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THIRD EDITION Sudhansu Chokroverty
Sleep Disorders Medicine
Basic Science, Technical Considerations, and Clinical Aspects

Chapter 18 – Clinical Polysomnography and the Evolution of Recording and Scoring Technique

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INTRODUCTION

Sleep can be defined many ways. Behaviorally, it is a reversible state of inactivity associated with decreased responsiveness. In humans, sleep usually begins in the late evening or early night. During sleep, our bodies cool and remain mostly immobile. As in coma, responsiveness to environmental stimuli declines, but unlike coma, the state can rapidly change to wakefulness, usually without lingering cognitive impairment. Coma passively results from brain stem and cortical metabolic depression; however, sleep is an active process.[1]

Although most often described as a “state,” sleep represents an essential brain process. As such, the traditional approach for investigating sleep involves comparing brain activity during sleep to brain activity accompanying wakefulness. One of the earliest tools available for investigating brain activity was electroencephalography (EEG). Thus, studying the EEG correlates of normal sleep was a logical place for the scientific study of sleep to begin.

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ELECTROENCEPHALOGRAPHIC AND ELECTRO-OCULOGRAPHIC WAVEFORMS IN SLEEP

Descriptions and Examples

Hans Berger, the father of EEG, made the first sleep recordings.[2] EEG activity during relaxed wakefulness (with eyes closed) was a nearly sinusoidal pattern in the frequency range of 8–13 cycles/sec. Berger named this rhythm “alpha” and found that it would diminish when the subject opened his or her eyes or engaged in mental arithmetic. Berger also discovered that alpha activity was replaced by low-voltage mixed frequency activity at sleep onset. Even to this day, this finding remains as the foundation of sleep-wake classification, with EEG alpha cessation defining the transition from wakefulness to sleep (Fig. 18-1). The low-voltage, mixed-frequency activity associated with sleep onset is also marked by a general slowing of EEG frequency and the appearance of a 4- to 7-cycle/sec waveform called EEG *theta activity*. By contrast, the abrupt diminution of alpha activity provoked by eye opening is usually characterized by high-frequency EEG beta activity (>13 cycles/sec), rapid eye movements associated with changes in direction of gaze, and/or blinking.

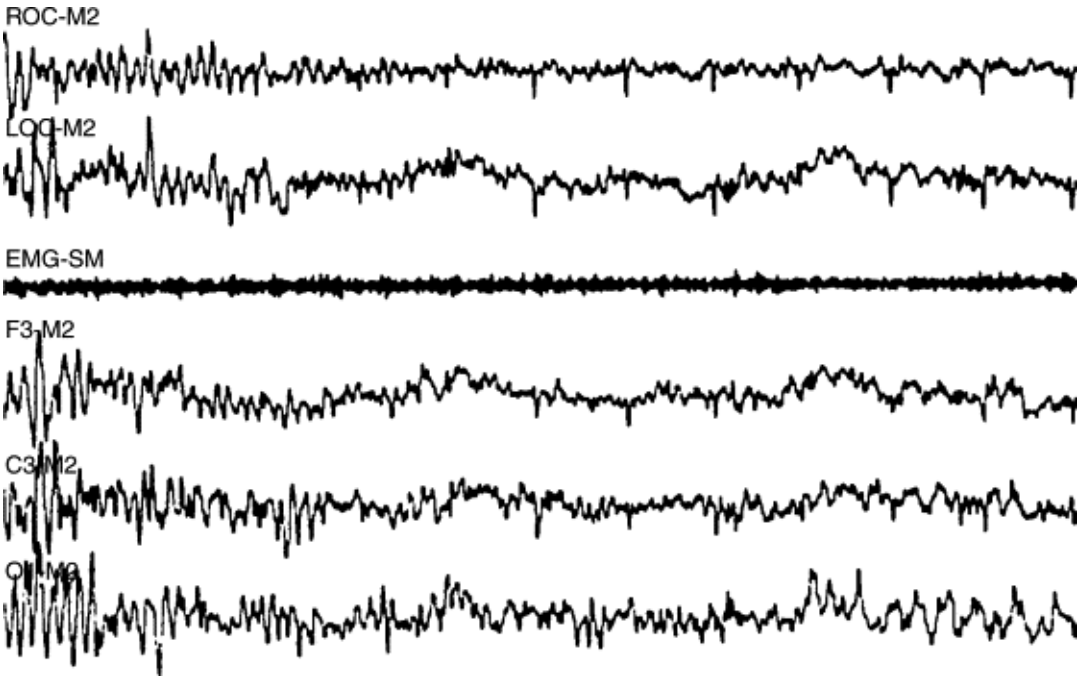

FIGURE 18-1 Transition from wakefulness to sleep. (C3, left central; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

The first continuous all-night EEG recordings of human sleep were not made until almost 3 decades after Berger first recorded the brain's electrical activity during sleep.[3] It became immediately apparent that sleep was not a single homogeneous process. The low-voltage, mixed-frequency activity seen at sleep onset would sometimes contain single, high-amplitude, negative-going, high-frequency wave bursts. These waveforms are known as *vertex sharp waves* and are commonly observed near sleep onset (Fig. 18-2). As sleep further progresses, there begin to appear short phasic bursts of discrete 12- to 16-cycle/sec waveforms typically lasting 0.5–1.5 seconds. The waveform envelope is “spindle” shaped, and consequently these bursts were named *sleep spindles* (Fig. 18-3). Sometimes in concert with or proximal to a sleep spindle, a high-amplitude, negative-going sharp wave appears and is immediately followed by a positive component. This waveform, referred to as a *K complex,* stands out from the background activity and has a total duration greater than 0.5 seconds (Fig. 18-4).

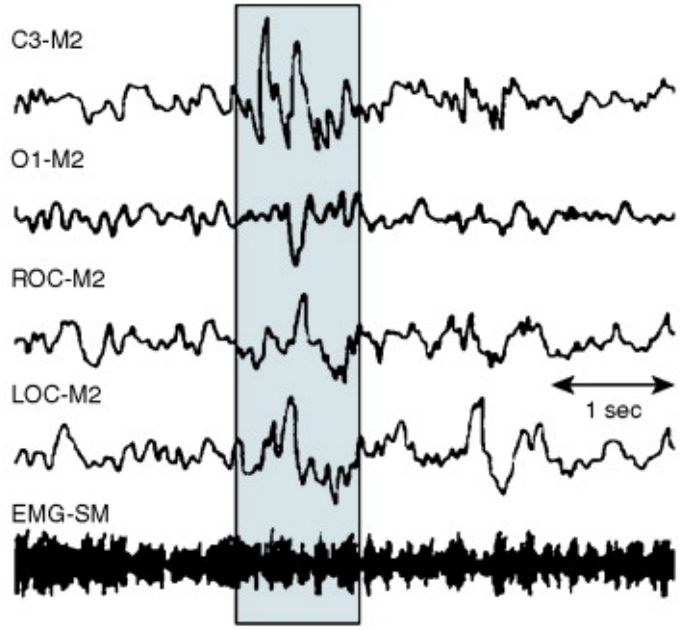

FIGURE 18-2 Vertex sharp wave. (C3, left central; EMG-SM, electromyogram-submentalis; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

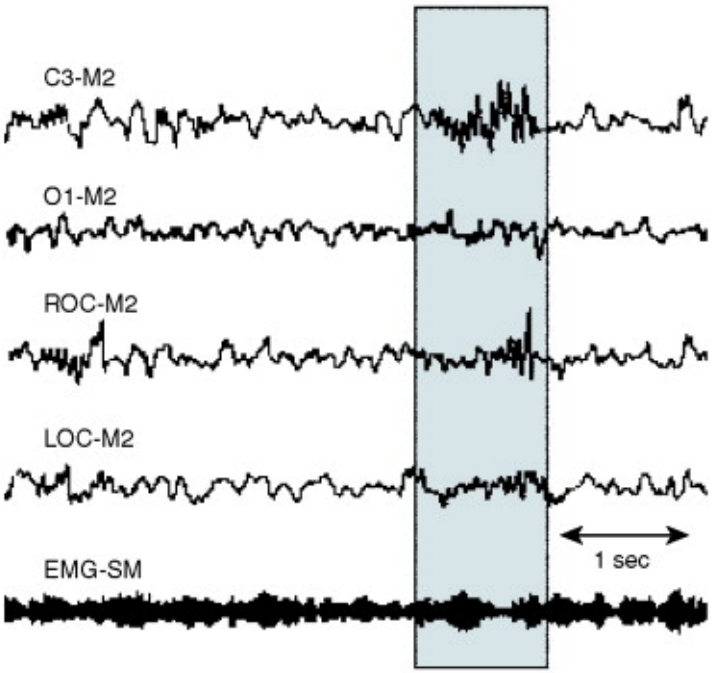

FIGURE 18-3 Sleep spindle. (C3, left central; EMG-SM, electromyogram-submentalis; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

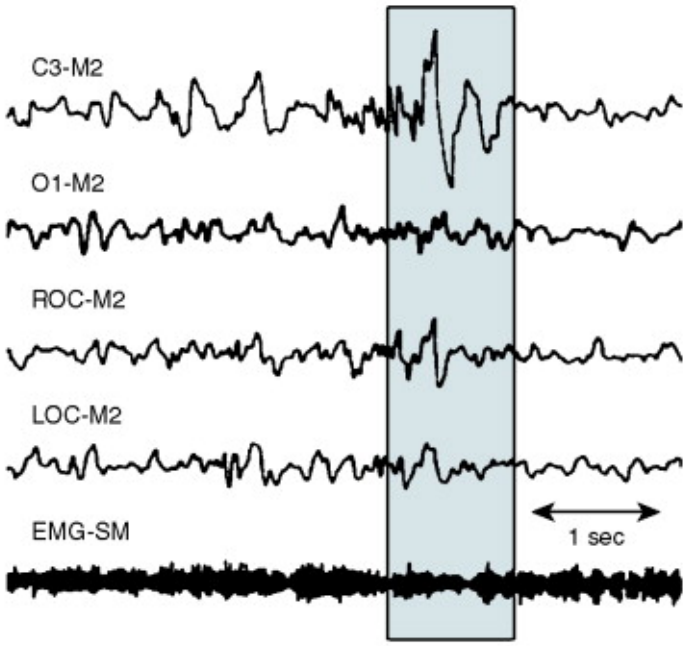

FIGURE 18-4 K complex. (C3, left central; EMG-SM, electromyogram-submentalis; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

Over the course of the first hour of sleep, most individuals will gradually have increasing amounts of high-amplitude, low-frequency activity called EEG *delta rhythm* (with frequencies <4 cycles/sec). In the young adult, delta rhythm and a subset called *slow waves* (with frequencies <2 cycles/sec) usually increase to the point that the low-voltage, mixed-frequency activity is completely replaced by these high-voltage synchronized waves (Fig. 18-5). Sleep spindles may persist, occurring in conjunction with the slow-wave activity, and can sometimes be seen “riding” on the slow waves. When the bout of delta and slow-wave activity subsides, a period of low-voltage, mixed-frequency activity re-emerges. During this period, theta activity is often observed. This theta rhythm's morphology differs slightly from that seen near sleep onset in that the waves have a notched appearance, resembling the teeth on a saw. These *sawtooth theta waves* (Fig. 18-6) usually do not occur interspersed with sleep spindles and K complexes but rather appear in conjunction with rapid eye movements.

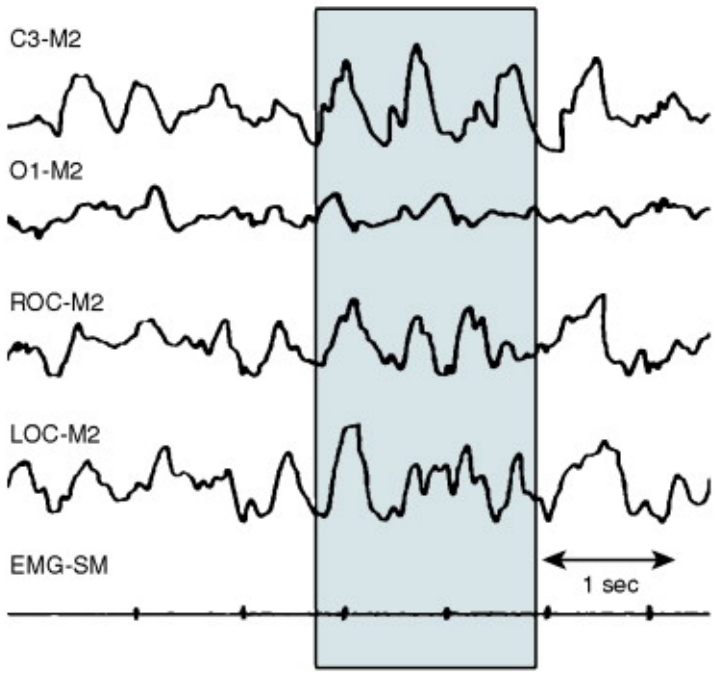

FIGURE 18-5 Slow-wave activity. (C3, left central; EMG-SM, electromyogram-submentalis; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

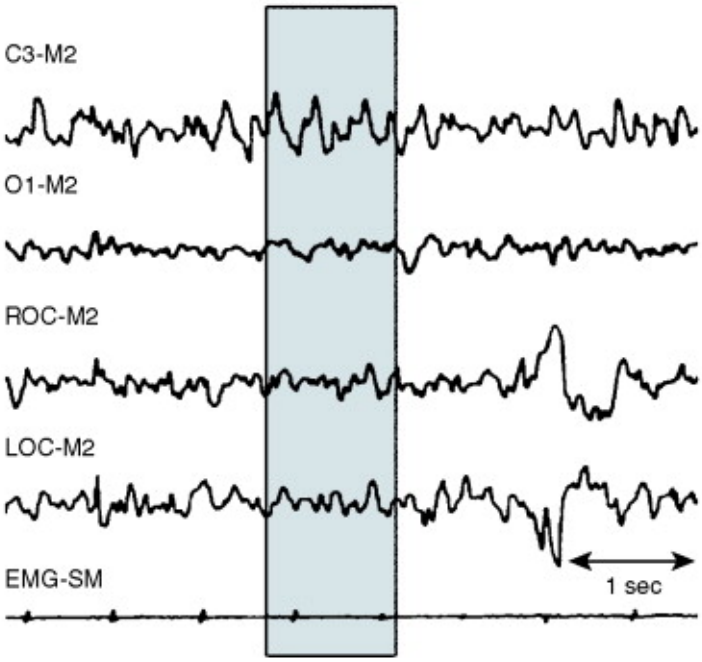

FIGURE 18-6 Sawtooth theta activity. (C3, left central; EMG-SM, electromyogram-submentalis; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

Eye movements can easily be recorded by placing an electrode on the face near the eye. The cornea has a positive charge and, as the eye moves toward an electrode, a large voltage change occurs. By making recordings with electrodes placed near the outer canthus of each eye, horizontal eye movements can be recorded. As one eye is moving toward its proximal electrode and the other eye is moving away from the other electrode, voltage deflections are in opposite directions (out of phase) on the recording. Consequently, these eye movements can be easily differentiated from EEG and in particular delta EEG activity (which is in phase).

Eye movements occur during both wakefulness and sleep. The saccade is a very rapid eye movement in which the brain moves the eye from one target to another (with ocular suppression occurring during the actual transit). The *rapid eye movements* (REMs) that accompany the low-voltage, mixed-frequency background EEG that are thought to correspond to direction of gaze during dreaming[4,][5] are slightly slower than eyes-open saccades. It has been demonstrated that volitional saccades in a dark field are of equivalent speed; therefore, the velocity loss appears to be visual field dependent rather than state dependent. Additionally, there are slow, rolling, almost pendular eye movements that can occur in the drowsy awake state, at sleep onset, and/or for up to several minutes after sleep has become well established. These *slow eye movements* in some instances may be correlated with hypnagogic or hypnapompic imagery. Rapid and slow eye movements during sleep are illustrated in Figure 18-7.

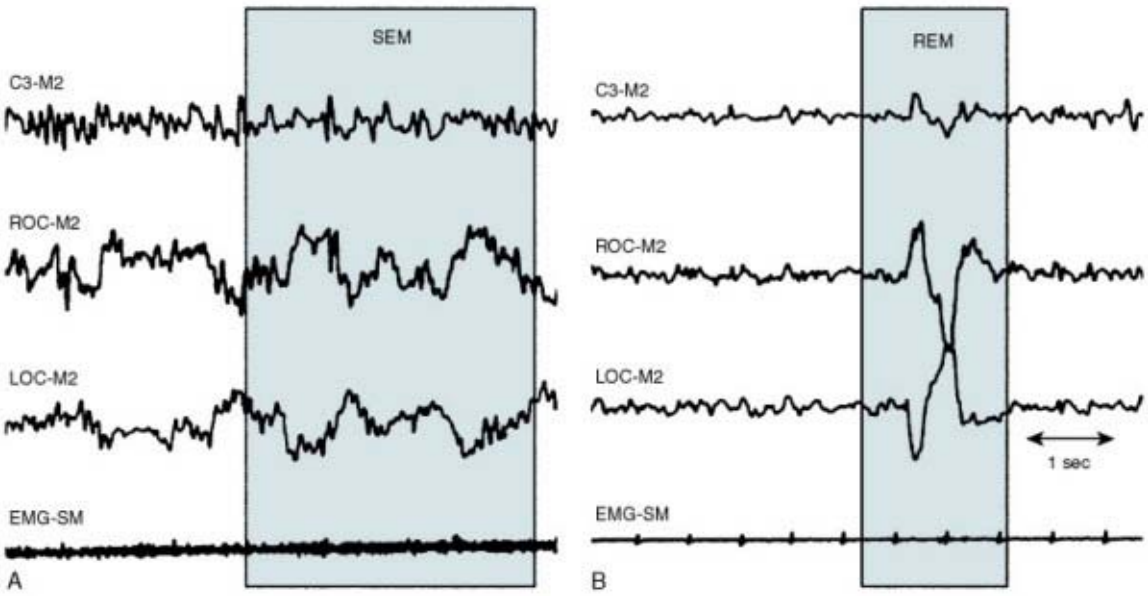

FIGURE 18-7 Sleep-related eye movements. **(A)** A typical slow eye movement (SEM) of the type usually seen at sleep onset. **(B)** A rapid eye movement

(REM) characteristic of REM sleep. (C3, left central; EMG-SM, electromyogram-submentalis; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

Summarizing Waveforms

Waveforms, like most physiologic parameters, can be characterized along the standard dimensions: frequency, magnitude, and duration. For example, sleep spindle density (the mean number of spindles per minute) is a common frequency index. Magnitude could be quantified in terms of the average peak amplitude or area under the curve normalized for overall duration. Mean spindle length would exemplify a duration measure. Obviously, because the number of waveforms occurring during a single night may be very large, hand measuring, tabulating, and statistically summarizing the frequency, magnitude, and duration of each could amount to a Herculean task. Therefore, automation was embraced early in sleep research laboratories. Using computers to perform complex EEG analysis (e.g., Fourier transforms, period-amplitude analysis, and complex demodulation) to characterize sleep EEGs and plot spectral arrays long predates their use as digital polysomnographs.[6]

Waveform analysis has been used to study sleep physiology,[7] psychiatric illness,[8] clinical pharmacology,[9] sleep disorders,[10] and aging.[11] Other computerized techniques examining connectivity between waveform events (e.g., Markovian chain analysis) attempt to understand the behavior of the underlying physiology. To date, there has not been any large-scale organized attempt to standardize sleep signal processing recording method, detection technique, quantitative analysis, or summarization. Each researcher tailors his or her technique to meet the particular investigative needs.

Waveform Changes Across the Night

Certainly the most profound intranight waveform change is the progressive decrease in delta (and slow-wave) activity observed in most individuals.[12] Commonly quantified as delta power (a measure combining magnitude and duration), some researchers posit delta activity as a measure of sleep homeostatic drive. The prototypical record shows rapid evolution of delta and slow waves dominating the EEG in the first 60–90 minutes of sleep. After a brief interval marked by low-voltage, mixed-frequency EEG activity, delta power again rises, but neither as high or for as long as in the first cycle. Another interval of low-voltage, mixed-frequency activity intervenes before another round of delta activity commences. Through each cycle, delta power diminishes. By contrast, REM activity generally increases as the night progresses. This gradual increase may also involve theta, and especially sawtooth theta, activity. Sleep spindles, theta activity, and other waveforms remain fairly stable across the night of sleep.

Changes as a Function of Age

Of all the age-related phenomena revealed by polysomnography, the decline in delta and slow-wave activity is perhaps the most striking.[13] Not only does the duration of slow-wave activity decline, but the amplitude of the waveforms declines as well. The extent of the decline is what makes it dramatic. Sophisticated analytic tools are not required to visualize the changes; they are apparent to the naked eye (Fig. 18-8). Not surprisingly, most of the other waveforms also show age-related decline. Studies of K complexes, sleep spindles, and theta activity reveal age-related alterations in density and amplitude. Similarly, REM density may be lower in some samples of elderly individuals.

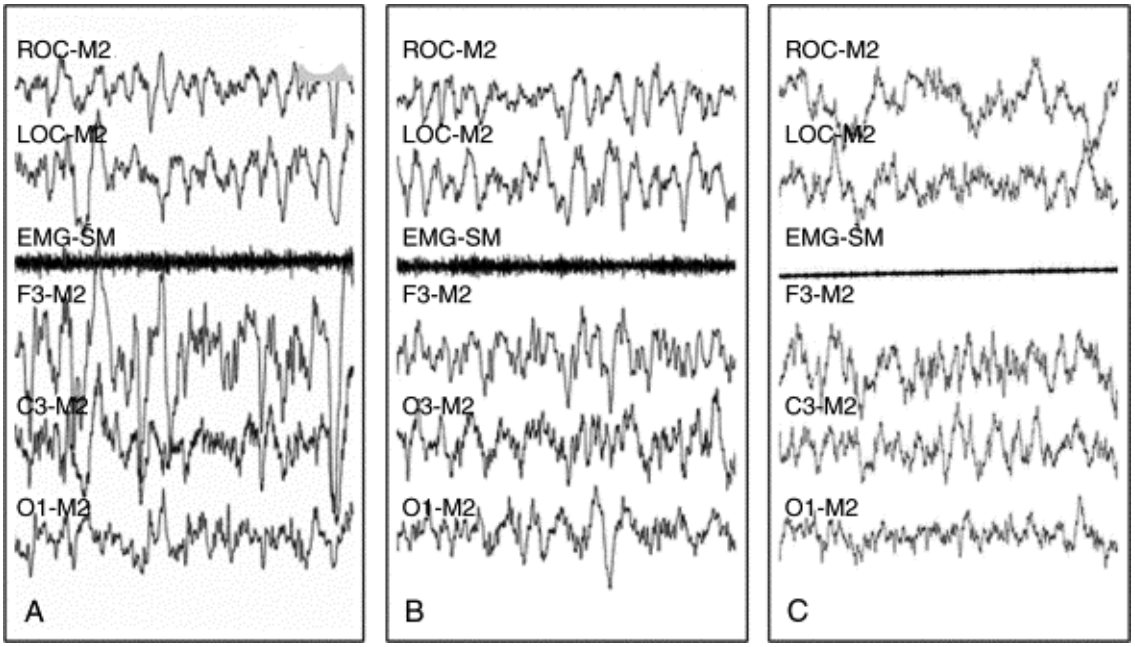

FIGURE 18-8 Slow Wave Activity in a Child (**A**), Young Adult (**B**), and Very Healthy Older Adult (**C**). This figure illustrates that in addition to decreased duration of sleep stages 3 and 4 (slow wave sleep), amplitude of slow activity also declines with age. (C3, left central; EMG-SM, electromyogram-

submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

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SLEEP STAGING

Recording and Scoring Techniques

It is hard to imagine how overwhelming the enormity of the data must have seemed to early researchers when they first began collecting continuous all-night polysomnographic recordings from human subjects. Information needed to be summarized into a manageable form. From the very start, schemes were developed to reduce data within a time domain; thus, *sleep staging* was invented.

Staging's basic principle involves nominally classifying a recording segment of set duration according to the activity observed during that time interval. In this case, the nominal classifications are categories called “stages” and the set duration is called an “epoch.” Also, until the mid-1980s, sleep studies were mainly recorded on paper. The duration of an epoch varied in different laboratories, undoubtedly influenced mainly by paper chart speed and paper size. Early fan-fold paper was 30 cm wide and came in boxes of 1000 sheets. If the chart speed was 10 mm/sec (as was common in many sleep laboratories), then one page of recording was 30 seconds in duration and one box of paper would hold 8 hours and 20 minutes worth of tracings. Designating one page as one epoch was very convenient, and it quickly became a de facto standard.

Categorizing each epoch as one or another sleep stage is based on similarities and differences in the activity present. An assortment of rules can be developed to guide categorization (re: staging). But how many categories are needed? How well do the generalizations work? Does sleep staging really characterize the bioelectrical activity associated with normal sleep? To explore these questions, consider the following exercise the senior author (M.H.) regularly conducts on the first day of class before the medical students, residents, and fellows have been taught anything about sleep medicine. Each student is given 100 randomly selected, 30-second pages selected from five different normal polysomnographic 4-channel (C3-A2 EEG, ROC-A2, A2-LOC, and submentalis EMG) recordings. Students are instructed to sort the pages into piles, based on similarities of the squiggly lines decorating each page. Recording channels, recording speed, and even page orientation remains unspecified. No other directions are given. Groups of two and three individuals are formed and the exercise is performed three times, each time with different partners. The first two trials are considered practice and the results of the third are tabulated. On the third trial of blindly categorizing paper tracings, novices create 5–10 categories and REM sleep is recognized with near universality. Pages with low-voltage, mixed-frequency activity that also have sleep spindles are grouped with great regularity. Furthermore, one to three categories containing slow waves are derived. Overall, the majority of pages are sorted into categories that roughly correspond to one or another stage definition. Thus, even with all the arbitrariness of the staging rules' specific details, every time we experimentally invent sleep staging we produce similar results. Therefore, the reader should not be surprised by the remarkable comparability of the different sleep staging systems described below.

Loomis (1936)

Recording Montage

As the first researchers to make continuous all-night sleep recordings, Loomis and colleagues[14] were also the first to face the daunting task of quantifying miles of paper tracings for summary analysis. They developed a data reduction scheme called sleep staging (*stages A, B, C, D, and E*). Staging was largely based on the presence of particular EEG activity. EEG activity included beta activity, sleep spindles, alpha rhythm, theta rhythm, delta rhythm, and slow waves recorded from three electrodes pairs.[14] One electrode was place above and to the left of the left eye and referenced to the mastoid behind the ear. This derivation was used to detect eye movements and frontal lobe activity. A second electrode, placed midline at the top of the head (roughly corresponding to Cz) was referenced to the left mastoid and was used to score sleep spindles and K complexes. The final active recording site was midline on the occiput (mastoid referred) and was used to maximally visualize alpha activity.

Sleep Staging

Stage A (Alpha) is described as containing alpha “trains” of varying durations possibly accompanied by slow, rolling eye movements. *Stage B (Low Voltage)* has neither alpha activity nor spindle activity; however, rolling eye movements may occur. *Stage C (Spindles)* is defined by the presence of sleep spindle activity with mostly low-voltage background. *Stage D (Spindles plus random)* is scored when sleep spindles occur together with large “random” waves (corresponding to delta and slow wave activity). Finally, *Stage E (Random)* is when delta and slow waves dominate the recording, or, as the authors put it “large random potentials persist and come from all parts of the cortex.” Interestingly, when sleep is graphically represented over the course of a night with stages arrayed from A through E on the *y* axis and time on the *x* axis, the histograms look remarkably similar to what is used today. Also noted on the histograms were subject reports of dreaming that occurred when subjects awoke from stage B.

Dement-Kleitman (1957)

Dement and Kleitman[15] developed criteria for classifying epochs of sleep according to EEG criteria. To a large degree, this technique was the foundation for most scoring systems used throughout the world, up to and including the present time. Noteworthy was the fact that it was the first scoring system that incorporated REM sleep, the new sleep stage recently discovered in their laboratory by Eugene Aserinsky. Essentially, each recording epoch was classified as awake or sleep stage 1, 2, 3, or 4. *Stage 1* was classified when a nonawake EEG with a low-voltage, mixed-frequency background was devoid of sleep spindles and K complexes. In general, Dement-Kleitman stage 1 corresponds to Loomis Stages A and B. Sleep *stage 2* was classified when sleep spindles and/or K complexes were intermingled with a low-voltage, mixed-frequency background EEG. *Stage 3* was characterized by appearance of slow waves (100 μV or more at a frequency ≤2 cycles/sec), and finally *stage 4* was assigned to epochs composed of 50% or more of these high-amplitude, slow waves. When REMs occurred during stage 1 sleep, the stage was designated *REM sleep*.

Williams and Karacan (1959)

Recording Montage

Robert L. Williams is one of the unsung pioneers of sleep medicine. In June of 1959, the sleep laboratories at the University of Florida College of Medicine in Gainesville began making continuous all-night sleep recordings in normal, healthy individuals.[16] With the rationale that, to understand sleep disorders, one must first characterize normal human sleep, “normative” data were collected over the next decade from both male and female children, adolescents, teenagers, young adults, adults, and seniors. In 1974, the compilation of normal values for sleep were published in book form, stratified by sex and age group (3–5 years, 6–9 years, 10–12 years, 13–15 years, 16–19 years, 20–29 years, 30–39 years, 40–49 years, 50–59 years, 60–69 years, and 70–79 years). The recording technique used by Williams and colleagues employed three channels of EEG derivations (F1-F7, C3-A2, and O3-OzPz) and two channels of electro-oculogram (EOG) derivations (Left Eye–A2 and A2–Right Eye). Notably absent from this montage was a channel for assessing submentalis muscle tone to detect REM sleep–related atonia. This was not an oversight; by contrast, the developers of this system provide a strong argument against including submentalis electromyography (EMG). The authors wrote, “Since there are wide individual differences in tonus of the chin muscles which begin to undergo changes during adulthood, this measure becomes less reliable with age starting as early as age 40 and is useless with elderly subjects.”

The selection of three EEG channels stemmed from recognition that EEG from particular derivations had differential usefulness for visualizing specific waveforms. A monopolar channel near the vertex (C3-A2) is generally reliable for recording most waveforms, including vertex sharp waves, K complexes, sleep spindles, theta activity, and slow waves. This is why most recording systems use a central lobe channel or even rely totally on this channel for all brainwave activity. Recording a single all-purpose EEG signal was advantageous back when amplifiers were expensive and the number of channels limited. Nonetheless, it was well known among sleep researchers and neurologists that EEG alpha alterations associated with wakefulness and sleep were most prominent in occipital leads. Therefore, Williams and colleagues incorporated O3-OzPz into the montage to facilitate and improve scoring reliability of sleep onset, awakenings, and central nervous system (CNS) arousals. The inclusion of a third EEG channel (recorded over the frontal lobe [F1-F7]) was designed to enhance delta (and slow-wave) activity detection and visualization. With their focus on sleep changes over the life span, Williams and colleagues were keen to obtain high-quality data for delta bandwidth activity because it was often the primary outcome measure in studies of sleep and aging.

Sleep Staging

Polygraph chart speed at the University of Florida College of Medicine in Gainesville was 15 mm/sec rather than the 10 mm/sec commonly used elsewhere. Consequently, each page contained 20 seconds of data and each set of 3 pages constituted a 1-minute epoch. In the Williams-Karacan scoring system, wakefulness is designated *stage 0*. An epoch is classified as stage 0 if it contained “at least 30 seconds of 8 to 12 Hz occipital activity, with a minimum amplitude of 40 microvolts peak-to-peak.” *Stage 1* criteria were (1) less than 30 seconds of occipital alpha activity and (2) no more than one well-defined spindle or K complex. It was permissible to use muscle artifact and eye movements to facilitate staging in alpha nonproducers. *Stage 2* required at least two well-defined sleep spindles or K complexes, or one of each. Additionally, the epoch could contain no more than 12 seconds of 40-μV (or greater), 1–3-cycles/sec slow waves. Between 13 and 30 seconds of 1–3-cycle/sec slow waves was obligatory for scoring *Stage 3,* and 30 seconds or more were necessary to classify an epoch as *Stage 4*. Eye movements were scored independently from the EEG activity. Epochs of stage 1 sleep that also contained REMs were designated as *Stage 1-REM*. On occasion, usually in connection with polysomnograms recorded from study subjects taking sedative-hypnotics (e.g., flurazepam), *Stage 2-REM* would also occur.

The Standardized Manual (1968)

As sleep research progressed, a problem began mounting. Technique and terminology could differ radically from one sleep center to the next; for example, REM sleep was also called paradoxical sleep, desynchronized sleep, active sleep, D sleep, and even unorthodox sleep. To alleviate this difficulty, an ad-hoc committee was formed by members of the sleep research society to develop *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects*.[17] This committee, chaired by Drs. Allan Rechtschaffen and Anthony Kales (thus the manual is consequently often called the *R&K*) was composed of a veritable pantheon of sleep luminaries, including William C. Dement, Michel Jouvet, Bedrich Roth, Laverne C. Johnson, Howard P. Roffwarg, Ralph J. Berger, Allan Jacobson,

Lawrence J. Monroe, Ian Oswald, and Richard D. Walter. This group developed a standardized set of examples and rules for scoring sleep stages.

Recording Montage

The R&K specifies that sleep is scored from a monopolar centrally derived (either C3-A2 or C4-A1) EEG tracing and EOGs recorded from the outer canthus of each eye (also referenced to A2). A fourth channel, EMG from the submentalis (chin), is also specified for detecting changes in the level of muscle tone. Using these four channels, rules are provided for classifying each epoch into wakefulness or one of five possible sleep stages.

Sleep Staging

According to the R&K, *stage W* (wakefulness) is scored when the EEG for more than 50% of an epoch contains alpha activity (while eyes are closed). For the small percentage of individuals who do not produce EEG alpha activity, blinking, high EMG, fast activity (EEG beta activity), and the absence of theta activity or vertex sharp waves may aid differentiation between wakefulness and sleep. *Stage 1* is scored when alpha comprises less than 50% of an epoch and the low-voltage, mixed-frequency EEG-EOG tracings do not contain K complexes, spindles, or REMs. Vertex sharp waves and slow rolling eye movements may be present. *Stage 2* is scored when there are sleep spindles and/or K complexes but high-amplitude delta and slow waves (≥75 μV) comprise less than 20% of the epoch's duration. *Stage 3* is scored when an epoch contains 20–50% delta or slow waves that are 75 μV or greater. *Stage 4* is scored when an epoch contains more than 50% delta or slow waves that are 75 μV or greater. *REM sleep* is scored when REMs and muscle atonia accompanying a stage 1 EEG pattern. Epochs could also be scored as *movement time* (MT) when artifact obscured the majority of the tracing and the preceding and following stage differed. In general, MT could be invoked when it was not possible to classify the epoch as W, 1, 2, 3, 4, or REM.

These stage scoring rules were not very different from existing systems of the day. Most laboratories were using some variant of the Dement-Kleitman system or the Williams-Karacan system. The real key to the success of this project was consensus. This is not to say that participants did not disagree, argue, debate, shout, and mutter epithets—they did. In fact, legend has it (and Allan Rechtschaffen corroborates it) that, in the heat of one particularly vehement argument, one of the chairmen barred the doors, decreeing that no one could leave until consensus was reached. The genius of the group was that they understood *consensus* had to be attained. If each participant had returned to his laboratory and ignored R&K recommendations in favor of continuing their existing practice, the project would have failed.

Smoothing Rules

Perhaps the most difficult, and most poorly understood, feature of the R&K system were the 3-minute rules. These were a special set of rules used to determine the beginning and ending of an episode of REM sleep or stage 2 sleep. At first blush, one might wonder why such rules should even be needed; however, when one begins considering the multitude of scenarios that actually occur, the necessity of specific rules becomes obvious. For example, consider an epoch of REM sleep that is followed immediately by another epoch that has no eye movements or sleep spindles but has a K complex and increased muscle tone at the 21st second of the epoch. Is the epoch scored as REM sleep or as stage 2 sleep? Furthermore, suppose there were 2 epochs with stage 1 EEG (without REMs) intervening the two epochs described previously. Would they be scored as REM sleep, stage 1, or stage 2? It is for these decisions that the 3-minute rules are invoked. The rationale for scoring these epochs as REM sleep is twofold. First, the central notion about sleep stages is that there are underlying neurophysiologic “state” generators. The REM state persists “tonically” even when eye movements are not present. Therefore, REM sleep continues until there is evidence that a new set of state generators have become activated, in this case producing a sleep spindle and ending REM-related muscle atonia. The second rationale for the 3-minute rules was that they smoothed over the irregularities that often accompany state-to-state transitions. This tends to minimize some of the individual differences between subjects and thereby highlights commonalities of sleep within and between individuals. Remember, early researchers were looking to characterize *normal* human sleep and consequently focused on how similar sleep was from night to night and from subject to subject.

Interestingly, this final issue—the R&K system's tendency to maximize polysomnographic similarities and to minimize, underplay, or even obscure differences between records—became a key point of criticism and contention. The 3-minute rules systematically reduce variability, as does the intrinsic nature of time domain classification. For example, an epoch in the middle of a continuing series of stage 2 sleep epochs that had a 5-second alpha burst related to an arousal would not be represented in sleep staging. Thus, a system designed to characterized normal sleep might not perform optimally when characterizing abnormal sleep. Indeed, the authors would wager that the vast majority of sleep studies conducted around the world tonight will be performed for clinical purposes. When these recordings are interpreted, their differences from normal sleep will be emphasized. Therefore, a recording and scoring system maximizing differences, rather than commonality, would be advantageous.

However, most of the differences observable on the multitude of polysomnograms recorded tonight for clinical purposes will be detected using channel tracings that were never even part of the R&K system. That is, arousals will be detected using occipitally derived EEGs; apneas, hypopneas, oxygen desaturations, and abnormal breathing patterns will be detected using airflow, effort, and oximetry channels; leg movements will be scored from anterior tibialis EMGs; and cardiac arrythmias will be noted from electrocardiographic recording. These all represent *extensions to* and not *modifications of* the R&K system. Thus, the main flaw, if it is a flaw, in the R&K system is omission of terminology, recording technique, and scoring systems for things other than “…Sleep Stages of Human Subjects,” the stated purpose.

The American Academy of Sleep Medicine Scoring Manual (2007)

In their preface, the American Academy of Sleep Medicine (AASM) Scoring Manual Steering Committee writes with reference to the R&K system, "…the rapidly emerging field of sleep medicine requires a more comprehensive system of standardized metrics that considers events occurring outside of normal brain activity."[18] In the almost 4 decades between these two publications, there certainly had been attempts to bridge the gaps. These include specific scoring guidelines developed by independent research groups and by fully sanctioned clinical society task forces. Terminology, technique, and scoring criteria have been published for CNS arousals, respiratory events, periodic limb movements, teeth grinding, middle ear muscle activity, sleep-related erections, and cyclic alternating patterns in the EEG. The AASM, however, reasoned that it would be helpful to bring the most relevant elements of clinical sleep methodology under one roof and into a single source book. This also offered an opportunity to apply principles of "evidence-based medicine" and the Rand/UCLA Appropriateness Method to decision making about recommended and optional guidelines. The project also offered the possibility for comprehensiveness, simplifications, and long-overdue minimum specification regarding computerization. In this section (to maintain the chapter's organization), we only review changes related to sleep staging. Other AASM recommendations follow in their appropriate sections.

Recording Montage

When it comes to recording methods for sleep staging, the expression *plus ca change, plus ca meme chose* couldn't be more appropriate. The "new" AASM recommended recording montage for sleep stage scoring includes frontal, central, and occipital EEG (see the section describing the Williams and Karacan [1959] system)—specifically, F4-M1, C4-M1, and O2-M1 with backup electrodes placed at F3, C3, O1, and M2. An alternative to this fully monopolar-based montage is also sanctioned: Fz-Cz, Cz-Oz, and C4-M1. Backup electrodes include placements at Fpz, C3, O1, and M2. The recommended eye movement recording remained the same as in R&K (with the designation "E" for "eye" rather than ROC and LOC [right outer canthus and left outer canthus] and the more accurate "M" designating mastoid reference behind the ear rather than the older designation "A"). Thus, the recommended eye movement recording montage is E1-M2 and E2-M1, with E1 placed 1 cm below the LOC and E2 placed 1 cm above the ROC (or vice versa). The 1-cm vertical displacement of LOC and ROC is to provide some ability to detect nonhorizontal eye movements. An alternative montage is offered for better detection of vertical eye movements (E1-Fpz and E2-Fpz, where both E1 and E2 are placed 1 cm below the outer canthus of each eye).

Sleep Staging

The AASM's updated scoring system firmly establishes epoch length at 30 seconds and has the scorer assign a stage to each epoch. In general, when an epoch contains features of more than one stage, the classification represents the stage characterizing the majority of that epoch. Consistent with previous systems, *stage W* is scored when alpha activity comprises more than 50% of the epoch. In the absence of clear alpha activity, wakefulness can be identified by eye blinks, saccadic eye movements (consistent with reading), or conjugated saccadic eye movements associated with high muscle tone. *Stage N1* predominately concords with R&K and Williams-Karacan stage 1 sleep. It is marked by theta activity, alpha slowing, vertex sharp waves, and slow eye movements. None of these features are required; however, one or more usually occur. The low-voltage, mixed-frequency background with theta activity in the absence of slow waves, sleep spindles, K complexes, and REMs is scored as stage N1 (Fig. 18-9). *Stage N2* epochs are recognized by the presence of a sleep spindle or K complex and the absence of significant delta activity. Significant delta activity is when 75 μV or more of frontally recorded delta activity lasts for more than 20% (6 seconds) of an epoch (Fig. 18-10). Epochs containing significant delta activity should be scored as *stage N3* (Fig. 18-11). Since stage N3 encompasses both stage 3 and stage 4, stage 4 has been eliminated. The AASM manual allows for using a bipolar montage (Fz-Cz) but does not indicate how amplitude criteria should be adjusted (if amplitude criteria are not adjusted, less N3 would be scored from the tracing of Fz-Cz activity). *Stage R* represents REM sleep (and will likely still be called REM sleep by most people). Stage R is scored when there is low-voltage, mixed-frequency EEG, low chin EMG levels, and REMs (Fig. 18-12). Finally, stage MT has been eliminated. Table 18-1 summarizes the EEG-EOG-EMG characteristics of each sleep stage.

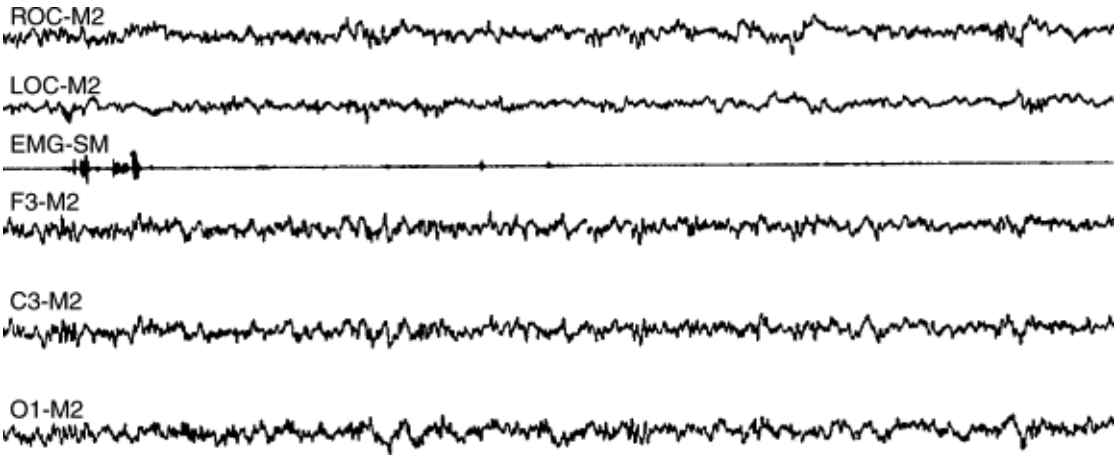

FIGURE 18-9 Sleep stage N1—a 30-second epoch of sleep classified as stage N1 that was recorded and scored according to the AASM scoring manual (C3, left central; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

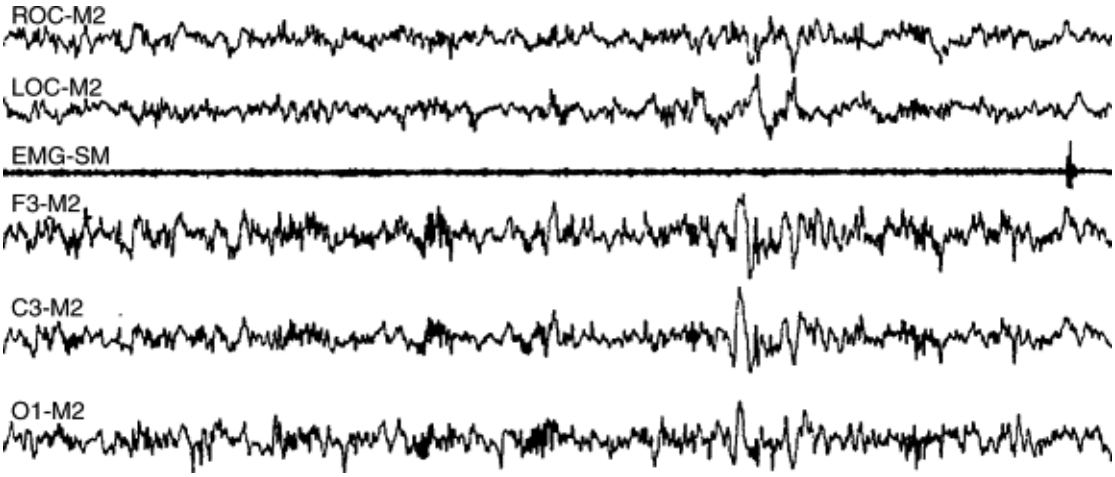

FIGURE 18-10 Sleep stage N2—a 30-second epoch of sleep classified as stage N2 that was recorded and scored according to the AASM scoring manual. (C3, left central; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

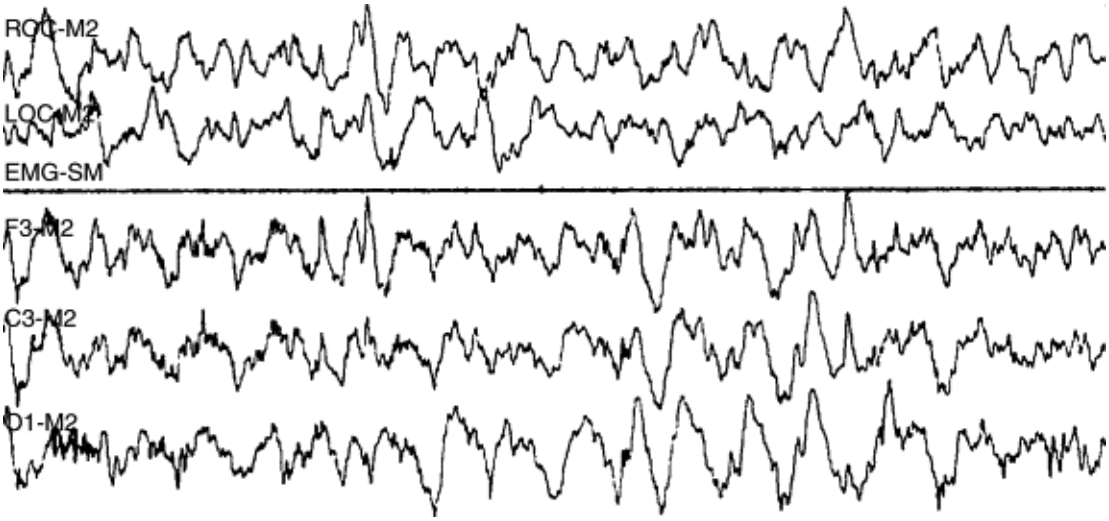

FIGURE 18-11 Sleep stage N3—a 30-second epoch of sleep classified as stage N3 that was recorded and scored according to the AASM scoring manual. (C3, left central; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

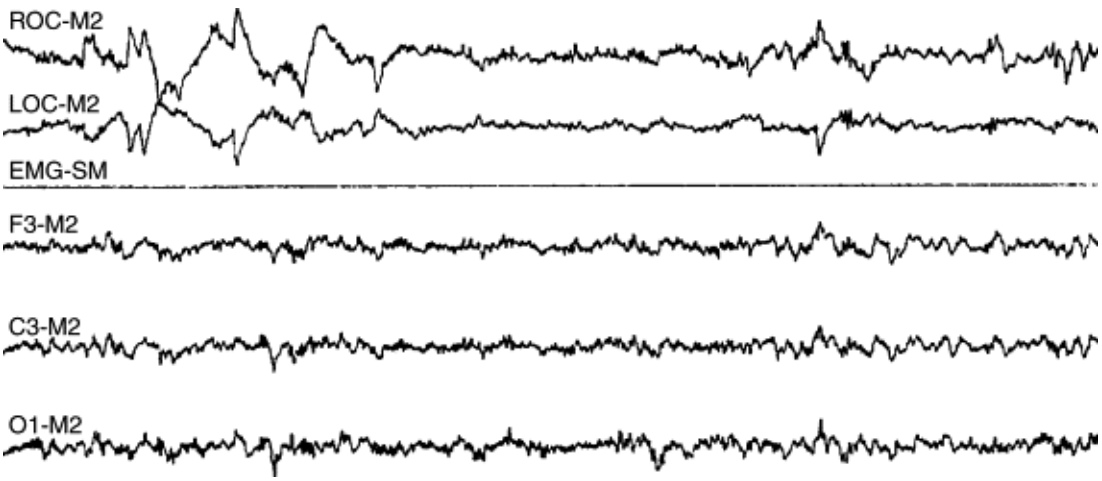

FIGURE 18-12 Sleep stage R—a 30-second epoch of sleep classified as stage R that was recorded and scored according to the AASM scoring manual. (C3, left central; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

TABLE 18-1 -- Electroencephalographic, Electro-oculographic, and Electromyographic (EMG) Characteristics of Each Sleep Stage

Stage*	Brain Wave Activity	EMs [†]	EMG	Delta	Spindle	Alpha	Beta	Mentation
W	Predominant alpha activity; ≥15 sec alpha activity in epoch	S & R	++	-	-	++	+	Thoughts
N1	Alpha activity replaced by low-voltage, mixed-frequency activity; vertex sharp waves	S	↓	-	-	+	-	Hypnagogic
N2	Sleep spindles and K complexes in background EEG; overall <6 sec delta activity in epoch	-	↓	+	++	-	-	-
N3	General EEG synchrony and ≥6 sec delta activity in epoch	-	↓	++	+	-	-	-
R	Low-voltage, mixed-frequency activity; sawtooth theta activity	R	-	-	-	+	+	Dreams

* R, rapid eye movement sleep; W, wakefulness.

† EMs, eye movements: R, rapid; S slow.

++ prominent defining feature;

+ increased level often seen;

- absent under normal circumstances;

↓ decreased from awake state.

Smoothing Rules

Generally speaking, most smoothing rules have been eliminated. Epochs are scored according to the characteristics that make up their majority. However, because not all epochs in stage R have associated eye movements, stage R continues to be scored until a spindle, K complex, or arousal occurs or when chin EMG increases. If the majority of the final epoch containing stage R meets criteria, it is scored as stage R; otherwise it is categorized as the predominating stage. Stage R terminates with a transition to stage W or N3, an increase in chin EMG level, or an arousal that is followed by low-voltage, mixed-frequency EEG and slow eye movements. Similarly, a body movement followed by slow eye movements will terminate a stage R episode. Finally, spindles or K complexes occurring in the first half of an epoch terminates stage R. The scoring manual has a detailed set of examples to guide scoring.

Sleep Stage Changes across the Night

Regardless of which system is used for staging, the overall normal sleep stage pattern (sometimes called sleep macroarchitecture) across the night is fairly consistent. A healthy young adult good sleeper will spend 7–8 hours in bed and sleep 85–90% of that time. It may take such sleepers 5–15 minutes to fall asleep, and normal entry into sleep for an adult is through stage N1, which quickly evolves into N2. N3 usually follows and persists for some time before giving way to an episode of stage R. Usually the duration of the first stage R episode is brief (5–15 minutes) and the sleeper then goes back into N2 and possibly N3 for the next hour and a half. Stage R re-occurs at this point and is usually longer in duration than the first episode. Succeeding N-R cycles usually have less stage N3, more N2, and longer stage R duration as the sleep period progresses. Thus, one could generalize a prototypical night's sleep as having most of the stage N3 in the first third of the night and most of the stage R in the second half of the night. The stage R comes in 4–6 discrete episodes occurring approximately 90–100 minute apart (Fig. 18-13). Overall, stage N2 will account for approximately half of the night's sleep and stage R will account for another fifth to quarter.[19] Stage N1 should encompass less than 5% of total sleep time, distributed mainly at sleep-wake transitions. The remainder of sleep time will consist of stage N3. Men and women will not differ much in sleep stage percentages; however, women may have slightly more stage N3 than men as age advances.

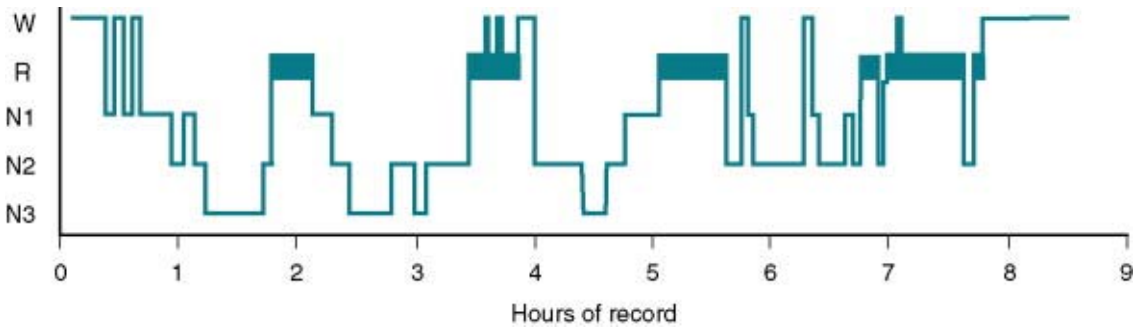

FIGURE 18-13 Sleep stage histogram for a normal, young adult subject. (N1, sleep stage 1; N2, sleep stage 2; N3, sleep stages 3+4; R, rapid eye

movement sleep; W, wakefulness.)

Sleep Stage Changes as a Function of Age

Over the life span, total sleep time gradually decreases. Stage N3 begins decreasing after adolescence. This trend continues as a function of age, and stage N3 may completely disappear in elderly individuals. REM sleep duration declines spectacularly at life's beginning, decreasing from more than 50% at birth to 20–25% at adolescence.[19] For the next 50 years, stage R percentage remains stable. However, after 65 years stage R may begin to decline again (Fig. 18-14).

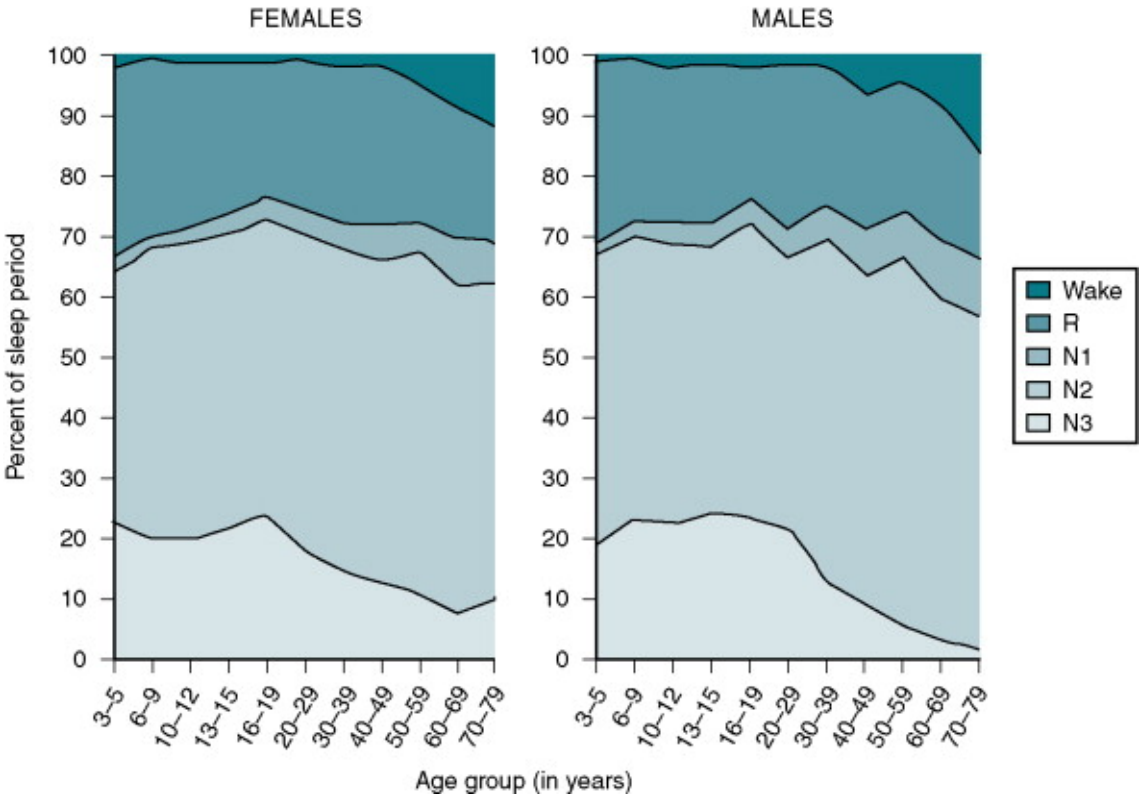

FIGURE 18-14 Sleep stage changes as a function of age. (N1, sleep stage 1; N2, sleep stage 2; N3, sleep stages 3+4; R, rapid eye movement sleep.) Data points extrapolated from Williams et al.[16]

The overall decline in total sleep time is also associated with increasing sleep fragmentation by CNS arousals and awakenings. Some of the sleep disturbances are produced by accumulated pathologies that adversely affect sleep (e.g., arousals and awakenings from nocturnal pain). However, some sleep disturbances are nonspecific or of unknown etiology and may relate to a age-related deterioration of the underlying physiologic sleep mechanism. Regardless of cause, many elderly will spend more time in bed but less time sleeping.

SLEEP DISTURBANCES AND INSTABILITY

Sleep Stage Measures

Polysomnography offers the opportunity to objectively assess sleep disturbances and instability. While it is well known that sleeping in the laboratory can produce sleep disruption, this “first night effect” quickly dissipates and successive nights can be used to evaluate sleep integrity. One approach is to calculate parameters from sleep stages. Some of the parameters proven useful in clinical trials and investigative sleep studies include latency to sleep, latency to persistent sleep, sleep efficiency, total sleep time, number of awakenings, wake time after sleep onset, the number of ascending stage 1 occurrences, and the total number of sleep stage shifts. When comparing recordings of different durations (total recording times), indexed measures are needed on parameters other than latencies to sleep. For example, awakenings per hour can be used as a indexed value for number of awakenings. By contrast, when clinical trials fix the time in bed as a constant, untransformed measures can be used directly (e.g., wake after sleep onset).

ASDA and AASM Arousal Scoring

The need for a technique to score brief CNS arousals became apparent when polysomnography began to be used clinically. A patient might have several hundred obvious sleep disturbances; however, they were not reflected in sleep stage scoring because their duration was insufficient to alter sleep stage. To qualify as an awaking, alpha activity must present for half the duration of an epoch—that is, be 15 seconds or longer in duration. Thus, the 5-second alpha intrusion routinely occurring at the termination of a breathing event or leg movement falls under the radar of sleep stage scoring systems, and therefore goes unnoticed. To correct this situation, an AASM (at that time called the American Sleep Disorders Association [ASDA]) task force was formed to develop criteria for scoring CNS arousals.[20] Derived largely from the work at Henry Ford Hospital, arousals were defined in terms of “EEG speeding.” This encompassed abrupt shifts from sleep (of at least 10 seconds' duration) to faster EEG activities (including theta, alpha, and beta). In REM sleep, the “EEG speeding” also had to be accompanied by increased activity in the submentalis EMG recording (Fig. 18-15). The minimum duration of “EEG speeding” was set at 3 seconds. This duration was based on what the members of the task force could reliably score by hand. It was recognized that computerized signal processing systems might well be able to reliably score shorter events; however, the rules being developed at that time were for visual scoring.

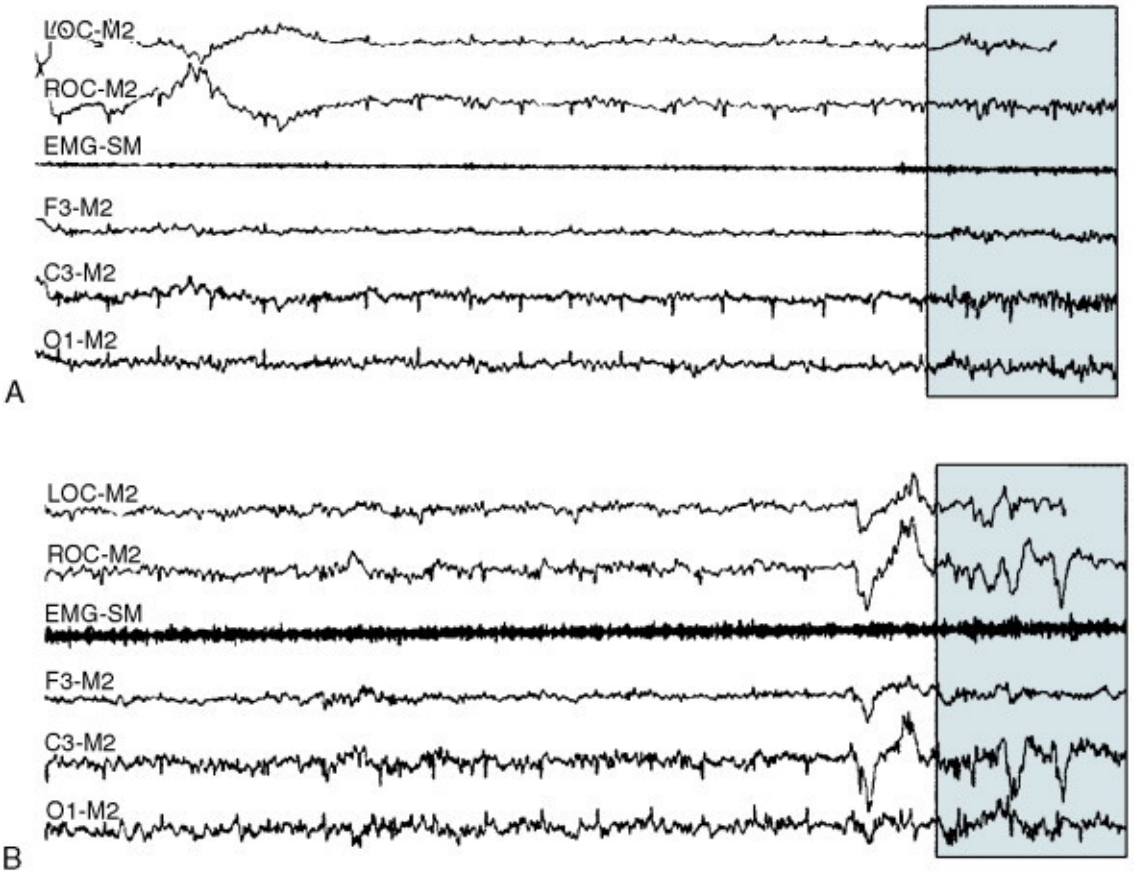

FIGURE 18-15 Examples of CNS arousals from REM sleep **(A)** and NREM sleep **(B)**. (C3, left central; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

The overall scoring rules for CNS arousals were not changed by the AASM Manual Arousal Scoring task force. However, the original 11 rules that defined what *did* and *did not* qualify as an arousal were simplified. This simplification was expressed as a single statement of what an arousal *is*, with two explanatory notes.

Cyclic Alternating Pattern

The cyclic alternating pattern (CAP) is a dynamic alternation of EEG bursts and quiescence that can be visualized during polysomnography.[21] As a technique to investigate sleep, CAP has been championed by Drs. Terzano and Parrino, whose steadfast interest and scholarly work with CAP has drawn other researchers' attention to this EEG activity. CAP consists of an active *A phase* that can be a vertex sharp wave; a K complex; a K-alpha; a burst of high-amplitude, low-frequency waves; a burst of polymorphic waves; or a burst of high-amplitude theta or alpha activity. These *A phases* are followed by quiescent *B phases*. These transient electrocortical events are distinguishable from background activity and repeat in a cycle, usually every 20–40 seconds (Fig. 18-16). There are three types of A phases. The first does not meet AASM criteria for arousal (A1). The second includes enough alpha activity to sometimes qualify as an arousal (A2); however, classification is determined by alpha percentage of the overall A-phase duration (i.e., desynchronized portion must be between 20% and 50%). Thus, whether an A2 meets AASM arousal criteria depends on whether the alpha-wave portion is 3 or more seconds in duration. Finally, the third type of A phase meets arousal criteria more than 95% of the time.[22]

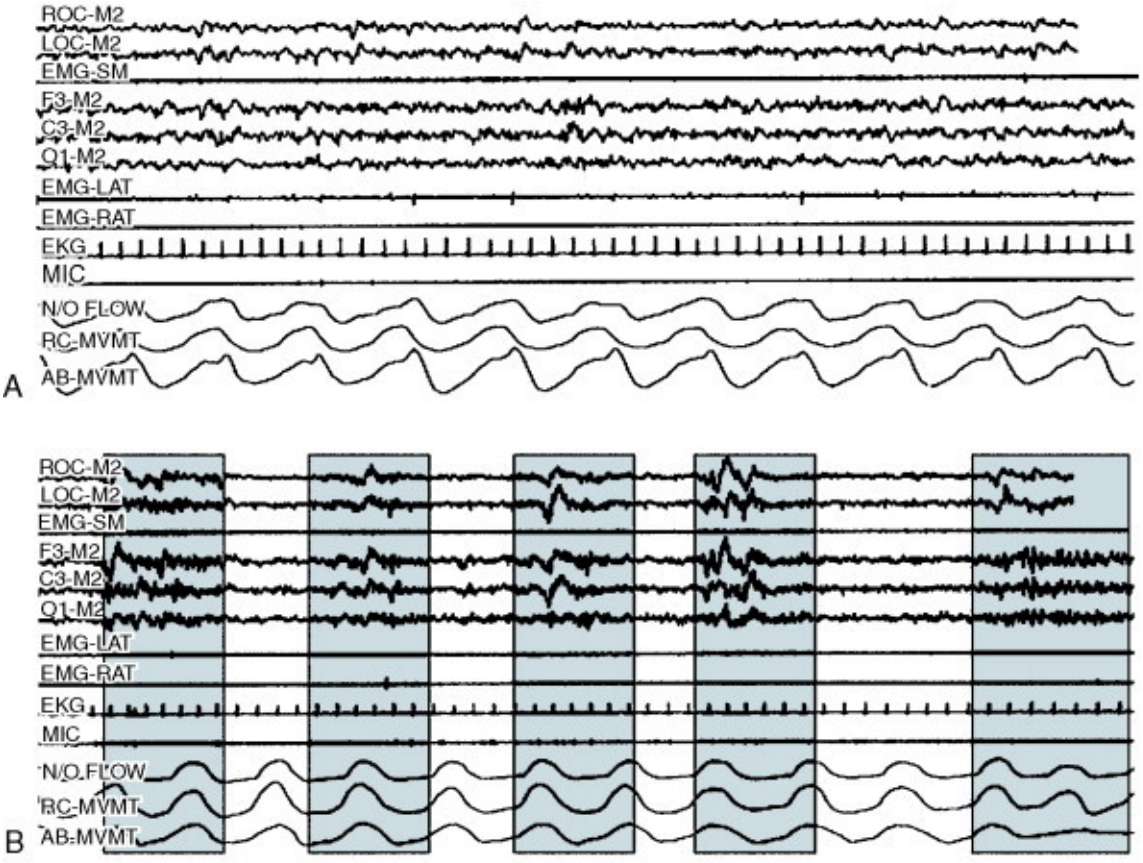

FIGURE 18-16 Samples of non-CAP activity **(A)** and CAP activity **(B)**. (AB-MVMT, abdominal movement; C3, left central; CAP, cyclic alternating pattern; EKG, electrocardiogram; EMG-LAT, electromyogram–left anterior tibialis; EMG-RAT, electromyogram–right anterior tibialis; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; MIC, microphone for snoring; N/O FLOW, nasal-oral airflow; O1, left occipital; RC-MVMT, rib cage movement; ROC, right outer canthus.)

It is theorized that the CAP's slow component represent a human correlate of higher brain reinforcement of subcortical antiarousal gating mechanisms. The thalamus appears to play an important role in preserving sleep by inhibiting transmission of incoming peripheral stimuli. The Wake Inhibition Sleep Preservation (WISP) hypothesis postulates that, when the protective ascending thalamic gate fails to block incoming activity, a descending cortical response can help reinforce the gate (as has been demonstrated in animals by Steriade and colleagues[23]). Sometimes the WISP system succeeds in preserving sleep and is reflected by a CAP A1. By contrast, the WISP system's attempt to preserve sleep will sometimes fail, and this process is seen as a CAP A2 or A3 (or AASM arousal). Sequences of CAP A1 in such a conceptualization would represent sleep instability (as often seen during sleep right before REM sleep occurs). By contrast, many CAP A2s and virtually all CAP A3s represent a failure of the system to preserve sleep (which would explain why the CAP rate is higher among individuals with insomnia) (Figs. 18-17, 18-18, and 18-19).

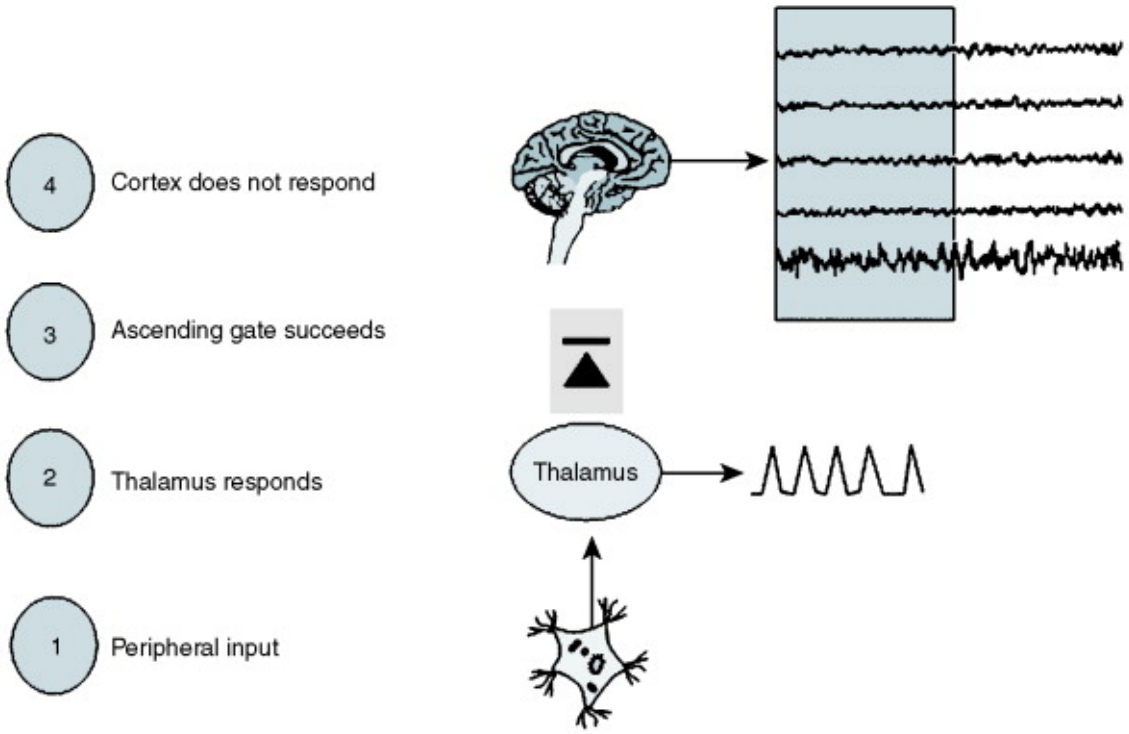

FIGURE 18-17 Dynamics of the Wake Inhibition Sleep Preservation (WISP) system during stable sleep.

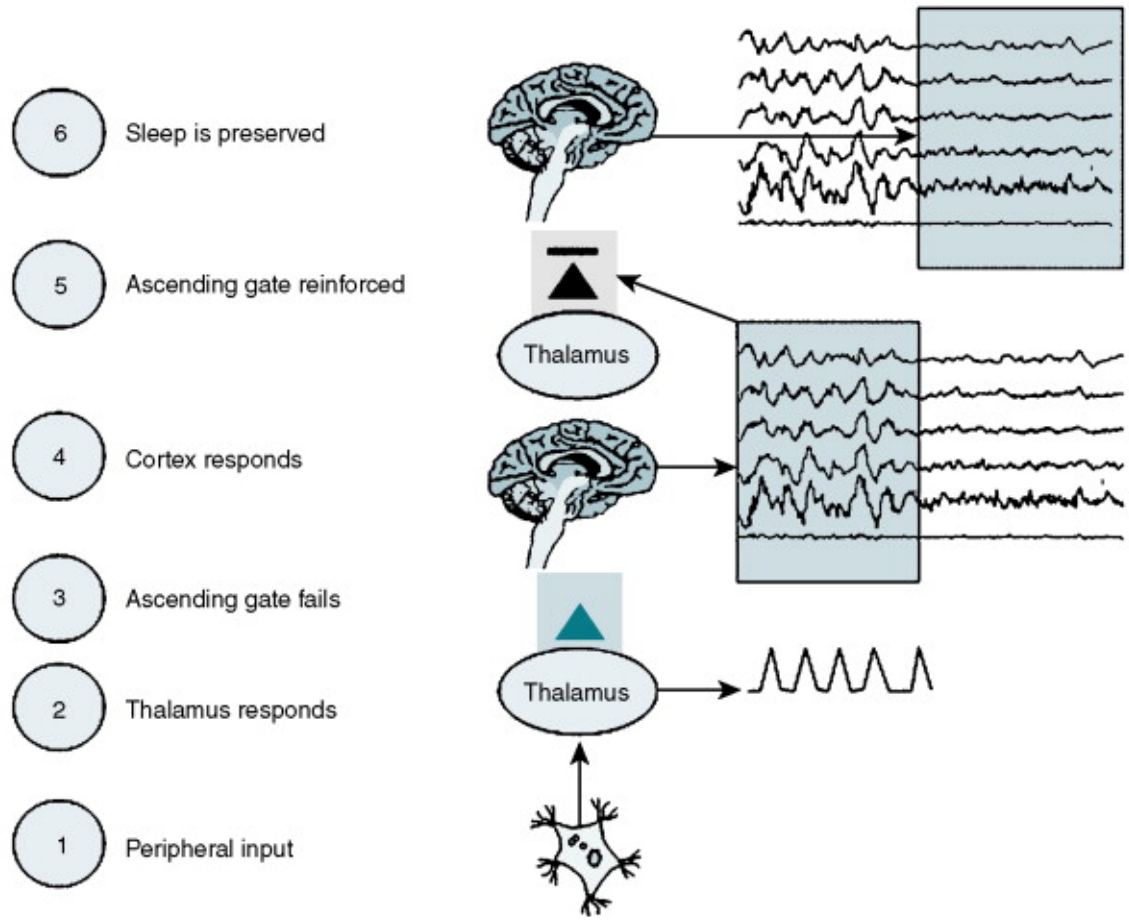

FIGURE 18-18 Dynamics of the Wake Inhibition Sleep Preservation (WISP) system during unstable but preserved sleep.

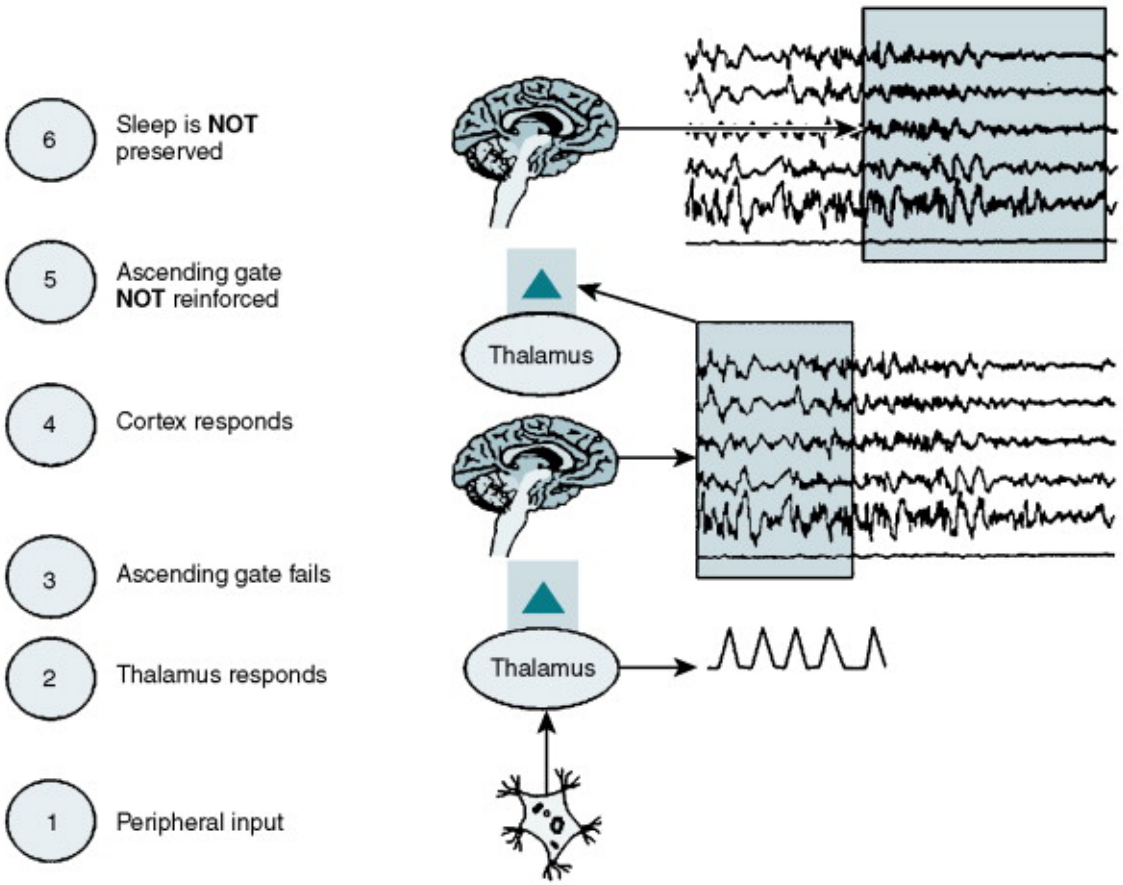

FIGURE 18-19 Dynamics of the Wake Inhibition Sleep Preservation (WISP) system leading to disturbed sleep.

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MOVEMENT

Leg and Limb Movement

In its simplest form, the movement that occurs in individuals with periodic limb movement disorder (PLMD) is a Babinski-like extension of the great toe. This movement, however, can sometimes also involve flexion of the ankle, the knee, and occasionally the hip and/or upper extremities. To detect these movements, two surface electrodes are placed on each leg on the anterior tibialis muscle (2–4 cm apart). Electrodes can be placed on the gastrocnemius muscle to assist with determining whether movement is artifact related; however, most sleep laboratories only use the anterior tibialis electrode placement. Both left and right legs should be recorded either on a single channel or preferably on separate channels.

The scoring rules for PLMD have not changed much since the technique was described in detail by Coleman.[24] An ASDA Task Force refined the rules slightly and developed standardized definitions and illustrations.[25] The recent AASM manual reasserted a distilled list of the rules and incorporated PLMD scoring into the larger domain of “Movement Scoring Rules.” These recent leg movement rules are partly derived from the guidelines developed by the World Association of Sleep Medicine–International Restless Legs Syndrome Study Group.[26] The AASM Scoring Manual[18] provides the following five rules for defining a leg movement event:

1. The movement must be one-half second or longer. This rule is unchanged from the ASDA guidelines.
2. The movement's total duration must be 10 seconds or less. This criterion is increased from a 5-second cutoff previously stipulated.
3. The minimum amplitude to score a leg movement must be at least an 8-μV increase over resting EMG level. This differs from the previous rule specifying that the burst had to be at least 25% greater than calibrated movements recorded during behavioral maneuvers during the polysomnographic presleep “biocalibration.”
4. Following logically from the requirement that leg movement detection hinges on a minimum 8-μV increase above resting level, the point at which the EMG exceeds the 8-μV threshold marks the leg movement onset.
5. Finally, the leg movement's end point occurs when EMG level drops to within 2 μV or less of the EMG resting level.

While it is true that an EMG, as recorded during polysomnography, is an uncalibrated signal, these adopted amplitude criteria have been successfully used to program automated leg movement detection that has a high degree of accuracy.

The AASM Scoring Manual continues with criteria for a periodic leg movement series or episode. A series of periodic leg movements must have a sequence of four or more movements (criteria are unchanged). The minimum interval between leg movements must be 5 seconds or more, while the interval maximum is 90 seconds. Leg movements can occur in association with arousal or without any sleep disturbance (Fig. 18-20). Movements of both of a person's legs (if recorded) that are separated by less than 5 seconds are considered a single movement (occurring in both legs). Alternating leg muscle activation must come in four groupings or more, have a minimum frequency of 0.5 cycles/sec and a maximum frequency of the alternating EMG bursts is 3.0 cycles/sec.

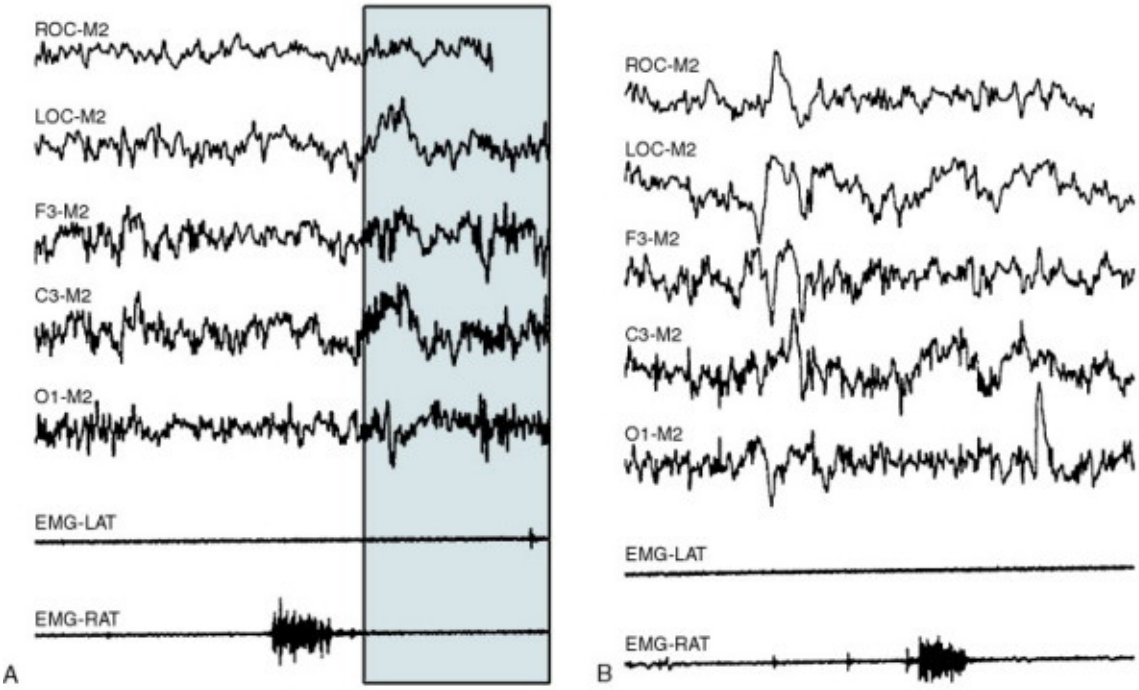

FIGURE 18-20 Leg movements with and without a CNS arousal. **(A)** A burst of right leg electromyographic activity immediately followed by an arousal (*shaded area*). **(B)** An electromyographic burst of similar magnitude without accompanying CNS arousal. (C3, left central; EMG-LAT, electromyogram–left anterior tibialis; EMG-RAT, electromyogram–right anterior tibialis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; O1, left occipital; ROC, right outer canthus.)

Other

A host of other movements are described in the AASM scoring manual. These include rules for defining hypnagogic foot tremor, excessive fragmentary myoclonus, sleep bruxism, REM sleep behavior disorder, and rhythmic movement disorder (see AASM scoring manual[18] for details).

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Sleep Disorders Medicine
Basic Science, Technical Considerations, and Clinical Aspects

SLEEP-RELATED BREATHING

Once it became clear that sleep-related breathing disorders (SRBDs) did not exclusively occur in hypoventilating morbidly obese patients, respiratory monitoring during polysomnography quickly became routine. The American Thoracic Society in conjunction with the ASDA published joint guidelines concerning medical outcomes research on sleep-disordered breathing but did not address the nitty-gritty of recording and scoring technique.[27] Thus, for a very long time there was little detailed guidance in this area, and the methods described in Bornstein's (aka, Sharon Keenan)chapter[28] in Guilleminault's *Sleeping and Waking Disorders: Indications and Techniques* became a de facto standard. It was not until many years later, and well after the book had gone out of print, that the Chicago Group's recommendations were published.[29] Even with that, their recommendations were principally for research rather than for standard clinical routine. In the meantime, all manner of controversy surrounding recording technique, nomenclature, and interpretation had surfaced.

Currently, the vast majority of patients evaluated in clinical sleep laboratories are referred for assessment of SRBDs. Ironically, the recording, scoring, and interpretation of sleep-related breathing data is probably the least standardized part of polysomnography. For years, Medicare used to consider 30 episodes of sleep apnea as the sole cutting score for sleep-disordered breathing; hypopnea and oxygen desaturation events were not even considered. In 2001, Medicare recognized hypopnea as a clinically significant sleep-related breathing event if it was associated with 4%, or greater, oxygen desaturations.[30] Unfortunately, Medicare did not recognize hypopneas that produce CNS arousals and sleep fragmentation. In the sleep and respiration methodologic arena, the AASM scoring manual was long overdue. Even though it leaves several issues unresolved, this professional organization–sanctioned guideline is a welcome and critically needed document.

Recording Technique

Four key aspects of breathing are measured during polysomnography: airflow, respiratory effort, blood oxygenation, and CNS arousal or awakening. Airflow can be measured using thermistors, thermocouples, nasal pressure transducers, capnographs, microphones, calibrated inductance plethysmography, or with a pneumotachometer. Respiratory effort can be measured with piezo-electric respiratory belts, inductance plethysmography, esophageal pressure devices, strain gauges, or intercostals EMG electrodes. Blood oxygenation is measured with oximetry, and CNS arousals and awakenings are scored from occipital and central EEG.[31]

From this array of transducers, devices, apparatus, and approaches, the AASM scoring manual[18] recommends the following methods as primary. Alternative sensors are for use when the recommended signal is not reliable; however, they are not recommended as alternatives for primary use. The AASM scoring manual recommendations are summarized as follows:

1. To identify apnea, use a thermal sensor placed at the nose and mouth (alternative sensor is the nasal pressure tranducer).
2. To identify hypopnea, use a nasal pressure transducer (alternative is inductance plethysmograph or oronasal thermal sensor).
3. To identify effort, use either an esophageal manometer or inductance plethysmograph (alternative sensor is intercostals EMG).
4. To identify O_2 desaturations, use a pulse oximeter with its signal averaged over 3 seconds or less.

To identify CNS arousal or awakening, no recommendation is made. However, it is probably safe to assume that staging and arousal scoring standard techniques would be recommended.

Terminology (Types and Classification of Events)

Apnea

In essence, apnea is a cessation of airflow for 2 or more respiratory cycles. The AASM scoring manual's operational definition is a 90% or greater drop in the peak-to-trough amplitude on the nasal/oral airflow channel for 10 seconds or more. Furthermore, the amplitude reduction must persist for at least 90% of the event's duration. Apneas can be classified as central, obstructive, or mixed. A *central apnea* is scored when inspiratory effort is absent throughout the duration of the event. An *obstructive apnea* is scored when there is continued or increasing inspiratory effort throughout the duration of the event. Finally, *a mixed apnea* is scored when there is a lack of inspiratory effort initially followed by a resumption of an unsuccessful attempt to breathe during the later portion of the event (Fig. 18-21).

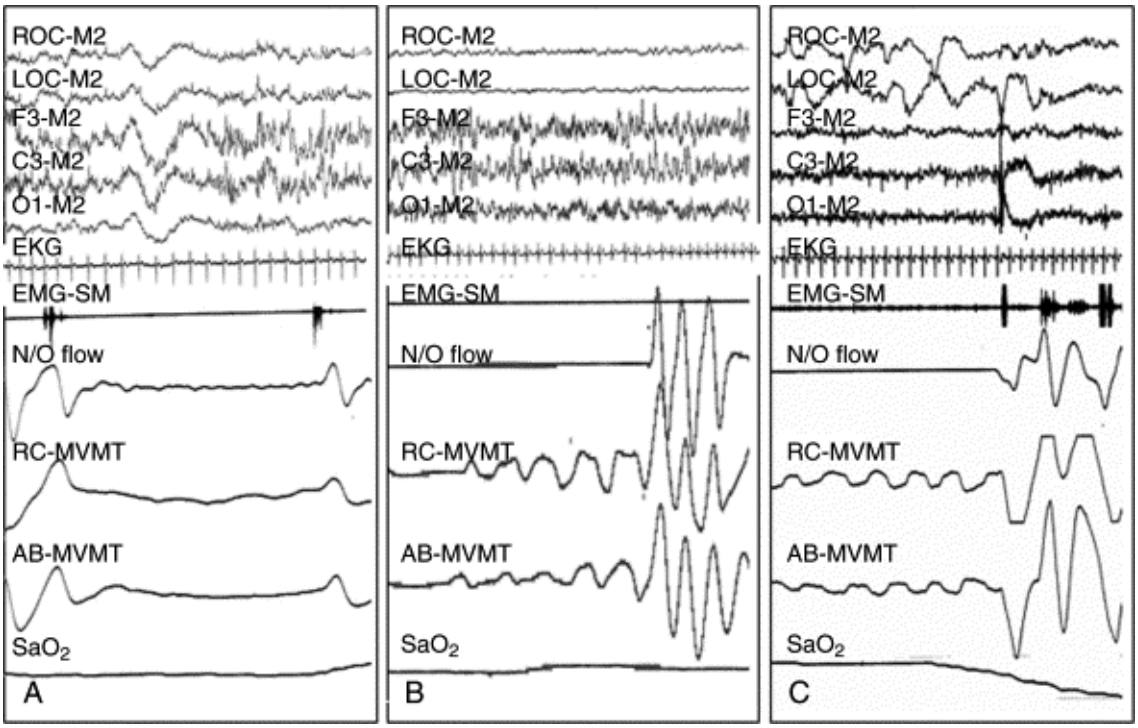

FIGURE 18-21 Episodes of sleep apnea. **(A)** A central-type episode during which the cessation of breathing is accompanied by no movement of the rib cage and abdomen (no respiratory effort). **(B)** A mixed-type episode that begins with a pause in respiratory effort (and airflow) but continues to have flow cessation even as respiratory effort begins and continues. **(C)** An obstructive-type episode in which there is a complete cessation of airflow notwithstanding an uninterrupted effort to breath. (AB-MVMT, abdominal movement; C3, left central; EKG, electrocardiogram; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; N/O FLOW, nasal-oral airflow; O1, left occipital; RC-MVMT, rib cage movement; ROC, right outer canthus.)

Hypopnea and Respiratory Effort–Related Arousals

In essence, a hypopnea is a shallow breath in which there is decreased tidal volume. Furthermore, there is nothing intrinsically pathophysiologic about a hypopnea. However, a hypopnea associated with flow limitation that ultimately produces an arousal so that the sleeper can dilate his or her airway and resume ventilation is a significant respiratory event. Additionally, a hypopnea associated with significant oxygen desaturations is medically noteworthy. Thus, it is not the hypopnea itself that is pathophysiologic but rather its consequence.

As straightforward and logical as this may seem, the operational definition for hypopnea remains controversial. One problem stems from recording technique; that is, most sleep laboratories measure flow qualitatively, and such measures do not proportionally estimate tidal volume. Therefore, couching airflow changes in terms of percentage decrease from baseline is problematic. Guilleminault et al.'s[32] original definition of hypopnea as a reduction in airflow without complete cessation of breathing adhered closely to the general principle but left open the question of how much decrease in airflow was minimally required to score a hypopnea. A wide assortment of definitions were developed using different cutting scores for percentage of airflow decrease. Then in 2001, the AASM Clinical Practice Review Committee[33] defined hypopnea largely based on the definition used in the Sleep Heart Health epidemiologic studies.[34] This definition was subsequently adopted by Medicare. Hypopnea was suddenly redefined as a 10-second or longer 30% reduction from baseline in thoracoabdominal effort or airflow accompanied by a 4% oxygen desaturation. This definition completely disregarded the consequence of a hypopnea on the sleeping brain. As a result, the respiratory events (formerly call hypopnea) that were associated with CNS arousal but less than 4% desaturation were reassigned to a category of events known as respiratory effort–related arousals (RERAs). RERAs originally were a category of events that were so subtle they might not even be detectable without esophageal manometry.

The AASM scoring manual[18] provides two sets of scoring rules for hypopnea. The first is an operationalized refinement of the Medicare definition and is recommended for clinical practice. The *recommended criteria* for scoring hypopnea are met when

1. the nasal pressure signal amplitude drops by 30% or more compared to baseline,
2. the event lasts 10 seconds or more,
3. at least 90% of the event duration maintains the 30% amplitude drop, and
4. a 4% or greater O_2 desaturation occurs as a result.

The AASM scoring manual's *alternative criteria* for scoring hypopnea represent a return to the scoring used before the Medicare redefinition came about. These better operationalized criteria specify that a hypopnea is scored when

1. the nasal pressure signal amplitude drops by 50% or more compared to baseline,
2. the event lasts 10 seconds or more,

3 at least 90% of the event duration maintains the 50% amplitude drop, and
4 a 3% or greater O_2 desaturation occurs “or the event is associated with arousal.”

Although the alternative criteria provide a welcome rediscovery of the *brain's* importance to sleep-disordered breathing, there is a rub! The U.S. Government prohibits treating Medicare patients differently than other patients in one's practice. Therefore, if your sleep program provides services for Medicare, you are constrained to always use the first set of criteria. However, if Medicare is irrelevant to your practice (e.g., you practice in Tasmania), you may choose whichever rule you prefer. Another problem created by having two definitions for the same term is ambiguity, and hypopnea now has two official definitions. It would have been preferable to either add a distinguishing modifier to the word *hypopnea* or to flat out create two new words. Perhaps *desaturating hypopnea* could be used for the first definition and *traditional hypopnea* for the latter. It really does not matter what names are chosen as long as they differ. One could call the first one George and the other Gracie; at least we would understand what each means without having to read the footnotes (Fig. 18-22).

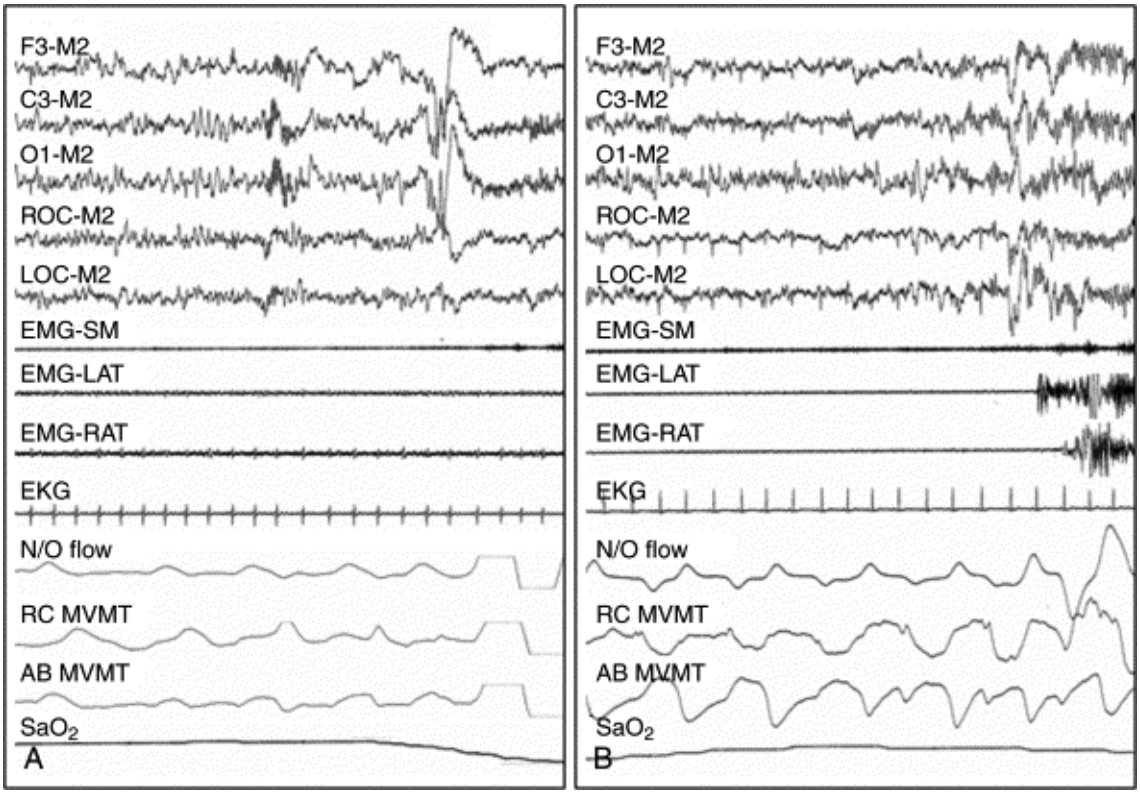

FIGURE 18-22 Episodes of sleep hypopnea. The AASM scoring manual defines two sets of rules for scoring hypopnea. **(A)** A hypopnea episode that meets criteria of the Recommended rule that requires a 4% drop in oxygen saturation. **(B)** An episode that would not be scored as a hypopnea using the Recommended rule but would qualify according to the Alternative rule (note the prominent CNS arousal terminating the event and disrupting sleep). (AB-MVMT, abdominal movement; C3, left central; EKG, electrocardiogram; EMG-LAT, electromyogram–left anterior tibialis; EMG-RAT, electromyogram–right anterior tibialis; EMG-SM, electromyogram-submentalis; F3, left frontal; LOC, left outer canthus; M2, right mastoid; N/O FLOW, nasal-oral airflow; O1, left occipital; RC-MVMT, rib cage movement; ROC, right outer canthus; Sao_2, arterial oxygen saturation.)

Apnea, Hypopnea, and RERA Summary Statistics

Traditionally, expression of nightly totals for sleep-related respiratory events included calculation of an apnea index (AI) and apnea-hypopnea index (AHI). The AI is the number of apnea episodes per hour of sleep, and the AHI is the number of apnea plus hypopnea episodes per hour of sleep. After the Medicarization of hypopnea took place, another summary statistic was added to include those hypopneas that were now tabulated as RERAs. Thus, a respiratory disturbance index (RDI) is routinely reported and is the number of apnea, hypopnea, and RERA episodes per hour of sleep. The irony here is that the acronym RDI originally stood for “respiratory desaturations index,” which was created to be distinct from the AHI. At some point, clinicians began using RDI synonymously with AHI. In the end, with the Medicare definition requiring 4% desaturations, the AHI actually became a desaturations index and the RDI is now used to indicate that non-desaturating events are also included. Therefore, if the first set of hypopnea criteria are used for scoring, the AI, AHI, and RDI should be reported. If the second definition is used, reporting the AI and AHI should be sufficient. Note that the AASM scoring manual does not discuss tabulating the RDI. Why is this important? When one attempts to titrate positive airway pressure and the first (Medicare-type) definition is used, the AI and AHI are insufficient to determine optimal pressure; the RDI must be calculated for each pressure to compare outcomes.

RECORDING EQUIPMENT AND ADVANCING TECHNOLOGIES

As is often the case with new technology, what we envision and what evolves travel two separate paths. Take, for example, the videophone, a device always right at home in our conception of what the future will look like. However, even though we've long had the technical capability to make videophones, and notwithstanding several attempts to market such devices, videophones are absent from the landscape. By contrast, the forerunner of our modern-day FAX machine was judged by Xerox to be not worth developing because no one would want one. Despite this prediction, the FAX machine became an integral component of business communication and has remained so for more than 2 decades.

Our expectations with respect to computerized sleep recording were similarly misguided. When thinking about digital polysomnography, we were mainly focused on the computer's potential for effortlessly detecting waveforms, tabulating sleep-disordered breathing events, and automatically scoring sleep stages. However, these systems found their way into our laboratories for other reasons entirely. As mundane as it may seem, the principal driving force behind sleep laboratory automation was the elimination of paper tracings. The paper polysomnograph is all but extinct. The last major polysomnographic paper manufacturer has closed its doors. Nonetheless, computerized sleep systems have yet to extract and organize the seemingly boundless wealth of physiologic information in new, clinically useful ways or provide reliable automatic sleep staging.

Digital Polysomnography

Technology constantly evolves, and sometimes the elegance of the older technology is sacrificed for greater efficiency and/or reduced cost. When sailing vessels declined in favor of motorized ships, many bemoaned the loss of such graceful vessels as clippers and caravels on the seas and their replacement by noisy, smoke-belching machinery. Much like that switch from sails to steam, the evolution from traditional paper polysomnographs to today's digital systems has been marked by both progress and regression. One of the serious hurdles facing early digital sleep system developers was the absence of guidance from scientific and clinical organizations. No standards were set a priori; therefore, developers had to rely on advice from individuals who appeared to have expertise in both engineering and sleep medicine. Sometimes the advice was good and sometimes not. To complicate matters, until this past decade, the computational and storage resources of microcomputers were only marginally up to the task. Video display resolution and speed were problematic unless nonstandard devices were integrated. Removable mass storage was expensive and finicky.

All that has changed. Today, CPUs are lightning fast, gigabytes of memory are standard, high-definition screens are affordable, and super-high-capacity DVD writers are ubiquitous. Free from technological limitations that shackle development, manufacturers have developed and marketed a dazzling array of digital polysomnographic systems. The market, however, is now being driven more by cost than quality, and decisions about purchase are being made by administrators rather than sleep specialists. The result is an ever greater need for guidance from the scientific and clinical community, and the AASM has responded. A digital polysomnography task force was appointed, co-chaired by the senior author (M.H.) and Thomas Penzel, and the process began. One of the AASM scoring manual's goals was to provide recommendations for digital polysomnography. Important progress was made on this front even though we were not able to address all of the relevant topics.

Sorely needed were recommendations concerning signal quality and resolution. It was easy to agree that at least 12 bits were needed to represent amplitude (this provides a number line from 0 to 4095 or, if 2s-complement is used to represent positive and negative values, from -2048 to +2047). This resolution is enough to adequately cover the plus or minus (approximately) 2.5-V range with regulated current (I_{REG}). Moreover, it makes it likely that minimum discernable voltage differences (exceeding the level of noise) will be detected. The recommendations concerning temporal resolution were more difficult to formulate, but after much discussion and significant compromise, we decided to provide both minimal and desirable sampling rates for each recording channel (Table 18-2). Sampling rates were set high enough to accurately reconstruct waveforms and provide enough data to potentially overcome frequency aliasing (if printed in high resolution). Additionally, frequencies appropriate for actual or digitally simulated high- and low-pass filter settings for each type of bioelectrical signal were determined (Table 18-3).

TABLE 18-2 -- AASM Digital Task Force Recommendations for Polysomnographic Signal Sampling Rates

Polysomnographic Signal Being Recorded	Desirable (Hz)	Minimal (Hz)
Electroencephalogram	500	200
Electro-oculogram	500	200
Electromyogram	500	200
Electrocardiogram	500	200
Airflow—thermistors and thermocouples	100	25
Airflow—nasal pressure	100	25

Respiratory effort—esophageal pressure	100	25
Respiratory effort—rib cage and abdominal movement	100	25
Snoring sounds	500	200
Oximetry	25	10
Body position	1	1

TABLE 18-3 -- AASM Digital Task Force Recommendations for Polysomnographic Signal Filter Settings for Routine Recording

	Setting (Hz)	
Polysomnographic Signal Being Recorded	Low Frequency	High Frequency
Electroencephalogram	0.3	35
Electro-oculogram	0.3	35
Electromyogram	10	100
Electrocardiogram	0.3	70
Respiration—airflow effort channels	0.1	15
Snoring	10	100

What Was Gained?

As already mentioned, sleep recordings became paperless (and inkless). Most of us do not miss lugging heavy boxes of paper around or the 5-foot-high piles of recordings leaned against the walls in corridors and hallways. Storage space was reduced from rooms to file drawers and record disposal became a trifle. There were no more pens and ink wells to clean, unclog, replace, stain carpets, and ruin clothing. Consumable supply costs plummeted. The machinery itself shrank, weighed less, and even became portable.

Digital polysomnography made data display dynamically scalable. With paper, the recording speed dictated the viewable product. There was no opportunity to expand or compress the recording (except to look at and flare the edge of the paper recording). What was recorded was what we got to see. This did mean, however, that we knew what the night technologist was seeing during the recording process. We also knew what the scorer was seeing when he or she scored each event and classified each epoch as a sleep stage.

The computerized systems also allow us to rapidly jump to any place in the recording. Some systems allow the user to split the screen and have two different time bases displayed simultaneously. Digital polysomnography makes data display ultimately manipulatable, after the fact. Channels can be moved, deleted, recolored, rescaled, inverted, and massaged with filters. With all of this transformational power, it becomes critically important for an audit trail to be kept so that the sleep specialist can see what the night technologist or scorer was seeing when he or she changed continuous positive airway pressures or scored a particular event or sleep stage. Finally, digital video (if properly implemented) allows the night technologist, scoring technologist, or interpreting clinician to see and hear the sleeping patient synchronized with the polysomnographic activity.

What Was Lost?

Casualties of the switch to computerized polysomnography include the disappearance of the selector panel, high-quality amplifiers, and in some cases calibration signals. The sounds associated with making an overnight recording have also vanished. The fact that one used to be able to actually audibly recognize REM sleep, periodic leg movements, and awakenings as distinctive sound patterns of pen chattering is no longer appreciated.

More importantly, amplifier quality and flexibility have seriously eroded. Many current systems provide signals that look “choppy.” Somehow, the inertial damping of the mechanical pen and the attenuating roll-off of analog filters created EEG, EOG, and EMG signals of a quality that has not been matched by digital systems. The sharp-edged, steep roll-off and notch filters mainly used today produce spikier waveforms that are more difficult to read. However, maybe that is a bias from having learned using paper tracings. The next generation may come to like the spiky, choppy display. One thing, however, that is not a matter of preference is the loss of flexibility to re-montage records on-the-fly. A simulated selector panel would solve many problems faced during recording.

The task force developed a wish list for manufactures. Content experts participated in a series of polls conducted according to the Rand/UCLA Appropriateness Method. As a result, some items on the list were elevated to the status of recommendations, others became options, and some were dropped (Table 184).

TABLE 18-4 -- AASM Digital Task Force List of Recommended and Optional Features for Computerized Polysomnographic Systems

Final Designation	Digital Polysomnography Feature Being Considered
	1600 × 1200 display resolution
	Video must be synchronized and be recorded at 1 frame/sec or more

Recommended	Independent 50/60-Hz filters for each channel
	Ability to independently set sampling rate for each channel
	Ability to check impedance for each channel against a selected reference
	Histogram with stage, breathing events, PLMs, Sao_2, and arousals.
	Histogram should have a cursor position page jump feature.
	Control key to toggle *biocalibration* section on/off, machine calibration section on/off
	Improved filter design that can functionally simulate analog-type roll-offs rather than removing all activity and harmonics within a bandwidth
	Recallable “see what tech was seeing” when the recording was made. Should include a complete audit trail of filter settings, resolutions, and other adjustments
	Recallable “see what tech was seeing” when the recording was scored. Should include a complete audit trail of filter settings, resolutions, and other adjustments
	Ability to time-scale on a single page ranging from 5 sec to the entire night
Optional	FFT or spectral analysis on window (omitting data artifact–marked segments)
	“Channel off” control key
	“Channel invert” control key
	Channel repositioning by click and drag
	Recallable display setup profiles (including colors) activated by control key sequences
	Page autoturning and autoscrolling
No Designation Provided	Channel control bar with display/nondisplay toggle
	Multiple window display with different time bases
	No toggle or key needed to display data
	Position cursor over channel and use up/down keys to rescale amplitude on-the-fly
	Page autoturning and autoscrolling speed control on mouse wheel
	Page autoturning and autoscrolling continuation during static lookback viewing
	Surround window for stages ± 4 with current in center
	Surround or simple window for body position, PAP setting
	Sounds accompanying autoscrolling (with volume control)

FFT, fast Fourier transform; PAP, positive airway pressure; PLMs, periodic leg movements; Sao_2, arterial oxygen saturation.

Attended and Unattended Sleep Recordings

In 1994, the AASM differentiated among four levels of sleep recordings.[35] The classification is based on the number and type of signals recorded. *Level I* is standard polysomnography—that is, the type of overnight sleep study that is routinely recorded in laboratories for clinical purposes with a sleep technologist present. It is sometimes called comprehensive polysomnography, attended laboratory polysomnography, CPT 95810, or CPT95811 (if positive airway pressure is administered). Regardless of the name, level I recordings include measures of EEG, EOG, submentalis EMG, heart rhythm, respiratory effort, oxygen saturation, body position, and anterior tibialis EMG (Table 18-5). *Level II* is equivalent to level I in all respects in terms of recording channels (i.e., it is comprehensive); however, the recording is made without an attendant present. *Level III* recordings are also known as *cardiopulmonary sleep studies*. These sleep studies include 4 or more channels. The recordings usually include measures of airflow, respiratory effort, oxygen saturation, and heart rhythm. Level III devices are recorders typically designed for home use, and seldom display the physiologic activity on a screen for monitoring purposes. Data are cached into memory and subsequently downloaded to a personal computer for display, scoring, editing, and generating a report of sleep study results. The recorders are compact, portable, and sometimes worn like Holter monitors. Finally, *level IV* devices are similar but record only one or two channels (e.g., pulse oximeters used to document the need for supplemental oxygen).

TABLE 18-5 -- AASM Classification System for Recording Techniques and Devices Designed to Evaluate Sleep and/or Sleep-Disordered Breathing

Level	Original Designation	Current Designation	Description
I	Standard polysomnography	Attended laboratory polysomnography	This is the type of study usually conducted in a sleep laboratory. It includes recordings of EEG, EOG, submentalis EMG, airflow, respiratory effort, ECG, Sao_2, and usually leg movement. A technologist is present throughout the study.
II	Comprehensive portable polysomnography	Unattended polysomnography	Recordings are similar or same as level I; however, the study is not continuously monitored by a technologist.
III	Modified portable sleep-apnea testing	Unattended cardiopulmonary recording	This is a recording specifically designed to detect sleep-disordered breathing. It usually consists of 4 or more channels that include measures of airflow, respiratory effort, electrocardiogram, and oximetry. Snoring sounds and body

			position are also sometimes recorded.
IV	Continuous (single- or dual-) bioparameter recording	Unattended single- or dual-channel recording	A portable unit that records and stores one or two data channels (e.g., a pulse oximeter)
Unclassified	None	Other	There are several devices that do not fit into the level I–IV categories; however, they can be used to test for sleep-disordered breathing (e.g., WatchPAT)

ECG, electrocardiogram; EEG, electroencephalogram; EMG, electromyogram; EOG, electro-oculogram; Sao_2, arterial oxyge saturation.

The AASM recommends that level I studies be used when laboratory facilities are readily available. The level I study provides the best sensitivity and specificity for the full range of sleep disorders and does not generate “errors of omission” when the pathophysiology is something other than a breathing disorder (a key issue with level III studies). Level III device use for diagnosis is rapidly evolving. By the time this is published, the Center for Medicare & Medicaid Services will have approved reimbursement for level III devices. AASM guidelines at the time of this writing provide for limited level III device use when there are extenuating circumstances.[36] AASM guidelines stipulate that (1) evaluations should be supervised by a practitioner who is board eligible or certified in sleep medicine, (2) evaluations should be performed only on patients having a high pretest probability of moderate to severe obstructive sleep apnea, and (3) evaluations may be performed on patients with immobility and safety issues that preclude in-laboratory study. These guidelines also indicate that portable studys should not be used for general screening in asymptomatic individuals, individuals who have concomitant sleep disorders, or individuals with comorbid illnesses that may compromise the testing system's accuracy.

Level III (cardiopulmonary) studies can diagnose SRBD in patients with high prestudy probabilities (i.e., those exceeding 70%). A variety of validated techniques can be used to calculate prestudy probability (we use the Multivariate Apnea Prediction Test[37] in conjunction with Epworth Sleepiness Scale[38] scores). AASM guidelines specify that level III studies can be performed when patients have severe symptoms and attended standard polysomnography is not readily available. The guidelines also allow level III studies for patients who cannot be studied in the sleep laboratory. Finally, level III studies can be used for follow-up after SRBD is diagnosed with attended standard polysomnography and therapy is initiated.

As useful as cardiopulmonary studies may be to confirm a positive diagnosis for SRBD, a negative cardiopulmonary study does not rule out all forms of SRBD (especially upper airway resistance syndrome) or non-SRBD sleep problems. Thus, a negative study using a level III device provides very little diagnostic information and requires follow-up evaluation, usually with level I, attended polysomnography.

Integrating New Technologies into a Sleep Disorders Program

A formidable array of level III and yet-to-be-classified devices are currently available. Most of these technologies are competent when placed in the right hands. Like any other form of new technology, the authors submit that the challenge is to determine how they best fit into our overall sleep programs. To make this determination, the goals of the program must be examined. If the goal is to provide sleep services in an optimal manner, there is likely a place for cardiopulmonary recording. If the goal is to maximize the number of laboratory studies performed, there may still be a place (as a case finding tool), but it is less likely. The time is long past for debating whether we should or should not used nonlaboratory assessment to diagnose SRBDs. *It is precisely that debate that has slowed our progress in determining how best to use the new technology*. Each technology has its own particular strengths and weaknesses. When properly combined, the unique properties of each can complement one another to a program's advantage.

Sleep programs that integrate home monitoring have been described in the literature.[39–41] Success seems to hinge on several factors, including (1) proper patient selection, (2) use of an appropriate portable recorder, (3) interpretation of the portable recording by a qualified sleep specialist, (4) readily available access to laboratory polysomnography (when needed), and (5) systematic follow-up.

Proper patient selection involves estimating prestudy probability for SRBD. An assortment of symptom checklists, questionnaires, and composite scales that use anthropometric and self-reported data exist. The patient can be referred for portable study when clinical suspicion is high. If prestudy probability is low, the patient is not sleepy, or overall symptom presentation is mixed, the patient should be referred for full laboratory assessment. Determining the competency of a portable recorder can be difficult. Relying on published literature is problematic because the technology advances so rapidly. For example, the particular model of instruments is often obsolete and unavailable by the time its validation study is published. Unfortunately, there is no “consumer report” or review clearing house for such products. Selection is based on collegial advice (word-of-mouth), experience during a rental period or manufacturer-arranged test trial period, device demonstrations, and aggressiveness of the sales representative.

Once the cardiopulmonary recording is made, it should first be technically validated by reviewing for signal quality and second be interpreted clinically (if it is technically adequate). Technical validation of the recording is much more crucial for level III studies than for laboratory polysomnography. After all, the technologist's primary function in attended, level I studies is to assure recording quality by reattaching transducers, re-referencing electrodes, minimizing electrical

interference, and even replacing recording devices, if needed. Monitoring transducers are appropriately attached, placed, and operationally checked by the technician. Finally, the recording is continuously monitored for whether intervention is required. By contrast, in home recordings, when a transducer falls off, it usually stays off; when a wire gets unplugged, it usually stays unplugged. Consequently, home studies, even though they have many fewer data channels than full polysomnograms, can be more challenging to read and interpret. An experienced sleep specialist familiar with cardiopulmonary recorder recordings should review the data and refer the patient for attended polysomnography if the test is negative. When the portable study is positive for obstructive sleep apnea, the patient is referred for either laboratory positive airway pressure titration or home self-adjusting autotitration positive airway pressure (APAP) (if such a procedure is used in the sleep program). Using APAP for unattended titration is beyond the scope of this chapter; the interested reader should see the AASM guidelines for more information.[42] Follow-up after the home recording is crucial to determine if anything unusual happened during recording. It is also important to explain treatment choices, provide cautions about the use of sedative-hypnotics and alcohol, emphasize the cardio- and cerebrovascular risk factors, and counsel patients about sleepiness-related dangers of driving and operating heavy equipment. Follow-up is key after therapy has been initiated to check for persistent signs or symptoms of sleep problems that were potentially missed by cardiopulmonary monitoring (Fig. 18-23).

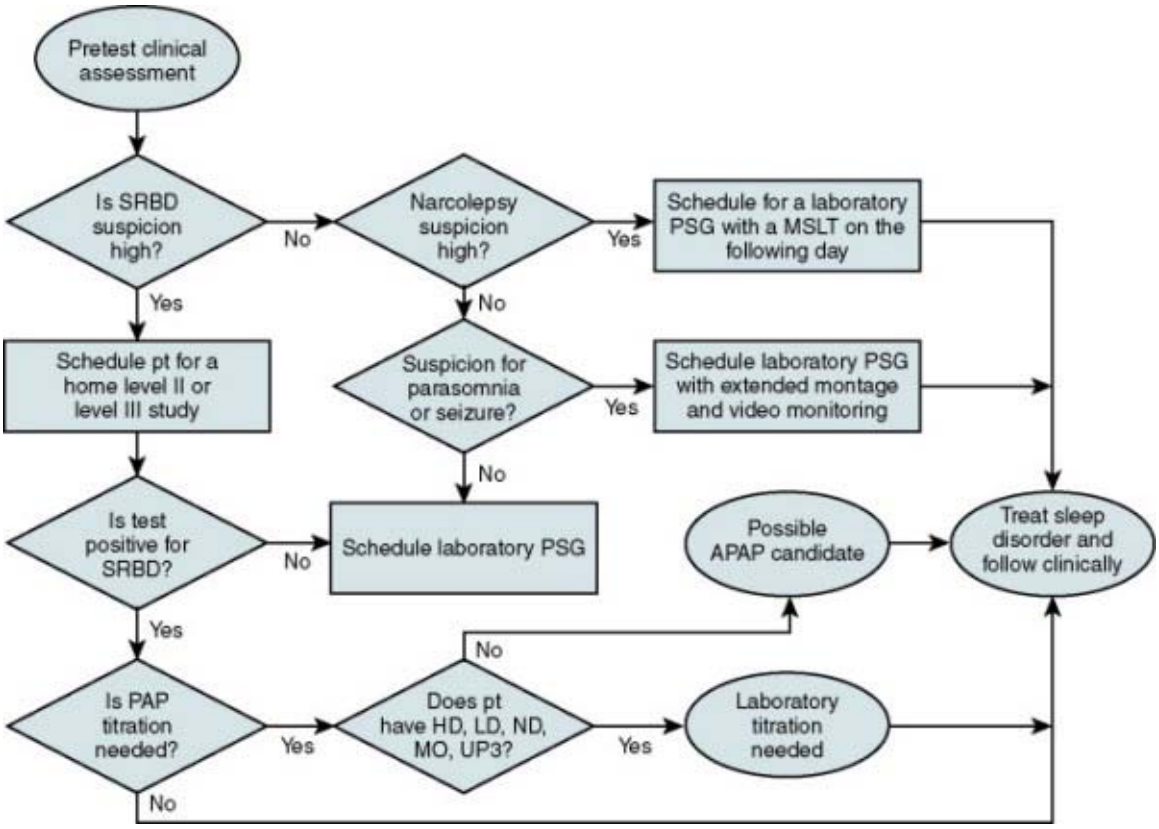

FIGURE 18-23 Integrating level II and III sleep recording devices into a clinical sleep program. (APAP, autotitrating positive airway pressure; HD, heart disease; LD, lung disease; MO, morbid obesity; MSLT, Multiple Sleep Latency Test; ND, neurologic disease; PAP, positive airway pressure; PSG, polysomnography; Pt, patient; SRBD, sleep-related breathing disorder; UP3, uvulopalatopharyngeoplasty.)

Level III recorders are another tool available for practicing sleep medicine. These devices do not replace polysomnography but rather complement it when appropriate. Properly integrated nonlaboratory assessment techniques can facilitate diagnosis of patients with more severe sleep-disordered breathing. In oversubscribed and/or capitated programs, home monitoring can free up laboratory resources for the more difficult cases.

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