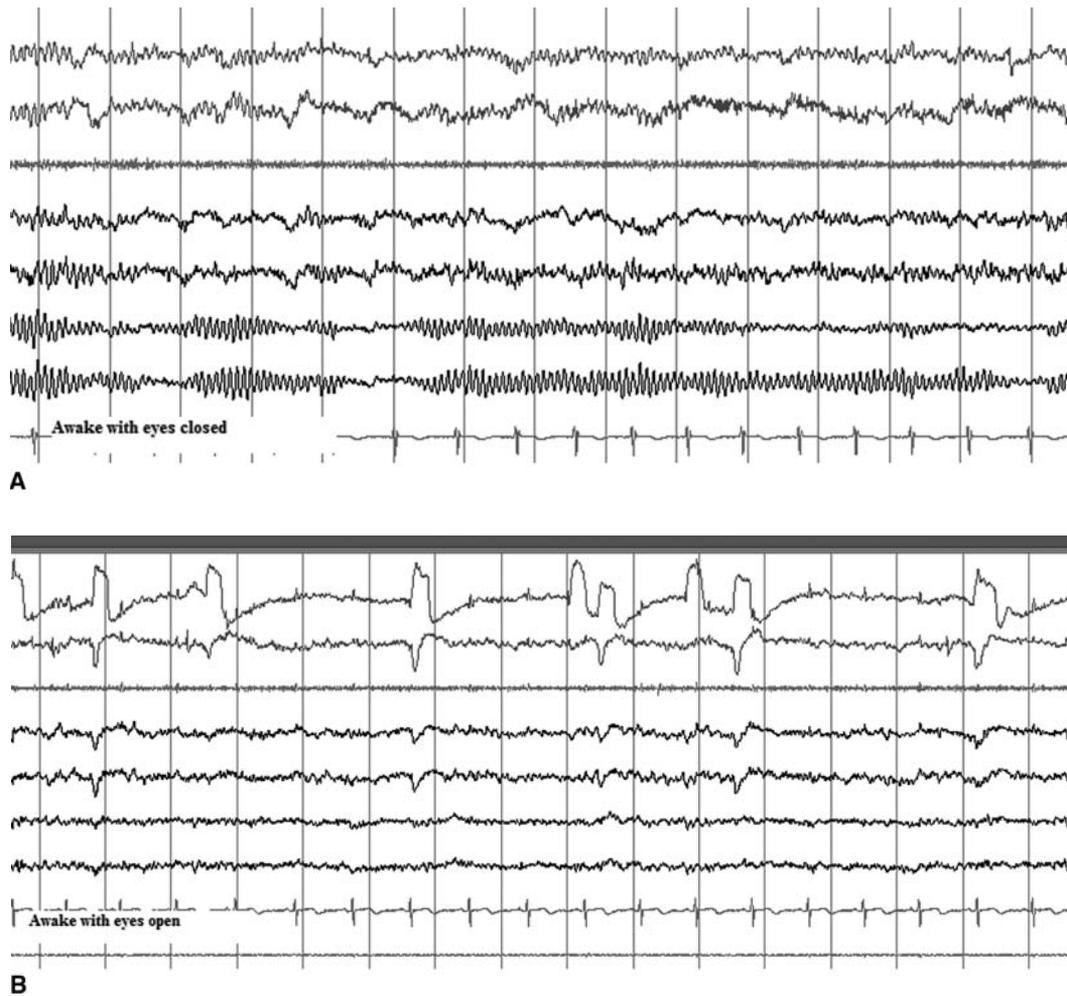
Usually, three EMG leads are placed in the mental and submental areas. The voltage between two of these three is monitored (e.g., EMG1–EMG3). If either of these leads fails, the third lead can be substituted. The gain of the chin EMG is adjusted so that some activity is noted during wakefulness. The chin EMG is an essential element only for identifying stage REM sleep. In stage REM, the chin EMG is relatively reduced—the amplitude is equal to or lower than the lowest EMG amplitude in NREM sleep. If the chin EMG gain is adjusted high enough to show some activity in NREM sleep, a drop in activity is often seen on transition to REM sleep. The chin EMG may also reach the REM level long before the onset of REMs or an EEG meeting criterion for stage REM. Depending on the gain, a reduction in the chin EMG amplitude from wakefulness to sleep and often a further reduction on transition from stage 1 to 3 may be seen. However, a reduction in the chin EMG is not required for stages 2 to 3. The reduction in the EMG amplitude during REM sleep is a reflection of the generalized skeletal-muscle hypotonia present in this sleep stage. Phasic brief EMG bursts still may be seen during REM sleep. The combination of REMs, a relatively reduced chin EMG, and a low-voltage, mixed-frequency EEG is consistent with stage REM.

## Sleep Stage Characteristics

The basic rules for sleep staging are summarized in Table 11-4. Note that some characteristics are required (bold) and some are helpful but not required. The typical patterns associated with each sleep stage are discussed below.

### Stage Wake

During eyes-open wake, the EEG is characterized by high-frequency, low-voltage activity. The EOG tracings typically show REMs, and the chin EMG activity is relatively high allowing



**FIGURE 11-3.** Typical patterns of eye movements are illustrated. Tracing **A**: SEMs are pendular and common in drowsy wake and stage 1 sleep. Tracing **B**: REMs are sharper (shorter duration) and seen in eyes-open wake or REM sleep.

differentiation from stage REM sleep. During eyes-closed drowsy wake, the EEG is characterized by prominent alpha activity (>50% of the epoch). Both slow, scanning and more rapid irregular eye movements are usually present. The level of muscle tone is usually relatively high (Fig. 11-4).

### Stage 1

The stage 1 EEG is characterized by low-voltage, mixed-frequency activity (4 to 7 Hz). Stage 1 is scored when less than 50% of an epoch contains alpha waves and criteria for deeper stages of sleep are not met (Fig. 11-5). Slow-rolling eye movements often are present in the eye movement tracings, and the level of muscle tone (EMG) is equal or diminished compared to that in the awake state. Some patients do not exhibit prominent alpha activity, making the detection of sleep onset difficult. The ability of a patient to produce alpha waves can be determined from biocalibrations at the start of the study. The patient is asked to lie quietly with eyes open and then with the eyes closed. Alpha activity usually appears with eye closure. When patients do not produce significant alpha activity, differentiating wakefulness from stage 1

**TABLE 11-4** Summary of Sleep Stage Characteristics

Stage	Characteristics <sup>a,b</sup>		
	EEG	EOG	EMG
Wake (eyes open)	Low-voltage, high-frequency, attenuated alpha activity	Eye blinks, REMs	Relatively high
Wake (eyes closed)	Low-voltage, high-frequency > 50% alpha activity	Slow-rolling eye movements	Relatively high
Stage 1	Low-amplitude, mixed-frequency <b>&lt;50% alpha activity No spindles, K complexes</b> Sharp waves near transition to stage 2	Slow-rolling eye movements	May be lower than wake
Stage 2	<b>At least one sleep spindle or K complex &lt;20% slow-wave activity<sup>b</sup></b>		May be lower than wake
Stage 3	>20% slow-wave activity	<sup>c</sup>	Usually low
Stage REM	<b>Low-voltage, mixed-frequency</b> Sawtooth waves—may be present	<b>Episodic REMs</b>	<b>Relatively reduced</b> (equal or lower than the lowest in NREM)

<sup>a</sup>Required characteristics in bold.

<sup>b</sup>Slow-wave activity, frequency < 2 Hz; peak-to-peak amplitude > 75  $\mu$ V; >50% means slow-wave activity present in more than 50% of the epoch; REMs, rapid eye movements.

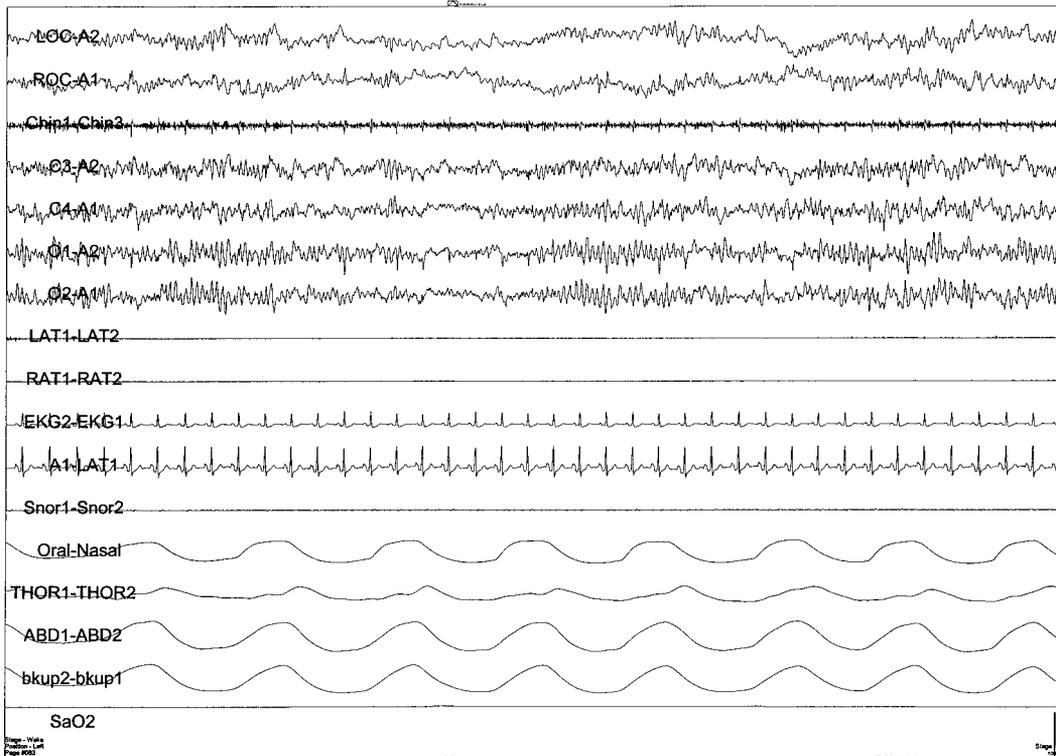
<sup>c</sup>Slow waves usually seen in EOG tracings.

sleep can be difficult. Several points are helpful. First, the presence of REMs in the absence of a reduced chin EMG usually means the patient is still awake. However, SEMs can be present during drowsy wake and stage 1 sleep. In this case, one must differentiate wake from stage 1 by the EEG. In wake, the EEG has considerable high-frequency activity. In stage 1, the EEG is mixed frequency with activity in the 4 to 7 Hz theta range. Note the slower (wider) EEG activity in Figure 11-5. Often, the easiest method to determine sleep onset in difficult cases is to find the first epoch of unequivocal sleep (usually stage 2) and work backward. The examiner can usually be confident of the point of sleep onset within one or two epochs.

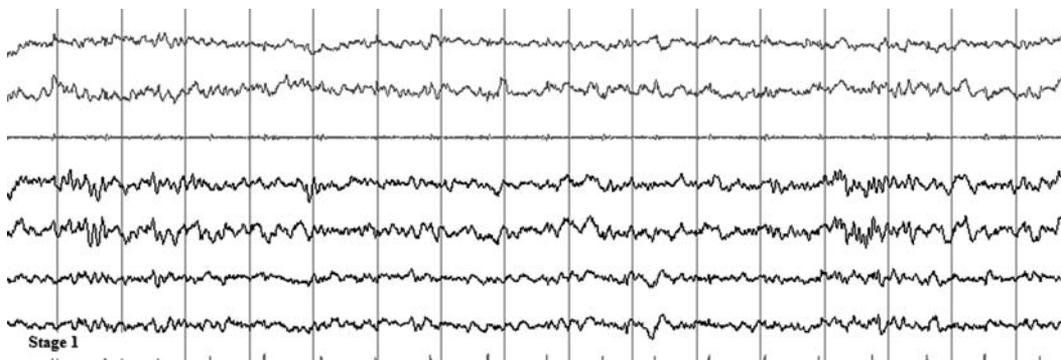
Vertex waves are common in stage 1 sleep and are defined by a sharp configuration maximal over the central derivations. Vertex waves should be easily distinguished from the background activity.

### Stage 2

Stage 2 sleep is characterized by the presence of one or more K complexes (Fig. 11-6) or sleep spindles (Fig. 11-7). To qualify as stage 2, an epoch also must contain less than 20% of slow (delta) wave EEG activity (<6 s of a 30-s epoch). Slow-wave activity is defined as waves with a frequency less than 2 Hz and a minimum peak-to-peak amplitude of more than 75  $\mu$ V. Stage 2 occupies the greatest proportion of the TST and accounts for roughly 40% to 50% of sleep.



**FIGURE 11-4.** Stage wake—eyes closed (drowsy). The EEG shows more alpha activity for more than 50% of the epoch, and the EOG tracing may show SEMs. The chin EMG is relatively high in amplitude. (From Geyer JD, Payne TA, Canny PR et al. *Atlas of Digital Polysomnography*. Philadelphia, PA: Lippincott Williams & Wilkins, 2000. (Ref. 6))

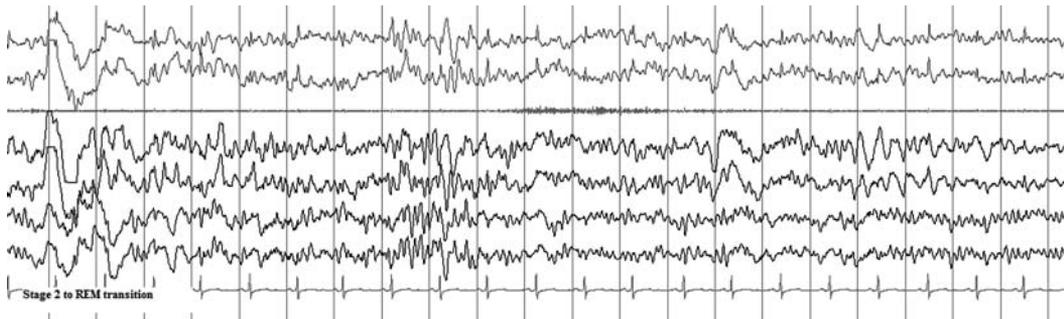


**FIGURE 11-5.** Stage 1 (30-s tracing). The EEG shows alpha activity for less than 50% of the epoch and has a low-voltage, mixed-frequency activity. Waves in the theta range are seen at (T). SEMs are usually present.

Stage 2 sleep ends with a sleep stage transition (to stage W, stage 3, and stage REM), an arousal, or a major body movement followed by SEMs and low-amplitude, mixed-frequency EEG.

### Stage 3 (formerly stages 3 and 4)

Stage 3 NREM sleep is called slow-wave, delta, or deep sleep. Stage 3 is scored when slow-wave activity (frequency < 2 Hz and amplitude > 75  $\mu$ V peak-to-peak) is present for greater than 20% of the epoch (Fig. 11-8). Spindles may be present in the EEG. Frequently, the high-voltage

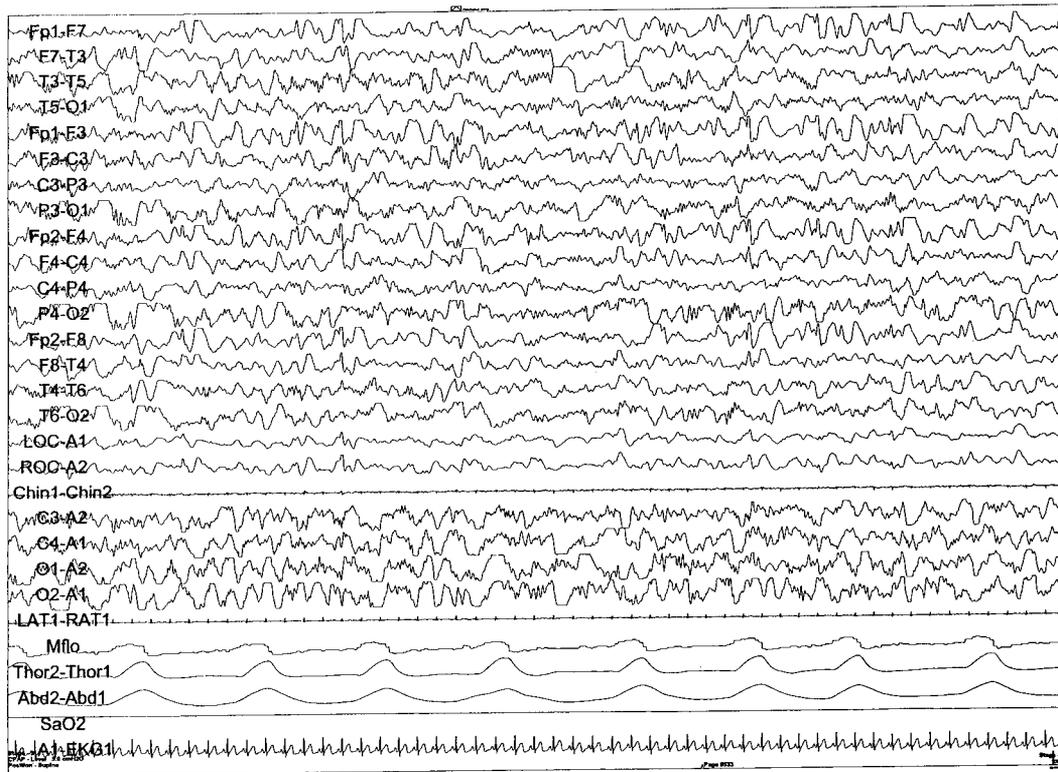


**FIGURE 11-6.** A 30-s tracing of stage 2 sleep is shown. The EEG shows a K complex that is transmitted to the eye channels (in-phase deflection).



**FIGURE 11-7.** A 30-s tracing of stage 2 sleep is shown. The EEG shows a sleep spindle, and the eye movement channels show an absence of SEMs.

EEG activity is transmitted to the eye leads. The EMG is often lower than during stages 1 and 2 sleep, but this is variable. In older patients, the slow-wave amplitude is lower, and the total amount of slow-wave sleep is reduced. The amplitude of the slow waves (and the amount of slow-wave sleep) is usually highest in the first sleep cycles. Typically, stage 3 occurs mostly in the early portions of the night. Several parasomnias (disorders associated with sleep) occur in stage 3 sleep and, therefore, can be predicted to occur in the early part of the night. These include somnambulism (sleep walking) and night terrors. In contrast, parasomnias occurring in REM sleep (e.g., nightmares) are more common in the early morning hours.



**FIGURE 11-8.** A 30-s tracing of stage 3 sleep is shown. There is a prominent slow-wave activity meeting voltage criteria ( $>75\mu\text{V}$  peak-to-peak) throughout the tracing. Note that slow-wave activity is also seen in the eye channels.

**QUESTION 11.2: True or False. Sleep spindles are seen only during stage 2 sleep.**

- True.
- False.

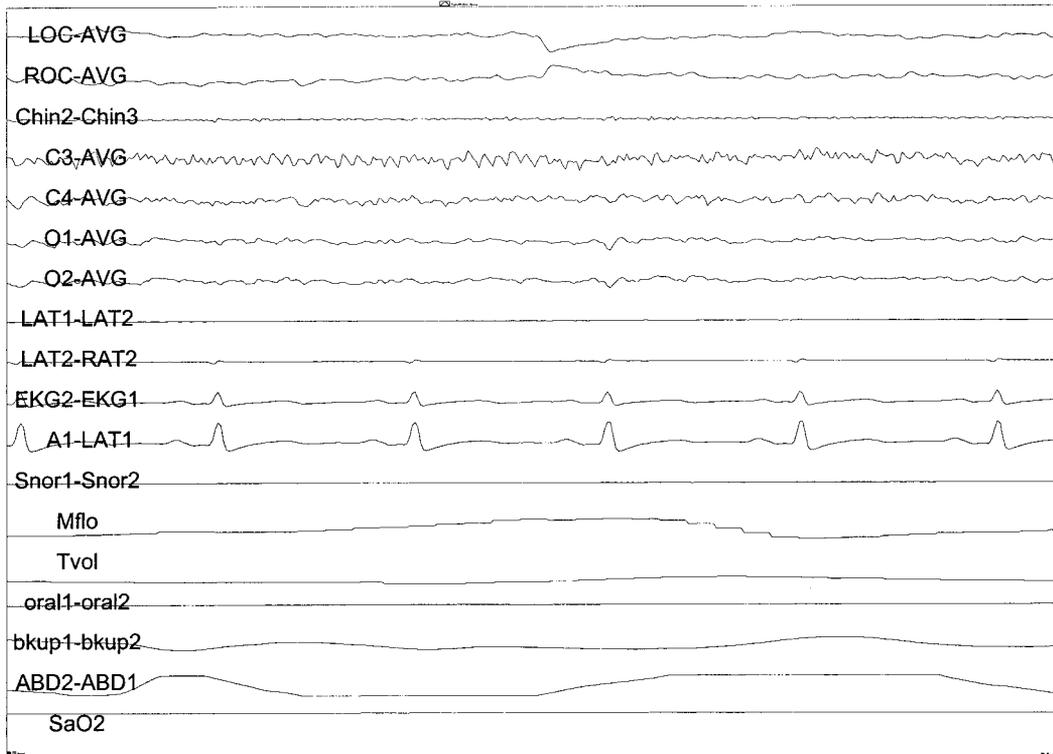
**ANSWER:** b.

**EXPLANATION:** Rare spindles may be seen during stage 3 sleep.

### Stage REM

Stage REM sleep is characterized by a low-voltage, mixed-frequency EEG, the presence of episodic REMs, and a relatively low-amplitude chin EMG. Sawtooth waves (Fig. 11-9) also may occur in the EEG. There usually are three to five episodes of REM sleep during the night, which tend to increase in length as the night progresses. The number of eye movements per unit time (REM density) also increases during the night. Not all epochs of REM sleep contain REMs. Epochs of sleep otherwise meeting the criteria for stage REM and contiguous with epochs of unequivocal stage REM (REMs present) are scored as stage REM (see Advanced Staging Rules). Bursts of alpha waves can occur during REM sleep, but the frequency is often 1 to 2 Hz slower than during wake.

Stage REM is associated with many unique, physiologic changes, such as widespread skeletal-muscle hypotonia and sleep-related erections. Skeletal-muscle hypotonia is a protective mechanism to prevent the acting out of dreams. In a pathologic state known as the REM behavior disorder, muscle tone is present, and body movements and even violent behavior can occur during REM sleep.



**FIGURE 11-9.** Stage REM. A 10-s tracing of stage REM is shown. The EEG is low-amplitude mixed-frequency. In this case, a sawtooth wave is noted in channel C3-AVG. The eye tracings show a rapid eye movement LOC and ROC. The chin EMG amplitude is low.

**QUESTION 11.3:** In which of the following stages of sleep can an alpha rhythm arise?

1. Stage wake, eyes closed.
  2. Stage 1 sleep.
  3. Stage 3 sleep.
  4. Stage REM sleep.
- a. 1.
  - b. 2 and 4.
  - c. 1, 2, and 4.
  - d. 1, 2, 3, and 4.

**ANSWER:** d.

### Arousals

Arousal from sleep denotes a transition from a state of sleep to wakefulness. Frequent arousals can cause daytime sleepiness by shortening the total amount of sleep. However, even if arousals are brief (1 to 5 s) with a rapid return to sleep, daytime sleepiness may result, although the TST is relatively normal.<sup>8</sup> Thus, the restorative function of sleep depends on continuity as well as duration. Many disorders that are associated with excessive daytime sleepiness are also associated with frequent, brief arousals. For example, patients with OSA frequently have arousals coincident with apnea/hypopnea termination. Therefore, determination of the frequency of arousals has become a standard part of the analysis of sleep architecture during sleep testing.

Movement arousals were defined in the Rechtschaffen and Kales (R&K) scoring manual<sup>1</sup> as an increase in EMG that is accompanied by a change in the pattern on any additional channel. For EEG channels, qualifying changes included a decrease in amplitude, paroxysmal high-voltage activity, or an increase in alpha activity. Subsequently, arousals were the object of considerable research, but the criteria used to define them were variable. A report from the Atlas Task Force of the American Academy of Sleep Medicine (formerly the American Sleep Disorders Association or ASDA) has become the standard definition.<sup>9</sup> According to the ASDA Task Force, an arousal should be scored in NREM sleep when there is “an abrupt shift in EEG frequency, which may include theta, alpha, and/or frequencies greater than 16 Hz, but not spindles,” of 3 s or longer duration. The 3-s duration was chosen for methodological reasons; shorter arousals may also have physiologic importance. To be scored as an arousal, the shift in EEG frequency must follow at least 10 continuous seconds of any stage of sleep. Arousals in NREM sleep may occur without a concurrent increase in the submental EMG amplitude. In REM sleep, however, the required EEG changes must be accompanied by a concurrent increase in EMG amplitude for an arousal to be scored. This extra requirement was added because spontaneous bursts of alpha rhythm are a fairly common occurrence in REM (but not NREM) sleep. Note that according to the above recommendations, increases in the chin EMG in the absence of EEG changes are not considered evidence of arousal in either NREM or REM sleep. Scoring of arousal during REM does however require a concurrent increase in submental EMG lasting at least 1 s. Similarly, sudden bursts of delta (slow-wave) activity in the absence of other changes do not qualify as evidence of arousal. Because cortical EEG changes must be present to meet the above definition, such events are also termed electrocortical arousals. Note that the above guidelines represent a consensus on events likely to be of physiologic significance. The committee recognized that other EEG phenomena, such as delta bursts, also can represent evidence of arousal in certain contexts.

The frequency of arousals usually is computed as the arousal index (number of arousals per hour of sleep). Relatively little data are available to define a normal range for the arousal index. Normal young adults studied after adaptation nights frequently have an arousal index of five per hour or less. In one study, however, normal subjects of variable ages had a mean arousal index of 21 per hour, and the arousal index was found to increase with age.<sup>10</sup> However, a respiratory arousal index (RAI) (arousals associated with respiratory events) as low as 10 per hour has been associated with daytime sleepiness in some individuals with the upper-airway resistance syndrome (UARS).<sup>11</sup> While some have argued that patients with this disorder really represent the mild end of the OSA syndrome, most would agree with the concept that respiratory arousals of sufficient frequency can cause daytime sleepiness in the absence of frank apnea and arterial oxygen desaturation.

**QUESTION 11.4: Which of the following frequency shifts does not accompany an arousal?**

- a. Theta
- b. Alpha
- c. Frequencies > 16 Hz
- d. Spindles

**ANSWER:** d.

### Advanced Sleep Staging Rules

Staging of REM sleep also requires special rules (REM rules) to define the beginning and the end of REM sleep. This is necessary because REMs are episodic, and the three indicators of stage REM (EEG, EOG, and EMG) may not change to (or from) the REM-like pattern simultaneously. Rechtschaffen and Kales recommend that any section of the record that is contiguous with unequivocal stage REM and displays a relatively low-voltage, mixed-frequency EEG be scored as stage REM regardless of whether REMs are present, providing the EMG is

at the stage REM level. To be REM-like, the EEG must not contain spindles, K complexes, or slow waves.

### Atypical Sleep Patterns

Four special cases in which sleep staging is made difficult by atypical EEG, EOG, and EMG patterns will be briefly mentioned. In alpha sleep, prominent alpha activity persists into NREM sleep. The presence of spindles, K complexes, and slow-wave activity allows sleep staging despite prominent alpha activity. Causes of the pattern include pain, psychiatric disorders, chronic pain syndromes, and any cause of nonrestorative sleep.<sup>12,13</sup> Patients taking benzodiazepines may have very prominent “pseudo-spindle” activity (14 to 16 Hz rather than the usual 12 to 14 Hz).<sup>14</sup> SEMs are usually absent by the time stable stage 2 sleep is present. However, patients on some serotonin reuptake inhibitors (fluoxetine and others) may have prominent slow and rapid eye movements during NREM sleep.<sup>15</sup> While a reduction in the chin EMG is required for staging REM sleep, patients with the REM sleep behavior disorder may have high chin activity during what otherwise appears to be REM sleep.<sup>16</sup>

### Sleep Staging in Infants and Children

Newborn term infants do not have the well-developed adult EEG patterns to allow staging according to Rechtschaffen and Kales rules. The following is a brief description of terminology and sleep staging for the newborn infant according to the state determination of Anders et al.<sup>17</sup> Infant sleep is divided into active sleep (corresponding to REM sleep), quiet sleep (corresponding to NREM sleep), and indeterminant sleep, which is often a transitional sleep stage. Behavioral observations are critical. Wakefulness is characterized by crying, quiet eyes open, and feeding. Sleep is often defined as sustained eye closure. Newborn infants typically have periods of sleep lasting 3 to 4 h interrupted by feeding, and total sleep in 24 h is usually 16 to 18 h. They have cycles of sleep with a 45- to 60-min periodicity with about 50% active sleep. In newborns, the presence of REM (active sleep) at sleep onset is the norm. In contrast, the adult sleep cycle is 90 to 100 min, REM occupies about 20% of sleep, and NREM sleep is noted at sleep onset.

The EEG patterns of newborn infants have been characterized as low-voltage irregular (LVI), tracé alternant (TA), HVS, and mixed (M) (Table 11-5). Eye movement monitoring

**TABLE 11-5 EEG Patterns Used in Infant Sleep Staging**

EEG Pattern	
Low-voltage irregular (LVI)	Low-voltage (14–35 $\mu$ V) <sup>a</sup> , little variation Theta (5–8 Hz) predominates Slow activity (1–5 Hz) also present
Tracé alternant (TA)	Bursts of high-voltage slow waves (0.5–3 Hz) with superimposition of rapid, low-voltage sharp waves (2–4 Hz) In between the high-voltage bursts (alternating with them) is the low-voltage, mixed-frequency activity of 4 to 8 s in duration
High-voltage slow (HVS)	Continuous moderately rhythmic medium- to high-voltage (50–150 $\mu$ V) slow waves (0.5–4 Hz)
Mixed (M)	High-voltage slow- and low-voltage polyrhythmic activity Voltage lower than in HVS

<sup>a</sup> $\mu$ V, microvolts.

is used as in adults. An epoch is considered to have high or low EMG if over one-half of the epoch shows the pattern. The characteristics of active sleep, quiet sleep, and indeterminant sleep are listed in Table 11-6. The change from active to quiet sleep is more likely to manifest indeterminant sleep. Nonnutritive sucking commonly continues into sleep.

As children mature, more typically adult EEG patterns begin to appear. Sleep spindles begin to appear at 2 months and are usually seen after 3 to 4 months of age.<sup>18</sup> K complexes usually begin to appear at 6 months of age and are fully developed by 2 years of age.<sup>19</sup> The point at which sleep staging follows adult rules is not well defined but usually is possible after 6 months of age. After about 3 months, the percentage of REM sleep starts to diminish and the intensity of body movements during active (REM) sleep begins to decrease. The pattern of NREM at sleep onset begins to emerge. However, the sleep cycle period does not reach the adult value of 90 to 100 min until adolescence.

Note that the sleep of premature infants is somewhat different from term infants (36 to 40 weeks gestation). In premature infants, quiet sleep usually shows a pattern of tracé discontinu.<sup>20</sup> This differs from TA as there is electrical quiescence (rather than a reduction in amplitude) between bursts of high-voltage activity. In addition, delta brushes (fast waves of 10 to 20 Hz) are superimposed on the delta waves. As the infant matures, delta brushes disappear and TA pattern replaces tracé discontinu.

**QUESTION 11.5: Which of the following statements regarding the development of stage 2 sleep is false?**

- Sleep spindles begin to appear at 2 months.
- Sleep spindles are usually present by 3 to 4 months of age.
- K complexes usually begin to appear at 6 months of age.
- K complexes are fully developed by 1 year of age.

**ANSWER:** d.

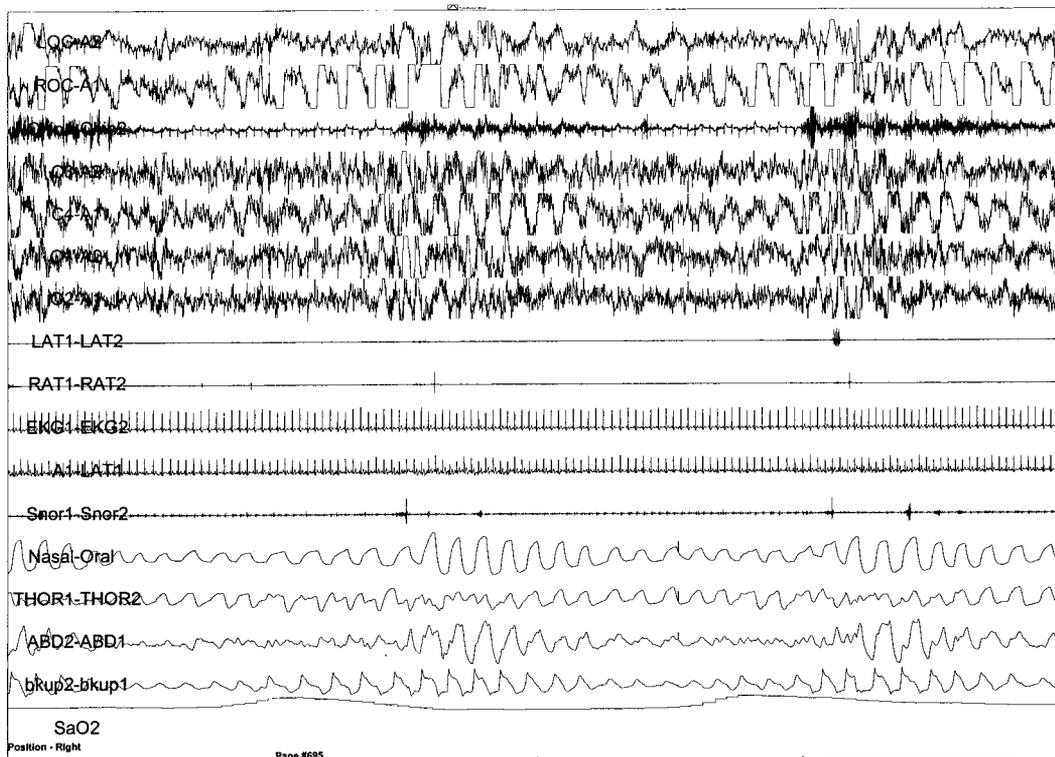
**TABLE 11-6** Characteristics of Active and Quiet Sleep

	Active Sleep	Quiet Sleep	Indeterminant
Behavioral	Eyes closed Facial movements: smiles, grimaces, frowns Burst of sucking Body: small digit or limb movements	Eyes closed No body movements except startles and phasic jerks Sucking may occur	Not meeting the criteria for active or quiet sleep
EEG	LVI, M, HVS (rarely)	HVS, TA, M	
EOG	REMs A few SEMs and a few dysconjugate movements may occur	No REMs	
EMG	Low	High	
Respiration	Irregular	Regular Postsigh pauses may occur	

## Respiratory Monitoring

The three major components of respiratory monitoring during sleep are airflow, respiratory effort, and arterial oxygen saturation<sup>21,22</sup> (Fig. 11-10). Many sleep centers also find using a snore sensor to be useful. For selected cases, exhaled or transcutaneous PCO<sub>2</sub> may also be monitored.

Traditionally, airflow at the nose and mouth was monitored by thermistors or thermocouples. These devices actually detect airflow by the change in the device temperature induced by a flow of air over the sensor. It is common to use a sensor in or near the nasal inlet and over the mouth (nasal-oral sensor) to detect both nasal and mouth breathing. While temperature sensing devices may accurately detect an absence of airflow (apnea), their signal is not proportional to flow and they have a slow response time.<sup>23</sup> Therefore, they do not accurately detect the decreases in airflow (hypopnea) or flattening of the airflow profile (airflow limitation). Exact measurement of airflow can be performed by the use of a pneumotachograph. This device can be placed in a mask over the nose and mouth. Airflow is determined by measuring the pressure drop across a linear resistance (usually a wire screen). However, pneumotachographs are rarely used in clinical diagnostic studies. Instead, monitoring of nasal pressure via a small cannula in the nose connected to a pressure transducer has gained in popularity for monitoring airflow.<sup>23,24</sup> The nasal pressure signal is actually proportional to the square of flow across the nasal inlet.<sup>25</sup> Thus, nasal pressure underestimates airflow at low flows and overestimates airflow at high flow rates. In the midrange of typical flow rates during sleep, the nasal pressure signal varies fairly linearly with flow. The nasal pressure versus flow relationship can



**FIGURE 11-10.** Obstructive hypopneas. There is a decrease in chest and abdominal movement during the hypopnea. There is paradoxical movement of the chest and abdomen.

be completely linearized by taking the square root of the nasal pressure signal.<sup>26</sup> However, in clinical practice, this is rarely performed. In addition to the changes in magnitude, changes in the shape of the nasal pressure signal can provide useful information. A flattened profile usually means that airflow limitation is present (constant or decreasing flow with an increasing driving pressure).<sup>23,24</sup> The unfiltered nasal pressure signal also can detect snoring if the frequency range of the amplifier is adequate. The only significant disadvantage of nasal pressure monitoring is that mouth breathing often may not be adequately detected (10% to 15% of patients). This can be easily handled by monitoring with both nasal pressure and a nasal-oral thermistor. An alternative approach to measuring flow is to use respiratory inductance plethysmography. The changes in the sum of the ribcage and abdomen band signals (RIPsum) can be used to estimate the changes in tidal volume.<sup>27,28</sup> During positive-pressure titration, an airflow signal from the flow-generating device is often recorded instead of using thermistors or nasal pressure. This flow signal originates from a pneumotachograph or other flow-measuring devices inside the flow generator.

In pediatric polysomnography, exhaled CO<sub>2</sub> is often monitored. Apnea usually causes an absence of fluctuations in this signal, although small expiratory puffs rich in CO<sub>2</sub> can sometimes be misleading.<sup>7,22</sup> The end-tidal PCO<sub>2</sub> (value at the end of exhalation) is an estimate of arterial PCO<sub>2</sub>. During long periods of hypoventilation that are common in children with sleep apnea, the end-tidal PCO<sub>2</sub> will be elevated (>45 mm Hg).<sup>22</sup>

Respiratory effort monitoring is necessary to classify respiratory events. A simple method of detecting respiratory effort is detecting movement of the chest and abdomen. This may be performed with belts attached to piezo-electric transducers, impedance monitoring, respiratory-inductance plethysmography (RIP), or monitoring of the esophageal pressure (reflecting changes in pleural or intrathoracic pressure). The surface EMG of the intercostal muscles or diaphragm can also be monitored to detect respiratory effort. Probably the most sensitive method for detecting effort is monitoring of the changes in esophageal pressure (reflecting changes in pleural pressure) associated with inspiratory effort.<sup>24</sup> This may be performed with esophageal balloons or small fluid-filled catheters. Piezo-electric bands detect movement of the chest and abdomen as the bands are stretched and the pull on the sensors generates a signal. However, the signal does not always accurately reflect the amount of chest/abdomen expansion. In RIP, changes in the inductance of coils in bands around the ribcage (RC) and abdomen (AB) during respiratory movement are translated into voltage signals. The inductance of each coil varies with changes in the area enclosed by the bands. In general, RIP belts are more accurate in estimating the amount of chest/abdominal movement than piezo-electric belts. The sum of the two signals [RIPsum = ( $a \times RC$ ) + ( $b \times AB$ )] can be calibrated by choosing appropriate constants:  $a$  and  $b$ . Changes in the RIPsum are estimates of changes in tidal volume.<sup>29</sup> During upper-airway narrowing or total occlusion, the chest and abdominal bands may move paradoxically. Of note, a change in body position may alter the ability of either piezo-electric belts or RIP bands to detect the chest/abdominal movement. Changes in body position may require adjusting the band placement or amplifier sensitivity. In addition, very obese patients may show little chest/abdominal wall movement despite considerable inspiratory effort. Thus, one must be cautious about making the diagnosis of central apnea solely on the basis of surface detection of inspiratory effort.<sup>30</sup>

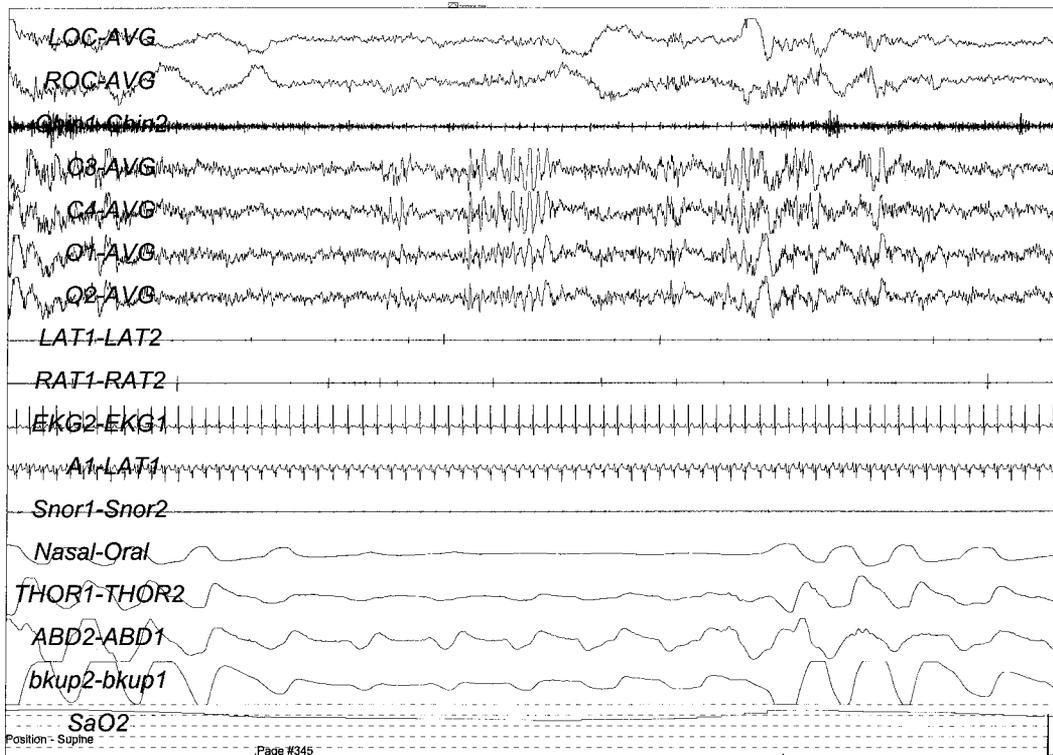
Arterial oxygen saturation (Sao<sub>2</sub>) is measured during sleep studies using pulse oximetry (finger or ear probes). This is often denoted as SpO<sub>2</sub> to specify the method of Sao<sub>2</sub> determination. A desaturation is defined as a decrease in Sao<sub>2</sub> of 4% or more from baseline. Note that the nadir in Sao<sub>2</sub> commonly follows apnea (hypopnea) termination by approximately 6 to 8 s (longer in severe desaturations). This delay is secondary to circulation time and instrumental delay (the oximeter averages over several cycles before producing a reading). Various measures have been applied to assess the severity of desaturation, including computing the number of desaturations, the average minimum Sao<sub>2</sub> of desaturations, the time below 80%, 85%, and

90%, as well as the mean  $\text{SaO}_2$  and the minimum saturation during NREM and REM sleep. Oximeters may vary considerably in the number of desaturations they detect and their ability to discard movement artifact. Using long averaging times may dramatically impair the detection of desaturations.

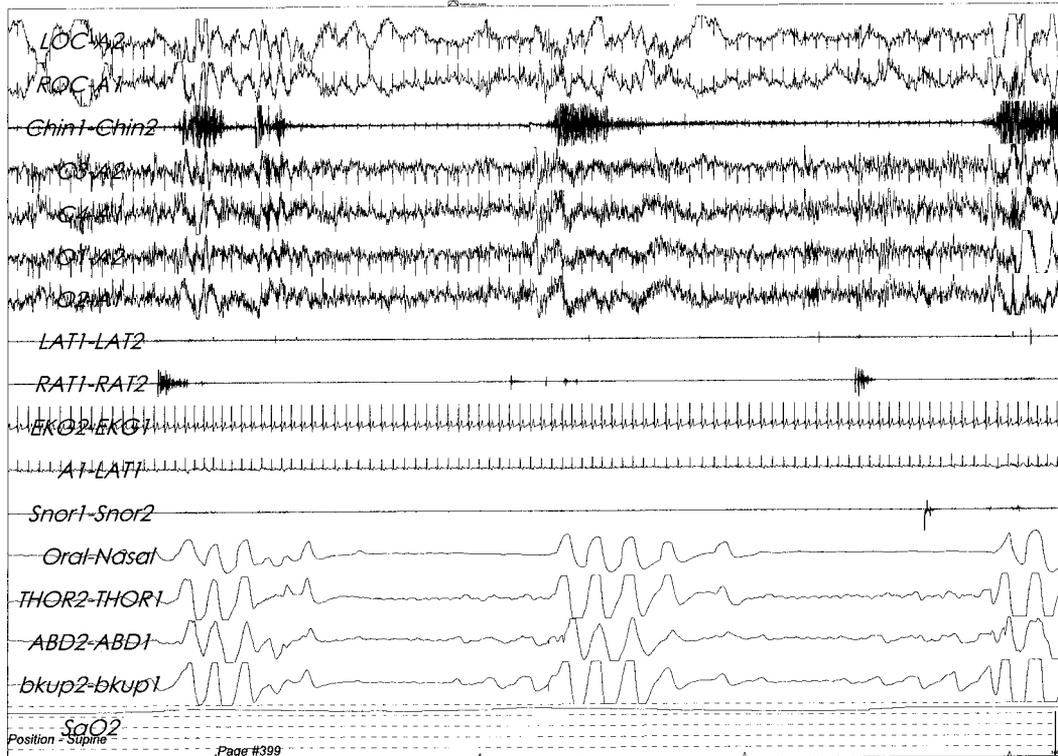
## Adult Respiratory Definitions

In adults, apnea is defined as the absence of airflow at the mouth for 10 s or longer.<sup>21,22</sup> If one measures the airflow with a very sensitive device, such as a pneumotachograph, small expiratory puffs can sometimes be detected during an apparent apnea. In this case, there is “inspiratory apnea.” Many sleep centers regard a severe decrease in airflow (to <10% of baseline) to be an apnea.

An obstructive apnea is cessation of airflow with persistent inspiratory effort. The cause of apnea is an obstruction in the upper airway. In Figure 11-11, an obstructive apnea (no airflow with coexistent movement in the chest and abdomen) is followed by the corresponding nadir in the arterial oxygen saturation. In central apnea, there is an absence of inspiratory effort. A mixed apnea is defined as an apnea with an initial central portion followed by an obstructive portion (Fig. 11-12). A hypopnea is a reduction in airflow for 10 s or longer.<sup>21</sup> The apnea + hypopnea index (AHI) is the total number of apneas and hypopneas per hour of sleep. In adults, an AHI of less than 5 is considered normal.



**FIGURE 11-11.** Respiratory monitoring in PSG includes airflow (Nasal/Oral) Respiratory effort (THOR-1-THOR2, ABD1-ABD2 and Bkup2-bkup1) and oxygen saturation ( $\text{SaO}_2$ ). This page shows an obstructive apnea, in which airflow ceases while respiratory effort in the abdominal, thoracic and backup lead continues. Note that the nadir of oxygen saturation lags the onset of the apnea. (From Geyer JD, Payne TA, Carney PR et al. *Atlas of Digital Polysomnography*. Philadelphia, PA: Lippincott Williams & Wilkins, 2000. (Ref. 6))



**FIGURE 11-12.** A mixed apnea with an initial central portion during which there is no inspiratory effort followed by an obstructive portion with paradoxical chest-abdominal movement. The small fluctuations in the chest and abdominal tracings during the central portion are from cardiac pulsations.

Hypopneas can be further classified as obstructive, central, or mixed. If the upper airway narrows significantly, the airflow can fall (obstructive hypopnea).<sup>31</sup> Alternatively, airflow can fall from a decrease in respiratory effort (central hypopnea). Finally, a combination is possible (mixed hypopnea) with both a decrease in respiratory effort and an increase in upper airway resistance. However, unless accurate measures of airflow and esophageal or supraglottic pressure are obtained, such differentiation is usually not possible. In clinical practice, one usually identifies an obstructive hypopnea by the presence of airflow vibration (snoring), chest-abdominal paradox (increased load), or evidence of airflow flattening (airflow limitation) in the nasal pressure signal. In Figure 11-10, two obstructive hypopneas, each followed by an arterial oxygen desaturation, are depicted. A central hypopnea is associated with an absence of snoring, a round airflow profile (nasal pressure), and an absence of chest-abdominal paradox. However, in the absence of esophageal pressure monitoring, a central hypopnea cannot always be classified with certainty. In addition, obstructive hypopnea may not always be associated with chest-abdominal paradox. Because of the limitations in exactly determining the type of hypopnea, most sleep centers usually report only the total number and frequency of hypopneas. The new requirements for an event to be classified as a hypopnea are as follows. A hypopnea should be scored only if all of the following criteria are present.<sup>32</sup>

- The nasal pressure signal excursions (or those of the alternative hypopnea sensor) drop by more than 30% of baseline
- The event duration is at least 10 s
- There is a greater than 4% oxygen desaturation from pre-event baseline

- At least 90% of the event's duration must meet the amplitude reduction of criteria for hypopnea

Alternatively, a hypopnea can also be scored if all of these criteria are present.

- The nasal pressure signal excursions (or those of the alternative hypopnea sensor) drop by more than 50% of baseline
- The duration of the event is at least 10 s
- There is a greater than 3% oxygen desaturation from preevent baseline or the event is associated with arousal
- At least 90% of the event's duration must meet the amplitude reduction criteria for hypopnea.

Respiratory events that do not meet criteria for either apnea or hypopnea can induce arousal from sleep. Such events have been called upper-airway resistance events (UARE), after the UARS.<sup>11</sup> An AASM Task Force recommended that such events be called respiratory effort-related arousals (RERAs). The recommended criterion for an RERA is a respiratory event of 10 s or longer followed by an arousal that does not meet the criteria for an apnea or hypopnea but is associated with a crescendo of inspiratory effort (esophageal monitoring) or a flattened waveform on nasal pressure monitoring.<sup>28</sup> Typically, following arousal, there is a sudden drop in esophageal pressure deflections. The exact definition of hypopnea that one uses will often determine whether a given event is classified as a hypopnea or an RERA.

One can also detect the flow-limitation arousals (FLA) using an accurate measure of airflow, such as nasal pressure. Such events are characterized by flow limitation (flattening) over several breaths followed by an arousal and sudden, but often temporary, restoration of a normal-round airflow profile. One study suggested that the number of FLA per hour corresponded closely to the RERA index identified by esophageal pressure monitoring.<sup>33</sup> Some centers compute a RAI, determined as the arousals per hour associated with apnea, hypopnea, or RERA/FLA events. The AHI and respiratory disturbance index (RDI) are often used as equivalent terms. However, in some sleep centers, the  $RDI = AHI + RERA$  index, where the RERA index is the number of RERAs per hour of sleep, and RERAs are arousals associated with respiratory events not meeting the criteria for apnea or hypopnea.

One can use the AHI to grade the severity of sleep apnea. Standard levels include normal (<5), mild (5 to <15), moderate (15 to 30), and severe (>30) per hour. Many sleep centers also give separate AHI values for NREM and REM sleep and various body positions. Some patients have a much higher AHI during REM sleep or in the supine position (REM-related or postural sleep apnea). Because the AHI does not always express the severity of oxygen desaturation, one might also grade the severity of desaturation. For example, it is possible for the overall AHI to be mild, but for the patient to have quite severe desaturation during REM sleep.

**QUESTION 11.6:** According to the primary definition of hypopnea, all of the following are true except...

- The nasal pressure signal excursions (or those of the alternative hypopnea sensor) drop by more than 30% of baseline.
- The event duration is at least 10 s.
- There is a greater than 4% oxygen desaturation from preevent baseline.
- At least 50% of the event's duration must meet the amplitude reduction of criteria for hypopnea.

**ANSWER:** d.

## Pediatric Respiratory Definitions

Periodic breathing is defined as three or more respiratory pauses of at least 3 s in duration separated by less than 20 s of normal respiration. Periodic breathing is seen primarily in premature infants and mainly during active sleep.<sup>34</sup> Although controversial, some feel that the presence of periodic breathing for more than 5% of TST or during quiet sleep in term infants is abnormal. Central apnea in infants is thought to be abnormal if the event is more than 20 s in duration or associated with arterial oxygen desaturation or significant bradycardia.<sup>34-37</sup>

In children, a cessation of airflow of any duration (usually two or more respiratory cycles) is considered an apnea when the event is obstructive,<sup>34-37</sup> i.e., the 10-s rule for adults does not apply. Of note, the respiratory rate in children (20 to 30/min) is greater than in adults (12 to 15/min). In fact, 10 s in an adult is usually the time required for two to three respiratory cycles. Obstructive apnea is very uncommon in normal children. Therefore, an obstructive AHI greater than 1 is considered abnormal. In children with OSA, the predominant event during NREM sleep is obstructive hypoventilation rather than a discrete apnea or hypopnea. Obstructive hypoventilation is characterized by a long period of upper-airway narrowing with a stable reduction in airflow and an increase in the end-tidal PCO<sub>2</sub>. There is usually a mild decrease in the arterial oxygen desaturation. The ribcage is not completely calcified in infants and young children. Therefore, some paradoxical breathing is not necessarily abnormal. However, worsening paradox during an event would still suggest a partial airway obstruction. Nasal pressure monitoring is being used more frequently in children and periods of hypoventilation are more easily detected (reduced airflow with a flattened profile). Normative values have been published for the end-tidal PCO<sub>2</sub>. One paper suggested that a peak end-tidal PCO<sub>2</sub> greater than 53 mm Hg or end-tidal PCO<sub>2</sub> greater than 45 mm Hg for more than 60% of TST should be considered abnormal.<sup>35</sup>

Central apnea in infants was discussed above. The significance of central apnea in older children is less certain. Most do not consider central apneas following sighs (big breaths) to be abnormal. Some central apnea is probably normal in children, especially during REM sleep. In one study, up to 30% of normal children had some central apnea. Central apneas, when longer than 20 s, or those of any length associated with SaO<sub>2</sub> below 90% are often considered abnormal, although a few such events have been noted in normal children.<sup>38</sup> Therefore, most would recommend observation alone unless the events are frequent.

### QUESTION 11.7: In the pediatric patient, which of the following statements regarding respiratory monitoring is false?

- Periodic breathing is defined as three or more respiratory pauses of at least 3 s in duration separated by less than 20 s of normal respiration.
- The presence of periodic breathing for more than 5% of TST or during quiet sleep in term infants is abnormal.
- Central apnea in infants is thought to be abnormal if the event is more than 10 s in duration or associated with arterial oxygen desaturation or significant bradycardia.
- In children, a cessation of airflow of any duration (usually two or more respiratory cycles) is considered an apnea when the event is obstructive.

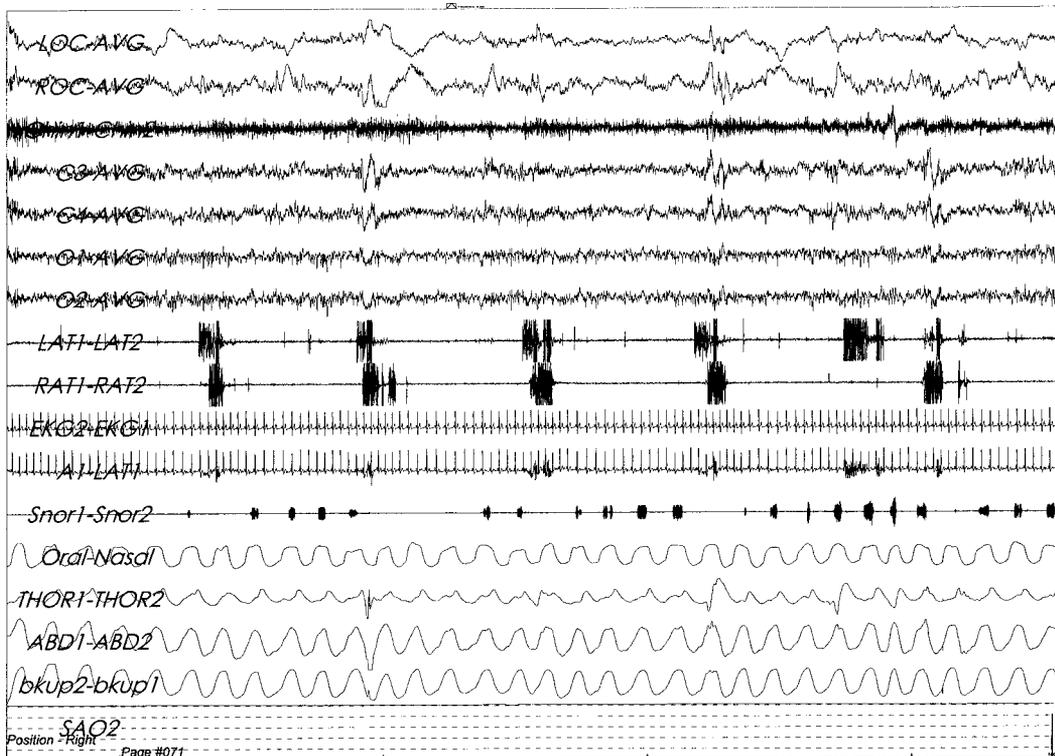
**ANSWER:** c.

## Leg Movement Monitoring

The EMG of the anterior tibial muscle (anterior lateral aspect of the calf) of both legs is monitored to detect the leg movements (LMs).<sup>39</sup> Two electrodes are placed on the belly of the upper portion of the muscle of each leg about 2 to 4 cm apart. A electrode loop is taped in place to provide strain relief. Usually each leg is displayed on a separate channel. However,

if the number of recording channels is limited, one can link an electrode on each leg and display both leg EMGs on a single tracing. Recording from both legs is required to accurately assess the number of movements. During biocalibration, the patient is asked to dorsiflex and plantarflex the great toe of the right leg and then the left leg to determine the adequacy of the electrodes and amplifier settings. The amplitude should be 1 cm (paper recording) or at least one half of the channel width on digital recording.

An LM is defined as an increase in the EMG signal of at least one fourth the amplitude exhibited during biocalibration that is one half to 10 s in duration.<sup>39</sup> Periodic LMs (PLMs) should be differentiated from bursts of spike-like phasic activity that occur during REM sleep. To be considered a PLM, the movement must occur in a group of four or more movements (see Figure 11-13), each separated by more than 5 and less than 90 s (measured onset to onset). To be scored as a PLM in sleep, an LM must be preceded by at least 10 s of sleep. In most sleep centers, LMs associated with termination of respiratory events are not counted as PLMs. Some may score and tabulate this type of LM separately. The PLM index is the number of PLMs divided by the hours of sleep (TST in hours). Rough guidelines for the PLM index are (>5 to <25 per hour) mild, (25 to <50 per hour) moderate, and ( $\geq 50$  per hour) severe.<sup>40</sup> A PLM arousal is an arousal that occurs simultaneously with or following (within 1 to 2 s) a PLM. The PLM arousal index is the number of PLM arousals per hour of sleep. A PLM arousal index of more than 25 per hour is considered severe. LMs that occur during wake or after an arousal are either not counted or tabulated separately. For example, the PLMW (PLM wake) index is the number of PLMs per hour of wake. Of note, frequent LMs during wake, especially at sleep onset, may suggest the presence of the restless legs syndrome. The latter is a clinical diagnosis made on the basis of patient symptoms.



**FIGURE 11-13.** Sample tracing of right and left legs on separate channels.

**TABLE 11-7 Biocalibration Procedure**

Eyes closed	EEG: Alpha EEG activity EOG: Slow eye movements
Eyes open	EEG: Attenuation of alpha rhythm EOG: REMs, blinks
Look right, look left, look up, look down	Integrity of eye leads, polarity, amplitude Eye movements should cause out-of-phase deflections
Grit teeth	Chin EMG
Breathe in, breathe out	Airflow, chest, abdomen movements adequate gain? Tracings in phase? (polarity of inspiration is usually upward)
Deep breath in, hold breath	Apnea detection
Wiggle right toe, left toe	Leg EMG, amplitude reference to evaluate LMs

### Polysomnography, Biocalibrations, and Technical Issues

In addition to the standard physiologic parameters monitored in polysomnography, body position (using low-light video monitoring) and treatment level (CPAP, bilevel pressure) are usually added in comments by the technologists. In most centers, a video recording is also made on traditional video tape or digitally as part of the digital recording. It is a standard practice to perform amplifier calibrations at the start of recording. In traditional paper recording, a calibration voltage signal (square wave voltage) was applied and the resulting pen deflections, along with the sensitivity, polarity, and filter settings on each channel, were documented on the paper. Similarly, in digital recording, a voltage is applied, although it is often a sine-wave voltage. The impedance of the head electrodes is also checked prior to recording. An ideal impedance is greater than 5,000  $\Omega$ , although 10,000  $\Omega$  or less is acceptable. Electrodes with higher impedances should be changed.

A biocalibration procedure is performed (Table 11-7) while signals are acquired with the patient connected to the monitoring equipment.<sup>4,5</sup> This procedure permits the checking of amplifier settings and integrity of monitoring leads/transducers. It also provides a record of the patient's EEG and eye movements during wakefulness with eyes closed and open. A summary of typical commands and their utility is listed in Table 11-7.

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