Monitoring and Staging Human Sleep

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ABSTRACT

Assessment of human sleep stages using electrophysiologic techniques—polysomnography (PSG)—is typically carried out in a sleep laboratory, although home-based PSG can also be performed. The scoring of sleep states and stages requires the acquisition of three core measures: the electroencephalogram (EEG), the electrooculogram (EOG), and the surface electromyogram (EMG). These basic PSG measures allow sleep staging according to conventions established in the 1960s to define two states of sleep—NREM and REM sleep—and four stages within NREM sleep. Stage 1 sleep is light sleep, occurring at sleep onset and transitions during the night; high levels indicate poor sleep. Stage 2 sleep is defined by sleep spindles and K-complexes in the EEG. Stages 3 and 4 sleep, collectively known as slow wave sleep, are characterized by high-voltage slow (two or fewer cycles per second) waves in the EEG, 20% to 50% for stage 3 and 50% or greater for stage 4. REM sleep is distinguished by a relatively low voltage mixed-frequency (desynchronized) EEG, bursts of rapid eye movements in the EOG, and suppression of EMG activity. The three core measures are usually supplemented by other measures, such as heart rate, breathing, limb movements, and so forth in the clinical setting. Sleep staging contributes to the definition of certain sleep disorders, can establish the severity of sleep disorders, and is useful for assessing the efficacy of therapeutic interventions.

The goal of this chapter is to summarize the procedures for monitoring and evaluating sleep in the laboratory setting. This material will not substitute for the standard manual; rather, it will be complementary. After recommended techniques and procedures are summarized, a few problematic areas are discussed briefly.

Although it is possible to monitor continuously and concurrently the activity of dozens of systems during sleep, just three systems need to be measured to assess sleep according to standard criteria. This standard system of sleep recording and staging criteria is firmly rooted in the U.S. sleep research tradition, and although certain of its criteria have been challenged in recent years, it is the only system established by a consensus of experts. (A working group of the American Academy of Sleep Medicine is currently [2004] addressing the issue of preparing a new sleep staging manual.)

Among the earliest descriptions of electroencephalographic (EEG) activity during sleep were those from the laboratory of Loomis and colleagues. These authors described five stages of sleep but failed to distinguish rapid eye movement (REM) sleep. Not until the landmark work of Kleitman’s group at the University of Chicago was REM sleep described, a description made possible by the addition of electrooculography (EOG) to the recording paradigm. The first comprehensive description of the nocturnal pattern of non–rapid eye movement (NREM) and REM sleep in humans remains a foundation of modern human sleep research and represents one of the most outstanding scientific achievements of the 20th century. The standard sleep staging system modified the EEG and EOG categorizations of Dement and Kleitman primarily by adding electromyography (EMG). The addition of EMG to the criteria was based on the research of Berger in humans and Jouvet and Michel in cats, which linked muscle atonia with REM sleep. The EMG provided a more stable marker for REM sleep than the intermittent bursts of rapid eye movements.

PROCEDURES FOR MONITORING SLEEP

The EEG is the core measurement of polysomnography. The four stages of NREM sleep are distinguished from each other principally along this dimension.

Electroencephalogram

Application

The reliable recording of EEG begins with accurate measurement of the skull according to the international 10-20 system of electrode placement. A skilled technologist can make the requisite measurements in 10 to 20 minutes. The “eyeball” or rule-of-thumb placement of EEG electrodes is not recommended because of the marked variability of electrode locations such practices engender, regardless of the technologist’s skills.

Figure 116–1 illustrates the 10-20 placement system, by which a grid is marked on the skull and points of intersection denote electrode placement locations. The name of the system derives from measurements made at intervals of 10% or 20%
was largely an economic issue for most laboratories, which at that time were limited to eight-channel recording systems on which two subjects were generally recorded simultaneously. Nevertheless, this economically dictated approach has proved to be a remarkably robust system.

Sleep stage scoring does not require measurement of focal EEG activity or regional comparisons, as might be performed in an EEG laboratory. Rather, all of the EEG waveforms used to distinguish sleep stages are well visualized at C3 or C4, particularly when signal amplitudes are optimized, with the relatively large interelectrode distance afforded by a contralateral reference. Thus, vertex sharp waves and K-complexes, which are maximal over the vertex, are clearly evident at C3 and C4; high-voltage slow waves characteristic of deep NREM sleep are seen maximally in frontal regions yet show clearly on central derivations; alpha rhythm, by contrast, is maximal over the occipital region but can be characterized centrally in most humans.

Therefore, only C3/A2 or C4/A1 is used in the standard assessment of sleep stages. Many laboratories, however, routinely record an occipital EEG (usually O1/A2 or O2/A1) as an adjunct to the central EEG, particularly for assessing sleep onset or arousals during sleep. Certain laboratories also routinely record from frontal placements. When the latter procedure is used for sleep staging, however, there is a tendency to observe a somewhat greater quantity of the deep NREM sleep stages (stages 3 and 4); therefore, such use should be documented in any published reports. Finally, clinical laboratories often record a more extensive EEG montage not used for sleep staging per se but for assessing potential regional or focal sleep-related EEG anomalies.

Electrooculography

Eye movement activity is recorded during sleep for two primary reasons. The more obvious is to record the cardinal sign of REM sleep—the phasic bursts of rapid eye movements—which provide an essential sleep stage scoring criterion. In addition, the onset of sleep in most humans is heralded or accompanied by slow, rolling eye movements, which also occur with transitions to stage 1 during the night. Although these slow eye movements (SEMs) are not essential to sleep staging, they often provide very useful information.

The EOG recordings are based on the small electrophotential difference from the front to the back of the eye. The cornea is positive with respect to the retina. Thus, the eyeball exists in the head as a potential field within a volume conductor. Because of this essentially constant potential difference, movement of the eyes can be measured from electrodes placed beside the eyes. An electrode nearest the cornea will record a positive potential; an electrode nearest the retina will record a negative potential. As the eye moves, the positions of the cornea and retina change relative to the fixed position of the electrode, and a potential change will register as a pen deflection at the polygraph.
Derivations

The standard manual recommends continuous referential recording of two EOG leads: one outer canthus placement referred to the auricular reference on the opposite side and the other to the same auricular reference (e.g., ROC/A1 and LOC/A1). Certain laboratories routinely use a contralateral reference for each outer canthus placement (ROC/A1 and LOC/A2). In the latter case, the contralateral references maximize the signal amplitude for both EOGs and equalize the amplitudes of pen deflections for conjugate eye movements. Either technique provides the capability to distinguish eye movements from electrode artifact. For example, in a montage recording ROC/A1 on one channel and LOC/A2 on another, conjugate eye movements will register as out-of-phase pen deflections. EEG activity reflected in the EOG channels (e.g., K complexes) will be seen as in-phase deflections, and electrode artifacts will register in phase or in only one channel.

When a major goal of an experiment is to determine more precisely the direction of eye movements, the EOG may be simultaneously recorded from horizontal and vertical placements. Thus, in addition to placements on the outer canthi, electrodes would be placed supraorbitally and infraorbitally. For exact determination of eye position, direct current recordings are used.

Electromyogram

In a standard polysomnographic recording, the EMG from muscles beneath the chin is used as a criterion for staging REM sleep. The EMG recordings from other muscle groups are sometimes used to assess certain sleep disorders. For example, the anterior tibialis EMG is useful to evaluate patients who have periodic leg movements during sleep. The intercostal EMG has been used to monitor respiratory effort in certain laboratories. Most EMG recordings during sleep require taping electrodes to the skin over the muscle group of interest.

Application

Three electrodes are placed beneath the chin, overlapping the mentalis/submentalis muscles. These placements are typically used for the sake of convenience. The chin is very accessible, and the electrode wires can be drawn together with the others to form a bundle or "pony tail" at the back of the head. As in preparation for the EEG and EOG leads, the skin is thoroughly cleansed of oils and dead skin cells before applying the electrodes, which are generally secured with tape. Particularly in the case of a patient with a beard, the EMG electrodes may be affixed using a collodion-soaked gauze pad in the manner of the EEG leads.

Derivations

The chin EMG is recorded bipolarly. Any combination of the three placements can be used; the pair selected should produce the record of highest quality. The primary reason for using three electrodes (even though only two are recorded at any given time) is to ensure that there is always a backup electrode in case of failure of one placement. Availability of a backup is important, especially if electrodes remain in place during the daytime when subjects are eating and talking. To monitor bruxism, EMG electrodes may be offset to a location over the masseter muscle.

General Considerations for Recording Sleep

A minimal four-channel montage for recording sleep is shown in Table 116-1. If channels are limited, it is possible to use a single EOG channel, although this practice is discouraged. If more channels can be devoted to the sleep portion of the recording, an occipital EEG will be the most helpful addition. In the rare cases when limited to four sleep channels, perhaps due to an extended clinical montage, some laboratories begin a night's recording with a central and an occipital EEG, a single EOG, and an EMG. After sleep onset, the occipital EEG is replaced with the second EOG. Depending on the purpose of the recording, other selected parameters will be added to the montage to record respiration, heart rate, blood pressure, esophageal pH, penile circumference, or any of the many other available systems.

Most sleep laboratories use digital recording systems, often configured to appear as if recorded at a chart paper speed of 10 or 15 mm/sec. Thus, one screen displays about 30 seconds of data. More compressed views are discouraged because clear visualization of alpha rhythm and sleep spindles becomes extremely difficult. Less compressed displays may be useful for some clinical recording purposes; however, sleep staging that applies standard criteria is best accomplished with 30-second screen images. The display typically provides a sensitivity that gives a deflection of 7.5 or 10.0 mm for a 50-μV signal for the EEG and EOG channels. The chin EMG amplification is often adjusted to provide an acceptable EMG recording for distinguishing NREM and REM sleep (see later). A known calibration of the central EEG leads is essential because of the amplitude criterion for scoring NREM stage 3 and 4 sleep. A calibration of 50 μV/cm is common.

Electroencephalographic filtering should allow suitable visualization of a fairly wide range of signals, from slow waves (2 cycles per second [cps] or less) to sleep spindles (12 to 14 cps). A high-frequency filter setting in the range of 30 to 35 cps is common for visualizing a signal that passes through the essential waveforms while minimizing high-frequency (e.g., EMG) interference. A time constant of 0.3 second or slower (corresponding to a half-amplitude low-frequency filter setting of about 0.3 cps) ensures adequate visualization of slow wave activity.

<table>
<thead>
<tr>
<th>Table 116-1. Montage for Monitoring Sleep States</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parameter</td>
</tr>
<tr>
<td>-----------</td>
</tr>
<tr>
<td>EEG</td>
</tr>
<tr>
<td>EOG</td>
</tr>
<tr>
<td>EMG</td>
</tr>
<tr>
<td>If additional channels are available, add the following:</td>
</tr>
<tr>
<td>EEG</td>
</tr>
<tr>
<td>EOG</td>
</tr>
</tbody>
</table>

Monitoring and Staging Human Sleep 1361
The same settings are recommended for EOG channels to provide visualization of both the rapid eye movements essential to scoring REM sleep and the SEMs characteristic of sleep onset and transitional stage 1 sleep. A faster time constant has been used in certain laboratories to reduce the contamination of EOG by EFG signals. In slow wave (stages 3 and 4) sleep, however, the EFG activity seen in the EOG channels (with a slow time constant) tends to have fewer overriding fast components than the central leads and may therefore be helpful in distinguishing the slow EFG components. Thus, the slower time constant for EOGs may be doubly helpful.

The EMG is generally recorded with a much higher setting on both high- and low-pass filters. A low-pass setting of 70 or 75 cps is common (with notch filtering of alternating current interference—e.g., 60 Hz). High-pass filtering at 10 cps (time constant = 0.015 second) is useful to prevent slow signals from interfering with the EMG tracing. The standard manual recommends a time constant for EMG of 0.1 second or faster. The absolute amplitude of EMG activity is not relevant to polysomnography; the emphasis, rather, is on relative changes in EMG amplitude. Thus, the EMG level is adjusted at the start of the record to provide an amplitude that permits such comparisons. An amplification of 20 µV/cm at the start of the recording will usually approximate a reasonable EMG level. A number of special cases may require modification of the sensitivity, filter, and time constant settings just described. In most digital recording systems, such modifications can be achieved on line and during playback. In certain patients (especially older individuals), the amplitude of the EEG using the standard setting may be so small that the record is extremely difficult to evaluate and is more easily appreciated when a zoom function is used to provide, for example, a deflection of 15 mm for a 50-µV signal. By contrast, the amplitude of the EEG in some young children is very large and may require reduction of the amplification (or zoom out) to appreciate the recording—for example, a deflection of 5 mm for a 50-µV signal.

The EEG and EOG channels may also pick up a very low-frequency artifact related to sweat. In this instance, the low-frequency cutoff may be set at 1.0 cps (time constant = 0.1 second) during recording. Injudicious use of filtering holds some risk, however, of distorting the signals of interest. For example, a 5-cps low-frequency cutoff setting for an EEG will eliminate slow frequency signals important for assessing stages 3 and 4 NREM sleep.

A sleep recording requires vigilance during acquisition, and a key concern is that the technologist stays awake and alert. The recording must be observed frequently to ensure that proper electrode connections are maintained and that recording artifacts are minimized, as well as to monitor the safety and comfort of the patient and observe clinically relevant behaviors. This requirement for technologist vigilance makes the process of laboratory sleep recording a labor-intensive procedure; however, it also eliminates the necessity of retesting because of lost data.

Several general concepts, referring primarily to the EEG, are helpful when approaching sleep stage scoring. It should first be noted that the sleep research community has adopted the EEG convention of "negative up," which simply means that a signal of negative polarity is shown as an upward peak deflection. Second, a number of the standard guidelines refer to the frequency of the EFG waves. Frequency is measured in cycles (each cycle is the complete series of potential changes before the series repeats) per second. A few common EEG frequency bands are as follows:

1. Alpha rhythm: 8 to 13 cps
2. Beta rhythm: more than 13 cps
3. Delta rhythm: less than 4 cps
4. Theta rhythm: 4 to 7 cps

The amplitude measures used in sleep staging are taken from trough to peak (or peak to trough) of the wave, rather than from baseline or zero crossing to peak or trough.

When a sleep recording is scored, it is customary to divide the recording into convenient segments and to assign a sleep stage value to each segment or epoch. The most common epoch length is 30 or 20 seconds, which corresponds to an analogue recording's single page of chart paper 300 mm wide recorded with a chart speed of 10 or 15 mm/sec. A 1-minute scoring epoch has also been used (see particularly Williams et al.13), although such a long epoch may overlook stage changes of relatively short duration.13 A scoring epoch shorter than 20 seconds is considered too tedious by most groups, although epochs as short as 3 seconds have been used for specific research purposes.

Each epoch is assigned a score that most appropriately characterizes the predominant pattern occurring during that interval. Thus, the purpose of epoch staging is to determine the single descriptive factor that most fully characterizes the epoch. Any number of additional codes may be used to denote activities or events occurring within (or across) an epoch. Thus, for example, the standard manual describes "movement arousals," which are short-lived events occurring within an epoch but not descriptive of the majority of the epoch. With the increasing application of polysomnography to clinical assessments, the variety of possible events to be evaluated has grown markedly. Table 116–2 gives a partial listing of events that are coded by various laboratories. To date, these events have usually been defined within each laboratory because standard consensus descriptions are generally lacking. Laboratories typically select those events that are of local experimental or clinical relevance. Event coding is an extremely valuable adjunct to sleep staging, but it is not a substitute for sleep staging.

**Sleep Staging in Normal Adult Humans**

The following material summarizes the criteria described in the standard manual1 for staging normal human sleep. Although these criteria apply most specifically to adults, they have also been used to characterize sleep in children and adolescents.13,15 A separate set of criteria, however, is generally deemed necessary in newborns16 and older infants.17 The standard sleep staging criteria in adults, according to the three electrographic parameters, are outlined in Table 116–3 and described next.
Table 116-2  Partial Listing of Events That May Be Coded Within or Across Epochs

<table>
<thead>
<tr>
<th>Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body movement</td>
</tr>
<tr>
<td>Movement arousal</td>
</tr>
<tr>
<td>Transient arousal</td>
</tr>
<tr>
<td>Microsleep episodes</td>
</tr>
<tr>
<td>K-alpha complex</td>
</tr>
<tr>
<td>Esophageal pH abnormalities</td>
</tr>
<tr>
<td>Respiratory abnormalities</td>
</tr>
<tr>
<td>Apnea</td>
</tr>
<tr>
<td>Obstructive</td>
</tr>
<tr>
<td>Central</td>
</tr>
<tr>
<td>Mixed</td>
</tr>
<tr>
<td>Hypopnea (usually ≥10 sec)</td>
</tr>
<tr>
<td>Obstructive</td>
</tr>
<tr>
<td>Central</td>
</tr>
<tr>
<td>Mixed</td>
</tr>
<tr>
<td>Paradoxical respiration</td>
</tr>
<tr>
<td>Cheyne-Stokes respiration</td>
</tr>
<tr>
<td>Periodic breathing</td>
</tr>
<tr>
<td>Periodic movements</td>
</tr>
<tr>
<td>With arousal</td>
</tr>
<tr>
<td>Without arousal</td>
</tr>
<tr>
<td>Penile tumescence</td>
</tr>
<tr>
<td>T&lt;sub&gt;up&lt;/sub&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;max&lt;/sub&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;down&lt;/sub&gt;</td>
</tr>
<tr>
<td>Heart rate irregularities</td>
</tr>
<tr>
<td>Asystole</td>
</tr>
<tr>
<td>Premature ventricular contraction (PVC)</td>
</tr>
<tr>
<td>Premature atrial contraction (PAC)</td>
</tr>
<tr>
<td>Tachycardia</td>
</tr>
<tr>
<td>Bradycardia</td>
</tr>
<tr>
<td>Oxygen saturation</td>
</tr>
<tr>
<td>Below 90%</td>
</tr>
<tr>
<td>Below 80%</td>
</tr>
<tr>
<td>REM sleep phasic events</td>
</tr>
<tr>
<td>Twitches</td>
</tr>
<tr>
<td>Rapid eye movements</td>
</tr>
<tr>
<td>Middle ear muscle activity</td>
</tr>
<tr>
<td>Periorbital integrated potentials</td>
</tr>
</tbody>
</table>

REM, rapid eye movement.

Relaxed Wakefulness

The majority of humans show an EEG of rhythmic alpha activity (in the range of 8 to 13 cps) when relaxed with the eyes closed (Fig. 116–2). This activity is maximal occipitally but also often occurs centrally. This rhythmic EEG pattern attenuates with attention, as well as when the eyes are open (Fig. 116–3), at which time the waking EEG pattern is best characterized as one of relatively low voltage and mixed frequency. In an excessively sleepy individual, rhythmic alpha activity may be present when the eyes are open and may attenuate with eye closure; in this case, alpha attenuation is related to the intrusion of stage 1 sleep.

When a person is awake, control of eye movements is voluntary. The waking EEG tracing generally consists of rapid eye movements and eye blinks when the eyes are open, and few or no eye movements with the eyes closed. Involuntary slow, rolling eye movements (with eyes closed) often characterize the EEG in the seconds to minutes preceding the EEG change to stage 1 sleep.

The EMG shows tonic activity of a relatively high level. Voluntary movements produce phasic increases of EMG amplitude. In very relaxed individuals, waking EMG tone may be indistinguishable from NREM sleep.

NREM Sleep

The four NREM sleep stages are distinguished, as mentioned previously, principally by changes in EEG pattern. The EEG and EMG patterns contribute little to NREM sleep staging, except in the case of transitional stage 1 NREM sleep, in which both may be useful. Therefore, the discussion here will focus on EEG.

Stage 1

The transition from wakefulness to stage 1 sleep (Fig. 116–4) is most clearly visualized on the EEG when the waking pattern has well-defined rhythmic alpha activity. It is for this reason that an occipital derivation is frequently added to the sleep recording montage, because waking alpha activity is most prominent in this cortical region. The EEG pattern of stage 1 is described as relatively low-voltage, mixed-frequency activity. Especially during stage 1 sleep occurring at the beginning of the night, vertex sharp waves (Fig. 116–5) are common. In addition, the EEG activity with the highest relative amplitude during stage 1 sleep is generally in the theta (3 to 7 cps) range. Bursts of relatively high-voltage, very synchronous theta activity are common during the onset of stage 1 sleep in children and young adolescents (Fig. 116–6).

The SEMs commonly precede the EEG transition to stage 1 sleep from wakefulness. Although the onset of SEMs usually leads the EEG transition by only 1 or 2 minutes, the lead time may occasionally—particularly in daytime recordings—he as long as 15 minutes. SEMs are very useful to distinguish stage 1 sleep transitions occurring during stage 2 NREM sleep or REM sleep.

Muscle tone is maintained during all NREM sleep stages and registers as low-amplitude EMG activity. There generally is no discrete change in EMG amplitude in the wake-to-sleep transition, although a gradual diminution of the EMG signal amplitude may occur within moments of the transition. During NREM sleep, the EMG is most helpful for distinguishing movement arousals, which is useful in certain stage change decisions. In addition, a rise in EMG activity is often the only discrete indicator of a transition to stage 1 sleep within a REM sleep episode (see Fig. 116–12).

Stage 2

The background EEG of stage 2 NREM sleep is a pattern of relatively low voltage, mixed-frequency activity. Stage 2 is distinguished from stage 1 on the basis of two specific EEG patterns that occur sporadically on this mixed-frequency background: the sleep spindle and the K-complex (Fig. 116–7).
### Table 16-5 | Outline of Sleep Scoring Criteria According to Standard Manual

<table>
<thead>
<tr>
<th>Stage/State</th>
<th>Electroencephalogram (EEG)</th>
<th>Electrooculogram (EOG)</th>
<th>Electromyogram (EMG)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relaxed</td>
<td><strong>Eyes closed:</strong> rhythmic alpha (8-13 cps); prominent in occipital, attenuates with attention</td>
<td>Voluntary control; REMs or none; blinks; slow eye movements (SEMs) when drowsy</td>
<td>Tonic activity, relatively high; voluntary movement</td>
</tr>
<tr>
<td>wakefulness</td>
<td><strong>Eyes open:</strong> relatively low voltage mixed frequency</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-rapid eye movement sleep (NREM)</td>
<td>Relatively low voltage, mixed frequency</td>
<td>SEMs</td>
<td>Tonic activity, may be slight decrease from waking</td>
</tr>
<tr>
<td>Stage 1</td>
<td>May be theta (3–7 cps) activity with greater amplitude</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Vertex sharp waves</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Synchronous high-voltage theta bursts in children</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 2</td>
<td><strong>Background:</strong> relatively low voltage, mixed frequency</td>
<td>Occasionally SEMs near sleep onset</td>
<td>Tonic activity, low level</td>
</tr>
<tr>
<td></td>
<td><strong>Sleep spindles:</strong> waxing, waning, 12–14 cps (≥0.5 sec)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td><strong>K-complex:</strong> negative sharp wave followed immediately by slower positive component (≥0.5 sec); spindles may ride on Ks; Ks maximal in vertex; spontaneous or in response to sound</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 3</td>
<td>≥20%, ≤50% high amplitude (≥75 µV), slow frequency (≤2 cps); maximal in frontal</td>
<td>None, picks up EEG</td>
<td>Tonic activity, low level</td>
</tr>
<tr>
<td>Stage 4</td>
<td>&gt;50% high amplitude, slow frequency</td>
<td>None, picks up EEG</td>
<td>Tonic activity, low level</td>
</tr>
<tr>
<td>Rapid eye movement sleep (REM)</td>
<td>Relatively low voltage, mixed frequency</td>
<td>Phasic REMs</td>
<td>Tonic suppression; phasic twitches</td>
</tr>
<tr>
<td></td>
<td>Sawtooth waves</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Theta activity; slow alpha</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Movement time</td>
<td>Obscured</td>
<td>Obscured</td>
<td>Very high activity</td>
</tr>
<tr>
<td>Anomalous sleep*</td>
<td>Similar to REM</td>
<td>Phasic REMs</td>
<td>Tonic activity; phasic twitches</td>
</tr>
</tbody>
</table>

*Described in reference 44.


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**Figure 116-2.** Rhythmic electroencephalographic (EEG) alpha activity is clearly evident in the C3/A2 and O2/A1 tracings of this young adult male volunteer who is awake with his eyes closed. Figures 116–2 to 116–7 and 116–9 to 116–13 are all taken from an overnight recording of this 19-year-old healthy man. All leads were recorded on a Grass Instruments Company Model 78 polygraph. The central and occipital EEGs and the electrooculograms (EOGs) used a low-frequency cutoff of 0.3 cps, a high-frequency cutoff of 30 cps, and a sensitivity of 50 µV/cm. The electromyogram (EMG) was recorded with a low-frequency cutoff of 10 cps, a high-frequency cutoff of 60 cps, and a sensitivity of 20 µV/cm. Paper speed was 10 mm/sec. In Figures 116–2 to 116–5, the EOG is monitored with a single lead (ROC/LOC). In Figures 116–9 to 116–13, the occipital tracing has been dropped, and the EOG is recorded from two leads, ROC/A1 and LOC/A2. (See text for comments on the latter procedure.)
Figure 116-3. Attenuation of waking EEG alpha activity with eyes open is illustrated in this tracing. Note the characteristic "relatively low-voltage, mixed-frequency" EEG activity.

Figure 116-4. Transition from wakefulness to stage 1 sleep is illustrated, which clearly shows the attenuation of alpha that marks the onset of stage 1 sleep. As described in Figure 116-3 for an EEG of wakefulness with the eyes open, the EEG pattern of stage 1 sleep is one of "relatively low voltage, mixed frequency." Note, too, the presence of slow eye movements in the EOG tracing.

Figure 116-5. Vertex sharp waves are a common feature of the onset of stage 1 sleep. Few were seen in this volunteer, however, although one (underlined) is illustrated in this figure. Note that the vertex sharp wave is visible in the C3/A2 lead but not in the O2/A1 lead, emphasizing localization to the vertex region.

Figure 116-6. Very high voltage, highly synchronous theta activity (underlined) is common during sleep onset stage 1 in children and young adolescents. This phenomenon is illustrated here in a tracing from a 14-year-old male volunteer. (Recording parameters are as described in the legend for Figure 116-2, with the exception that EEGs were recorded with a low-frequency cutoff of 1.0 cps.)
Because these stage 2–defining EEG patterns occur episodically, the standard staging criteria provide for a default to stage 1 sleep if neither a sleep spindle nor a K-complex occurs within a 3-minute span when the EEG is of relatively low voltage and mixed frequency (the "3-min" rule).

In their most pure presentation, sleep spindles have a waxing and waning spindle shape (Fig. 116–8), composed of waves in the range of 12 to 14 cps, with a duration of about 0.5 to 1.5 seconds. Sleep spindles are a common feature of mammalian sleep, and when recorded using identical techniques, they are indistinguishable—for example, between humans and cats. Sleep spindle activity occurs during stage 2 sleep, with a frequency of about three to eight spindles per minute in normal adults or insomniac adults, and spindle rate appears to be a fairly stable individual characteristic. Incipient sleep spindles may appear near the stage 1 to stage 2 transition early during sleep; however, "the presence of a spindle should not be defined unless it is of at least 0.5 sec duration, i.e., one should be able to count 6 or 7 distinct waves within the half-second period." From an ontogenetic perspective, sleep spindles in humans usually develop before age 3 months. In infants with mental retardation, sleep spindles are slower to develop and occur less frequently than in normal infants. In older adults, sleep spindles tend to lose their classic morphology and have a slightly slower frequency, lower amplitude, and shorter duration than in the young adult. Benzodiazepine hypnotics tend to increase the density of sleep spindles in stage 2 sleep.

Sleep spindles (the term will be used interchangeably with K-complex here) are absent in stage 1 NREM but may occur in REM sleep, particularly in subjects or patients whose sleep has been restricted or fragmented. If a single sleep spindle occurs in the middle of a REM sleep episode, it is not considered to be indicative of a stage change. If, however, two sleep spindles bracket half a scoring epoch or longer with no intervening REMs, the interval between spindles is considered a stage 2 sleep interruption of the REM episode.

The K-complex (see Fig. 116–7) is another sleep-specific EEG waveform that is characteristic of stage 2 sleep. This paroxysmal wave complex consists of a "well-delineated negative sharp wave which is immediately followed by a positive component. The total duration of the complex should exceed 0.5 sec." The standard manual provides no amplitude criterion for K-complexes. There usually is very little difficulty in discerning K-complexes in stage 2 sleep. The following definition used by electroencephalographers for the term complex is very helpful when a K-complex distinction is in doubt, as may occur in stage 3 and 4 sleep, when it is sometimes difficult to differentiate K-complexes from high-voltage, slow wave activity. A complex is a "group of two or more waves, clearly distinguished from background activity and occurring with a well-recognized form or recurring with consistent form." A key part of this definition is that the complex is distinct from the ongoing background activity, which makes the K-complex in stage 2 very clear, whereas the same morphology embedded within a series of high-voltage, slow wave activity during stage 3 or 4 would probably not stand out from the background.

K-complexes are maximal over the vertex. It is very common for spindle activity (12 to 14 cps) to ride over the K-complex. In young adults, the typical density of K-complexes in stage 2 is about 1 to 3 per minute, although there is considerable individual variability. K-complexes occur spontaneously during stage 2 sleep and are also evoked in response to auditory stimuli.

At the beginning of the night, SEMs may infrequently and only very briefly persist after the appearance of sleep spindles and K-complexes. Because the EOG channels also register EEG activity, K-complexes can reflect on these channels (see Fig. 116–7). They are generally easily distinguished from rapid eye movements because the pens on the two channels deflect in phase and because the central EEG amplitude of a K-complex is usually much greater than any EEG activity related to eye movements. The EMG during stage 2 sleep is tonically active, generally at a low amplitude relative to wakefulness.
Stages 3 and 4

The EEG of stages 3 and 4 sleep is defined by the presence of high-voltage, slow wave activity (Figs. 116-9 and 116-10). In stage 3 sleep, “at least 20 per cent but not more than 50 per cent of the epoch consists of waves of 2 cps or slower which have amplitudes greater than 75 µV from peak to peak (the difference between the most negative and positive points of the wave).” In stage 4 sleep, such waves predominate (more than 50% of the epoch). Sleep spindles can occur during stages 3 and 4, as can K-complexes; however, they are only infrequently distinct from the background EEG activity, particularly in stage 4 sleep. Eye movements do not occur during stages 3 and 4 sleep, although the EOG registers the high-voltage, slow wave activity. The EMG during stages 3 and 4 is tonically active, although the tracing may occasionally achieve very low levels, nearly indistinguishable from that of REM sleep.

REM Sleep

Staging REM sleep requires the coincidence of specific activities in all three electrographic measures: “activated” or desynchronized EEG, bursts of rapid eye movements, and suppression of EMG activity (Fig. 116-11). The REM sleep EEG pattern is characterized as one of “relatively low voltage, mixed frequency.” An EEG pattern called sawtooth waves—because of their notched morphology—is fairly common during REM sleep, particularly in proximity to eye movements, but it is by no means a universal phenomenon. Thus, the presence of sawtooth activity is not required for staging REM sleep, although it may be very useful in equivocal instances. Sawtooth waves achieve the highest amplitude at the vertex and, like much other REM sleep EEG activity, have a frequency in the theta range. Activity in the alpha range (usually 1 to 2 cps slower than waking alpha activity) may also be seen in the REM sleep EEG.

Ponto-geniculo-occipital (PGO) spikes are a definitive feature of feline REM sleep, and rhythmic hippocampal theta activity is a prominent REM feature in many primates, cats, dogs, and rodents. In cats, PGO spikes occur singly in the transition to REM sleep and in bursts during REM sleep, usually leading other REM sleep phasic events. The scalp EEG routinely recorded in humans is not clearly related to these characteristic REM sleep patterns of other species. Hodes and Dement, however, suggested that K-complexes in humans may be an analogue of the pre-REM PGO spikes because both pre-REM events are similarly associated with EMG and reflex suppression. Depth EEG recordings in humans have also suggested the presence of PGO spikes in REM sleep.

The EOG reveals bursts of rapid eye movements at intervals during REM sleep (see Fig. 116-11). The acronym REM originated with these eye movements, of course, although the term is now used to denote the full constellation of physiologic events constituting this state. The density of rapid eye movement bursts within REM sleep varies with time of night; thus, earlier REM episodes contain fewer rapid eye movements than do later REM episodes. The episodic nature of this sign of REM sleep often requires the sleep record scorer to scan the chart in advance of the epoch currently under scrutiny. The criteria of the standard manual provide contingencies for such contextual decisions, as will be described later.

For an epoch to be considered REM sleep, in addition to the activated EEG and REM bursts, an EMG recorded in the manner described previously must obtain its lowest value. A universal feature of REM sleep in the intact organism is the tonic suppression of skeletal muscle tone and reflexes via a circuit that involves pontine activation of medullary inhibitory centers and culminates in postsynaptic hyperpolarization of brainstem and spinal motor neurons. Superimposed on this background of tonic motor inhibition can be seen occasional twitches of distal muscles. In household pets, for example, paws, face, and
whiskers show twitches in REM sleep. In human polysomnographic recordings, twitches appear as very short-lived EMG elevations, usually in proximity to eye movement bursts (see Fig. 116–11). Prolonged EMG elevation (15 seconds or longer) in REM sleep, even in the absence of an EEG change, requires a stage shift (Fig. 116–12). Brief EMG elevations associated with an alteration in EEG or EOG activity (i.e., movement arousal) may signal a stage change, depending on the relative size of this movement and duration of the EEG and EOG alterations.

Both REM sleep and NREM stage 2 sleep require the presence of episodic events: bursts of rapid eye movements in REM sleep and spindles or K-complexes in stage 2. In both, the background EEG is similar—that is, relatively low voltage, mixed frequency. Scoring the transitions from stage 2 to REM sleep (Fig. 116–13) and from REM level to stage 2, as well as stage 2 interruptions of REM sleep (Fig. 116–14), is therefore sometimes problematic. The following two fundamental guidelines from the standard manual\(^1\) make it possible to deal with virtually every contingency:

1. Any section of record contiguous with stage REM sleep in which the EEG shows relatively low voltage and mixed frequency is scored stage REM sleep regardless of whether rapid eye movements are present, providing EMG is at the stage REM level and there are no intervening movement arousals.

2. An interval of relatively low-voltage, mixed-frequency EEG record between two sleep spindles or K-complexes is considered stage 2 regardless of EMG level, if there are no rapid eye movements or movement arousals during this interval and if the interval is less than 3 minutes long.

The manual provides a variety of specific examples that apply these guidelines, and the reader is urged to review them.

**Movement Time**

Gross postural readjustments are fairly common during sleep, often occurring in the vicinity of REM episodes.\(^4\) When such movements arise from sleep or immediately precede sleep, and obscure the EEG activity (and usually the EOG as well) for at least one half of the scoring epoch, that epoch is designated “movement time.” If this pattern is preceded or followed by wakefulness, it is scored as an awake pattern.

**Considerations for Staging Sleep in Pathologies**

The standard manual was developed to provide guidelines for staging sleep in normal adults, and its recommendations are suitable for many pathologic conditions as well. Nevertheless, full characterization of sleep in a number of sleep-related pathologies at times requires a departure from the standard procedures. The following material briefly reviews certain issues that may arise in specific disorders and suggests alternatives for addressing these issues.

**Narcolepsy**

The sleep of patients with narcolepsy is characterized by sleep onset REM episodes (the occurrence of rapid eye movements within 15 minutes of sleep onset), mixtures of stage 2 and REM sleep, and arousals that are more numerous than seen in healthy persons.\(^41\) Each of these phenomena can be
characterized using the guidelines of the standard manual. Particular care is often required, however, and one may wish to use additional procedures, such as adding a vertical EOG, to assist in identifying a brief, early REM episode. An early REM episode may not last sufficiently long (e.g., longer than 15 seconds) to characterize a full epoch as REM sleep; its occurrence may nonetheless be diagnostically relevant and must be noted.

The most problematic area with narcolepsy generally involves patients whose medication regimen results in excessive motor activity during sleep, as characterized by an elevated EMG in the presence of an activated EEG and phasic rapid eye movements occurring with a periodicity similar to that of REM sleep.42,43 (This pattern may also occur in nonnarcoleptic patients, such as those with Alzheimer's or Parkinson's disease.) This might be characterized as an anomalous state, as shown in Table 116–3. Thus, epochs of anomalous sleep may be accounted for outside the standard criteria and staged as neither REM nor NREM sleep.44

Sleep Apnea Syndromes

Patients with a sleep apnea syndrome experience a great increase in the frequency of arousals from sleep and in the number of body movements. Both types of activity have an impact on sleep staging. For example, a patient may be clearly asleep and apneic for 10 seconds, and movement associated with the termination of the apnea may obscure the remainder of the epoch (Fig. 116–13). Another common occurrence in patients with a sleep apnea syndrome is the appearance of K-complexes almost exclusively at the termination of the apneas. If scored exclusively using the standard guidelines and a 30-second epoch, sleep might not be found in such patients or might appear as only stage 1 and movement time. The following suggestions (modified from Flagg and Coburn45) for scoring sleep in such patients attempt to account for these pathologic events:

1. Follow standard guidelines for entry into stage 1 from wakefulness and stage 2 from stage 1. (Coding “microsleep” episodes [less than half the epoch with stage 1 EEG] at the onset of sleep may be useful.)
2. Once stage 2 sleep is scored, continue stage 2 through any arousal that does not result in a transition to wakefulness (more than half the epoch with waking EEG). (Coding “transient arousals” [see later] may be useful.)
3. In REM sleep, ignore EMG elevations that are clearly associated with snoring.
4. In adults, stages 3 and 4 may be combined. (Some investigators46 recommend combining stages 2, 3, and 4 in patients with sleep apnea. Such crude categorization may obscure clinically relevant information and is not recommended, particularly for children.)

Alpha-Delta Sleep

An EEG pattern of alpha intrusion into NREM sleep was first noted in patients with psychiatric disorders.46 The pattern was described as “a mixture of 5-20 per cent delta waves (more than 75 μV, 0.5-2 cps) combined with relatively large amplitude, alpha-like rhythms (7-10 cps). These alpha rhythms are usually 1-2 cps slower than waking alpha.” A similar pattern has been related to a complaint of nonrestorative sleep in patients with musculoskeletal pain or fibrositis.47 This EEG
pattern might legitimately be scored as NREM stage 1 or 2, but the clinical implications of this type of sleep require that it be noted and remarked on. Thus, one might define a separate sleep “stage” or use an event code to make this pattern accessible for separate analysis.

**Transient Arousals**

Many sleep disorders involve frequent, brief arousals that do not alter sleep stage scoring but that may be clinically relevant. Such arousals are a common feature of normal aging as well. Clinical implications of these brief arousals have been shown in several types of studies. For example, brief arousals induced experimentally into the sleep of normal volunteers resulted in daytime sleepiness, even though the total amount of sleep was unchanged. In addition, spontaneously occurring transient arousals in older adults have been correlated with waking alertness level. This type of arousal occurs frequently in patients with sleep apnea syndromes, periodic movements in sleep, and other sleep disorders. Therefore, cataloging such events may be relevant in several clinical and nonclinical populations. The following definition has been proposed for transient arousals:

“Any clearly visible EEG arousal (usually alpha rhythm) lasting two seconds or longer, but not associated with any stage or state change in the epoch scoring. These brief arousals are sometimes, but not always, associated with a body movement or respiration event.”

To this definition we add the recommendation that transient arousals in REM sleep be coded only when EEG alpha activity is associated with another sign of arousal (e.g., increased heart rate, EMG elevation, or respiration irregularity), because alpha activity is a fairly common feature of REM sleep. A task force of the American Sleep Disorders Association (ASDA) has defined a set of scoring rules and has provided examples for coding EEG arousals during sleep.

The ASDA coding system has initiated a renewed interest in sleep-related arousals, an aspect of sleep staging that has beleaguered sleep researchers and clinicians for decades. For example, a 1999 task force of the American Academy of Sleep Medicine focused on the role of respiratory effort-related arousal events in helping define the severity of the obstructive sleep apnea—hypopnea syndrome (see Chapter 92). Furthermore, as more investigators examine sleep in newly described sleep disorders and other medical disorders, this interest in sleep fragmentation strengthens. For example, the description of upper airway resistance syndrome, which has rekindled interest in EEG arousals because the respiratory signs of this syndrome are less obvious than those in frank obstructive sleep apnea syndrome and only subtle indicators may be available. In addition, the ASDA arousal staging system and related research have stimulated an examination or a reexamination of arousals in other disorders such as allergic rhinitis, juvenile rheumatoid arthritis, and Parkinson’s disease.

Others have begun to examine variations of the ASDA arousal scoring schema, and still others suggest that non-EEG markers may be important and even more reliable signs of arousal than the EEG. Pitson and Stradling, for example, note that transient changes in blood pressure may signify arousals more reliably than cortical EEG, although Lofaso and colleagues indicate that autonomic changes are highly correlated with the extent of EEG arousals. Less well studied is the possibility that certain sleep-fragmenting phenomena are associated with subcortical events not visible in the cortical EEG signal. This may be the case in sleep studies of children who manifest few cortical arousals even with prominent obstructive sleep apnea syndrome.

A relatively new area of interest has established a pattern called cyclic alternating pattern, defined as “repetitive stereotyped EEG patterns lasting <60 seconds and separated by time-equivalent intervals of background activity.” A manual has been established for analyzing these patterns, and clinical correlates are being identified. See Chapter 104 Appendix, “Technical Considerations and Scoring Criteria.”
Automatic Sleep Stagers

Although many groups in the United States continue to analyze sleep data using manual scoring of polysomnographic or digitized acquired tracings, several systems for automatically staging sleep have been proposed, and a number are commercially available. Certain of these systems are based on the standard guidelines, although several approaches that confront the sleep staging issues more from the perspective of available technologies have also been used. Thus, instead of adapting digital computer technology to human eyeball scoring criteria, they use such techniques as frequency spectrum analysis or multidimensional scaling, adaptive segmentation and fuzzy subset theory, or expert systems approaches. No single automated stage has yet emerged as the ideal alternative to hand scoring, and space does not permit a review of available systems. The following questions are offered as a basis for evaluating automatic sleep staging systems:

1. Has the system been validated against another known assessment technique?
2. Is the system valid for the types of studies for which it will be used—for example, sleep only, sleep and breathing, sleep and movements, and so forth?
3. Is the system valid for the types of patients in whom it will be used—for example, patients with sleep apnea versus patients with narcolepsy, medicated versus nonmedicated patients, and so on?
4. Is the system valid for the age groups in which it will be used—children versus adults versus older adults?
5. Is the system compatible with available laboratory hardware?
6. Does the system require excessive operator input (e.g., knob turning, "tweaking," fine tuning) that takes as much time as hand scoring?
7. Does the system provide output verification? That is, can the raw data be reviewed?
8. If the system does not automatically assess relevant events, can hand-scored events be accurately correlated to the stage's output?
9. Is the system sufficiently flexible to support new foreseeable applications? Such applications might include changes in patient population, recording equipment, research orientation, and so forth.
10. Does the system reliably identify differences known to affect sleep staging, such as age, prior sleep history, and diagnosis?
11. Is the system supported by accessible consultants?

SUMMARIZING SLEEP STAGE DATA

After the sleep data are scored, they must be summarized into a comprehensible form. No consensus format has been achieved, and certain areas of controversy exist; however, a number of conventions are fairly common and include the following types of analysis. (Alternative calculation paradigms are sometimes used and have been noted where appropriate.) Figure 116–16 shows the output of one type of sleep data summary sheet.

Stages

A summary of the night of sleep will invariably include the time spent in each of the sleep stages, as well as the time awake and movement time. This type of summary is relatively straightforward and noncontroversial. Calculation of percentage distributions is not quite as clear cut, as various groups may calculate sleep stage percentages based on total recording time (dark time), on total sleep time (total NREM stages 1 to 4 plus REM), or on sleep period time (time from sleep onset to sleep offset, including intervening arousals). The example in Figure 116–16 uses all three alternatives.

Latencies

The topic of latencies is associated with a certain amount of controversy, particularly because the type of patient may affect the appropriateness of specific definitions. Thus, although sleep latency, defined as elapsed time from lights out until the first of three consecutive epochs of stage 1 or the first of any other stage, may be appropriate for normal, noncomplaining individuals or for hypersonomolent patients, it may not be appropriate for patients with sleep onset insomnia. One alternative to this definition requires stage 2 sleep (spindle or K-complex) to define sleep onset. To account for instances in which a patient may have 2 or 3 minutes of sleep followed by a lengthy awakening, definitions that require, for example, 5 consecutive minutes of sleep have been suggested by others. The issue of the definition of sleep onset is not trivial because latencies to stage 4 or REM sleep are generally calculated from sleep onset. Certain analysis programs have the capability to provide several calculations of sleep onset, whereas others provide the flexibility to redefine the criterion for individual cases. In the absence of a comprehensive database, it is not possible to make a sweeping generalization. A safe alternative for most clinical studies is probably to choose the conservative 5-minute rule or even a 10-minute rule, although a briefer requirement might be more appropriate for patients with sleep syndromes in which frequent awakenings may preclude such a sleep onset entirely.

Once a definition of sleep onset is established, determining stage 3 or 4 or REM latency is fairly straightforward: elapsed time from the (start of) defined sleep onset to the first epoch of stage 3 (or 4) or REM sleep. Certain groups may apply a three-epoch rule to this definition—that is, three consecutive epochs of the target stage are required. One point of dispute regarding REM latency calculation concerns whether or not to include any waking intervals that may occur between sleep onset and REM onset. No firm recommendation can be made; however, it is still generally assumed that waking is included in the calculation, unless otherwise noted. Such considerations are crucial when data are compared across groups, which is particularly relevant when norms from another laboratory are being used.

Cycles

A description of the NREM-REM cycle is a common feature of the night’s sleep summary. Unfortunately, the defining characteristics for such a description are not standardized, and therefore, a number of idiosyncratic approaches have been used. One common way of defining cycles is as the elapsed time from the end of each REM episode to the end of the next REM episode, whereas another uses the time from the start of one REM episode to the start of the next. The consequences of choosing one alternative over the other have not been clearly established. Another difficulty for defining REM cycles arises
**T.A.**

Subject's name: T.A.
Subject's gender: M
Subject's age: 14.0000 years
Subject's date of birth: 1/11/73
Subject's tanner stage: 1
Recording date: 1/11/87
Name of study: T/A

**T/A, Pretreatment**

Milestones:
- Lights out: 22:14:00 (Epoch 27 of page 1)
- Sleep onset: 22:28:33 (Epoch 57 of page 1)
- Last sleep epoch: 7:19:00 (Epoch 602 of page 13)
- End of night: 8:00:30 (Epoch 685 of page 13)

**Group:**
- Recording condition: Pretreatment
- Recording technician: Carlskodon
- Scoring technician: Mancuso
- Data entry technician: Mancuso

Minimum epoch length: 0.4800 minutes (Epoch 478 of page 11)
Maximum epoch length: 0.5294 minutes (Epoch 965 of page 10)
Average epoch length: 0.4975 minutes

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**REM summary:**

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Cycles: 192.77 111.64 104.66 111.89 520.95 130.24
End of last REM from end of night: 51:00

**Analysis by fraction (1/3):**

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*Figure 116–16.* A sleep stage summary sheet. This sample represents only one of many possible summary formats. The data are taken from a night of sleep recorded in a 14-year-old boy with enlarged tonsils and adenoids who had moderately disordered breathing during sleep. The 25-minute combining rule was used to define REM sleep periods. The following definitions were used to derive specific items presented in this summary: SPT, sleep period time (elapsed time from sleep onset to last epoch of sleep); TDT, total dark time (elapsed time from lights out to end of night); TMT, total amount of movement time; TNREM, total stages 1 + 2 + 3 + 4 sleep; TREM, total amount of REM sleep; TS1 to TS4, total amount of stage 1, 2, 3, and 4 sleep; TST, total sleep time; TSW, total amount of stages 3 + 4 sleep; TWT, total amount of wakefulness; WAFA, wake time after final awakening; WASO, wake time after sleep onset. Definitions in the REM summary are as follows: Cycles, elapsed time from sleep onset to end of first REM period, from end of first to end of second REM period, and so on; REMT, amount of REM sleep; S1, S2, WT, and MT, as defined for their total amounts; SEG, number of REM sleep segments within the REM period; TT, total time of the REM episode. All other items are self-explanatory.
Partitioning the Night

For many years, it has been a common practice to examine at least waking, slow wave (stages 3 plus 4) sleep, and REM sleep by thirds of the night. This practice, at least for REM sleep, seems to have originated in early studies of insomnia and sleeping pills. Its usefulness derives primarily from normative studies in young adults, in which one sees a predominance of stage 3 and 4 NREM sleep in the first third of sleep and a predominance of REM sleep toward the last third of sleep. In patients with insomnia, preferential distribution of waking to a third of the night may provide insight regarding the type of sleep problem. Although these specific comparisons are not always useful or appropriate, the thirds-of-night analysis remains a valuable thumbnail description of a night’s sleep. Other variations of this partitioning technique may use halves or quarters of the night or even hour-by-hour assessment (Fig. 116–18).
Events

Events coded during sleep (see Table 116-2) are frequently tabulated according to whether they occurred in NREM or in REM sleep. Finer distinctions are rarely made. When the event (e.g., respiratory disturbance) spans more than one epoch, it is recommended that it be catalogued according to the stage in which it began.

Sleep Hypnogram

Another way to examine events is in correlation with the ongoing pattern of nocturnal sleep, as visualized using hypnogram plotting techniques. Figure 116-19 shows an example of such a plot. Sleep stages and transitions are illustrated in the upper portion of the plot and show the unfolding of sleep versus time. This type of graphic display has been used from the earliest modern sleep studies. Events are usually plotted below the histogram, aligned temporally with their occurrence. This plotting technique provides a sometimes very helpful visual representation of the data, and many software packages that provide data reduction of sleep stages and events also have such plotting capabilities.

WHY STAGE SLEEP?

A number of investigators and clinicians coming to the field of sleep from other disciplines question the necessity for evaluating sleep stages, particularly in clinical conditions. Thus, for example, a pulmonary specialist may question sleep staging in a patient with apnea for whom the key issues to the specialist may be length of apnea, degree of desaturation, cardiac arrhythmias, and so forth. A urologist’s focus may be penile circumference, which does not require distinctions of sleep staging. Thus, an argument can be and has been made for focusing on the pathologic event rather than sleep per se. A few counterarguments follow.

Regulatory Physiology Differs from Waking to NREM Sleep to REM Sleep

As increasing numbers of systems are evaluated during naturally occurring wakefulness and sleep, it has become quite clear that many regulatory mechanisms are affected by state. For example, the ventilatory responses to oxygen and carbon dioxide (see Chapter 18) are somewhat damped in NREM sleep and may be absent in REM sleep. Another dramatic example concerns thermoregulation. Thermoregulatory responses are only slightly altered in NREM sleep and almost totally lacking in REM sleep. Such marked state-dependent alterations in regulatory physiology must be taken into account to assess fully the implications of observed sleep-related pathologies.

![Figure 116-19. A sleep histogram of the same night of sleep summarized in Figure 116-16. The sleep histogram provides a graphic display of the night using an analogue plot of sleep-wake stages across time (upper portion). Arrayed beneath this plot are event markers, which are temporally aligned.](image-url)
Pathologic Events Disturb Sleep

Patients with sleep apnea syndromes, for example, have markedly disrupted sleep. In children, the disruption may preferentially reduce stage 3 and 4 sleep, and sleep apnea may be associated with growth problems. In adults, sleep apnea is more likely to occur during, and be disruptive of, REM sleep. The possible clinical relevance of such a sleep disturbance in a child is obvious but cannot be appreciated if sleep stages are not evaluated. Documentation of recovery sleep after treatment may also provide insights into therapeutic efficacy that may be unrelated to the pathologic events (i.e., the apneas) per se.

Arousals consequent to sleep pathologies are also clinically relevant and require assessment to fully characterize the pathology. Thus, as mentioned previously, arousals are clearly related to daytime sleepiness. In the case of patients with sleep apnea syndromes, a given treatment may improve the apnea—as documented by the maintenance of arterial oxygen saturation (SaO2) at more than 85%, reduction in cardiac arrhythmias, and conversion of apneas to hypopneas—yet the patient may still suffer arousals from sleep sufficient to impair waking function or to be associated with a vulnerability to unintentional sleep episodes. Hence, it is relevant to evaluate sleep and arousals as well as the respiratory function in such patients. Arousals may be a relevant issue in the case of periodic movements during sleep as well. One study has documented clinical improvement of patients in whom periodic movements during sleep were treated with benzodiazepine hypnotics, although the number of movements was unchanged from pretreatment. The number of associated arousals and the amount of transitional stage 1 sleep were reduced, however—a factor that would have been overlooked had sleep staging not been performed.

Sleep State May Affect Pathology

It has been known for many years that penile tumescence occurs in association with REM sleep in normal males of virtually all ages. This phenomenon has been capitalized on to assess erectile dysfunction by recording REM sleep-related nocturnal penile tumescence (NPT). Various clinicians, however, have attempted to perform studies of NPT in patients outside the sleep laboratory using such techniques as the "postage stamp" method. Other methodologic considerations aside (because the argument obtains even if appropriate NPT techniques are used without monitoring sleep stage), one cannot achieve a valid test of NPT if sleep is not assessed. This is true simply because an "abnormal" NPT can result if REM sleep is abnormal, disrupted, or absent. Sleep disorders themselves may have an impact on NPT as well. Therefore, without evaluating sleep, it is not possible to determine whether tumescence did not occur because erectile function was impaired or because sleep was disturbed.

Another example again concerns sleep apnea syndromes. As mentioned previously, sleep apneas in a percentage of adult patients may occur preferentially in REM sleep. It has been suggested that diagnostic assessment of sleep apneas can be performed by monitoring respiration during a daytime nap. In this case, in particular, sleep monitoring is essential because REM sleep may not occur during a daytime nap (depending on the time of the nap), and, therefore, the severity of sleep apnea may be very greatly underestimated. Without documentation of sleep state, such important clinical judgments (including optimal continuous positive airway pressure) may not be possible.

An Abnormal Sleep Architecture May Be a Marker of Pathology

Patients with narcolepsy often enter sleep through REM sleep rather than experiencing the normal transition from waking to NREM sleep. Because the symptom presentation of narcolepsy is variable, it is relevant to document the sleep onset transition in patients with complaints of hypersomnia. Relatively short REM onset latencies are also thought to be a marker of endogenous depression.

In summary, laboratory monitoring and staging of sleep remain important components in the assessment of patients with sleep disorders. The techniques derive directly from the earliest studies following the discovery of REM sleep in the 1950s, and some might criticize that the procedures have not kept pace with technologic advances. Because automated, ambulant systems that are inexpensive, validated, and reliable are marketed, it is likely that polysomnography has begun to advance accordingly.

The Newborn Infant

Because of rapid changes in the nervous system after birth, the well-defined stages seen in the adult are not present, and special criteria are used to define states. The standard scoring manual for neonates defines sleep stages using behaviors, respiration, eye movements, the EEG, and muscle tone. The following states were defined: active-REM sleep, quiet sleep, and indeterminate sleep. Criteria for defining these states with examples of sleep recordings in neonates are found in the standard scoring manual for neonates.

Clinical Pearls

Although ancillary physiologic data collected during sleep are important for many diagnostic purposes, sleep staging and assessment of arousals remain crucial for a full evaluation of sleep and sleep-related processes. Sleep stage scoring in clinical settings may require certain modifications from the standard manual, such as combining stages 3 and 4 to slow wave sleep or collapsing across NREM stages. The appropriate definition of sleep onset latency may vary according to the nature of the clinical condition under study. Arousals during sleep are valuable to understanding the sleep-disrupting impact of clinical conditions that might otherwise be unappreciated.

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