INTRODUCTION TO SLEEP ELECTROENCEPHALOGRAPHY

SELIM R. BENBADIS
University of South Florida College of Medicine, Tampa, Florida

INTRODUCTION

Basic Principles
EEG records synaptic potentials from pyramidal cells. It is critical to remember that there is no absolute voltage measurement in clinical EEG. Rather, an EEG trace records potential differences between two electrodes. By convention of the amplifiers used in clinical EEG, an upgoing deflection indicates that input 1 (grid 1) is more negative (or less positive) than input 2 (grid 2). This is an arbitrary but critical rule (Figure 130.1). Thus statements like "positivity is up" or "negativity is up" make no sense unless it is stated whether the positivity is at grid 1 or grid 2.

It should be emphasized that the way recordings are displayed can be modified at will with digital acquisition, and this "post-hoc" reformatting is one of the major advantages of modern digital systems.

Types of EEG in Clinical Practice
Routine EEG is typically a brief recording of 20–30 min. The main limitation with EEG is its poor sensitivity for epilepsy. The generally accepted numbers are that the yield of a single routine EEG in epilepsy is ~50% and increases with repeated EEG recordings to reach about 80% by the third recording [1]. For practical purposes, if a diagnosis of epilepsy is strongly suspected clinically, and EEG confirmation or more precise diagnosis is needed, other options should be used. The two options are ambulatory EEG and prolonged EEG-video monitoring.

Ambulatory EEG is to the brain what Holter monitoring is to the heart. Here the patient is hooked up to the EEG and goes home with the intent of recording a seizure or an episode [2]. Ambulatory EEG can occasionally be performed with video, and this option is now emerging.

EEG-video monitoring is the highest level of epilepsy monitoring and the gold standard. This is the basic activity of comprehensive epilepsy centers [3] and certainly the starting point when drugs fail to control seizures [4]. There is no strict "cutoff" for when EEG-video monitoring is indicated, but some guidelines state that referral to a specialized epilepsy center is appropriate if seizure control is not achieved within 9 months [5]. As a general rule, prolonged EEG-video monitoring should be obtained on any patient who continues to have seizures frequently (1/week) despite antiepileptic drugs [4]. In the vast majority of situations, this allows one to confirm the diagnosis of epilepsy or to rectify a wrong diagnosis of epilepsy. If epilepsy is confirmed, it is then usually possible to (1) determine whether it is localization-related or generalized; (2) distinguish, among generalized epilepsies, between the "idiopathic" type and the symptomatic (cause known) or cryptogenic (caused unknown); and (3) differentiate, among localization-related epilepsies, between mesiotemporal and extratemporal/neocortical epilepsy. Based on this precise classification of the epilepsy syndrome, treatment options can then be examined. Invasive EEG is
limited to specialized surgical epilepsy centers and is beyond the scope of this chapter (for review, see [6, 7]).

Technical Aspects

In human clinical EEG, electrodes are placed according to a standard system known as the 10-20 system (Figure 130.2). It uses four anatomical landmarks (nasion, inion, and the two preauricular points) from which measurements are made and electrodes are placed at 10% or 20% of the distances.

There are two types of montages: bipolar and referential. In a bipolar montage, each electrode is linked to the next along a chain (i.e., A–B, B–C, C–D, D–E). The typical longitudinal or anteroposterior bipolar montage is often referred to as a “double banana.” Another common bipolar montage is the transverse (from left to right across the head). In a referential (or monopolar) montage, each electrode is linked (compared) to a common reference. A helpful analogy is that measuring voltage fields with electrodes is akin to measuring mountain peaks/altitudes with surveyors [8] (Figure 130.3). In terms of localization, maximum voltage (altitude) is indicated by a phase reversal on bipolar montages, or by maximum amplitude on referential montages (Figure 130.3). Contrary to a common misconception, phase reversals are not at all indicative of an abnormality, and in fact have nothing to do with the nature of a voltage field (i.e., what the discharge is). Instead, phase reversals indicate the maximum (negativity or positivity)

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**Polarity and phase reversals**

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\begin{align*}
  &A-B \\
  &B-C \\
  &C-D \\
  &D-E \\
\end{align*}
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**Figure 130.3** Measuring a voltage field is analogous to measuring the altitude of a mountain. (a) On a bipolar montage, a maximum peak (voltage) is indicated by a phase reversal. (b) On a referential montage, a maximum peak (voltage) is indicated by the highest amplitude.

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**Figure 130.2** The 10–20 system of electrode placement. This uses four anatomical landmarks (nasion, inion, and the two preauricular points) from which measurements are made and electrodes are placed at 10% or 20% of the distances. Letters refer (grossly) to the lobe (i.e., Fp, frontopolar; F, frontal; T, temporal; P, parietal; O, occipital). Odd-numbered electrodes are on the left, even-numbered electrodes on the right, and midline electrodes are designated as “z.”

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**Polarity Convention**

<table>
<thead>
<tr>
<th>G1</th>
<th>G2</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>-</td>
<td>+</td>
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<tr>
<td>+</td>
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</tbody>
</table>

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**Figure 130.1** Polarity convention. This is an arbitrary but critical rule. By convention of the amplifiers used in clinical EEG, an upgoing deflection indicates that input 1 (grid 1 or G1) is more negative (or less positive) than input 2 (grid 2 or G2). Thus statements like “positivity is up” or “negativity is up” make no sense unless it is stated whether the positivity is at grid 1 or grid 2.

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**10-20 system & montages**

- **Longitudinal bipolar**: (double banana)
  - Fp1-F7
  - F7-T3
  - T3-T5
  - T5-O1
- **Transverse bipolar**
  - T3-C3
  - C3-Cz
  - Cz-C4
  - C4-T4
- **Referential (monopolar)**
  - Fp1-A1
  - F7-A1
  - T3-A1
  - T5-A1

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**Figure 130.4**
and have everything to do with the location of the discharge (i.e., where the discharge is).

The display on EEG typically uses a “paper speed” (obsolete concept with digital recordings) of 3 cm/s (in the United States), that is, 3 times faster than the PSG “paper speed” of 1 cm/s. By convention, in the United States, montages are usually displayed “left over right.”

Filters and their principles are similar to those used in polysomnography.

In clinical neurophysiology (e.g., EEG, PSG), waveforms and discharges are described and characterized by the following parameters:

- **Amplitude:** how high the voltage is (in µV).
- **Duration:** how long the discharge is (in ms or seconds).
- **Frequency:** how frequently a waveform repeats itself (in cycles per second or hertz). The frequency is the reciprocal of the duration (e.g., a duration on 200 ms is the same as a frequency 0.5 Hz.)
- **Morphology:** the shape and configuration of the discharge (this is qualitative).
- **Latency:** the delay between an arbitrary event (e.g., stimulus) and another event (in ms).
- **Location:** where the discharge is.
- **Reactivity:** what affects the discharge.

### ARTIFACTS

Although EEG is designed to record cerebral activity, it also records electrical activities arising from sites other than the brain. The recorded activity that is not of cerebral origin is termed artifact and can be conveniently divided into physiologic and extraphysiologic artifacts. **Physiologic artifacts** are generated by the body but arise from sources other than the brain. **Extraphysiologic artifacts** arise from outside the body (i.e., equipment, environment).

#### Physiologic Artifacts

**Muscle (Electromyogram) Activity**  Myogenic potentials are probably the most common artifacts and are seen on virtually all EEG performed in clinical practice. Frontalis and temporalis muscles (e.g., clenching of jaw muscles) are particularly common. As a general rule, the potentials generated in the muscles are of shorter duration than those generated in the brain and are identified easily on the basis of duration, morphology, and high frequency of 50–100 Hz (Figures 130.4 and 130.10). A particular type of muscle artifact is chewing, characterized by rhythmic bursts of muscle maximum in the temporal chains (Figure 130.5).

![Figure 130.4](https://example.com/130.4.png)  **Figure 130.4**  Muscle (EMG) and EKG artifact. Muscle artifact is seen as high-amplitude, very fast (>50 Hz), and variable frequency discharges, which are more prominent on the left in this sample. EKG is easily identified in several channels as clearly simultaneous to the EKG channel.
**Glossokinetic Artifact**  In addition to muscle activity, the tongue (like the eyeball) functions as a dipole, with the tip negative with respect to the base. In this case the tip of the tongue is the most important part because it is more mobile. The artifact produced by the tongue has a broad potential field that drops from frontal to occipital areas, although it is less steep than that produced by eye movement artifacts. The amplitude of the potentials is greater inferiorly than in parasagittal regions; the frequency is variable but usually in the delta range and occurs synchronously when the patient says “Lah-lah-lah-lah,” which can be verified by the technologist. Chewing and sucking (pacifier) can produce similar artifacts.

**EKG Artifact**  Some individual variations in the amount and persistence of EKG artifact are related to the field of the heart potentials over the surface of the scalp. Generally, subjects with short and wide necks have the largest EKG artifacts on their EEG recordings. The voltage and apparent surface of the artifact vary from derivation to derivation and, consequently, from montage to montage. The artifact is observed best in referential montages using ear electrodes (A1 and A2). EKG artifact is recognized easily by its rhythmicity and coincidence with the EKG tracing (Figure 130.4). The situation becomes difficult when abnormal cerebral activity (e.g., sharp or slow waves) appears intermixed with EEG artifact.

**Pulse**  Pulse artifact occurs when an EEG electrode is placed over a pulsating vessel. The pulsation can cause slow waves that may simulate EEG activity. A direct relationship exists between EKG and the pulse waves. The QRS

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**Figure 130.5**  Chewing artifact. This EMG artifact occurs in rhythmic bursts of muscle maximum in the temporal chains.
complex (i.e., electrical component of the heart contraction) happens slightly ahead of the pulse waves 200–300 msec delay after EKG.

**Eye Movements**  
Eye movements are observed on all EEG recordings and are useful in identifying sleep stages. The eyeball acts as a dipole with a positive pole anteriorly (cornea) and a negative pole posteriorly (retina). When the globe rotates about its axis, it generates a large-amplitude alternate current field, which is detectable by any electrodes near the eye. The other source of artifacts comes from EMG potentials from muscles in and around the orbit. Vertical eye movements typically are observed with blinks (i.e., Bell's phenomenon). A blink causes the positive pole (i.e., cornea) to move closer to frontopolar (Fp1–Fp2) electrodes, producing symmetric downward deflections. During downward eye movement the positive pole (i.e., cornea) of the globe moves away from frontopolar electrodes, producing an upward deflection best recorded in channels 1 and 5 in the bipolar longitudinal montage. Lateral eye movements affect lateral frontal electrodes F7 and F8 (which are just about where “eye electrodes” of the PSG would be). During a left lateral eye movement, the positive pole of the globe moves toward F7 and away from F8. Using a bipolar longitudinal montage, there is a maximum positivity in electrode F7 and maximum negativity simultaneously in electrode F8 (Figure 130.6). A so-called lateral rectus spike (Figure 130.7) may be present in electrode F7. With right lateral eye movement, the opposite occurs.

*Figure 130.6*  
Eye movements and sawtooth waves in REM sleep. During this rapid lateral eye movement to the left, there is a maximum positivity in electrode F7 and maximum negativity simultaneously in electrode F8. In the vicinity of the rapid eye movement, there are typical sawtooth waves in the central region (C3 and C4).
Respiration Artifacts  Respiration can produce two kinds of artifacts. One type is in the form of slow and rhythmic activity, synchronous with the body movements of respiration and mechanically affecting the impedance of (usually) one electrode. The other type can be slow or sharp waves that occur synchronously with inhalation or exhalation and involve those electrodes on which the patient is lying. Several commercially available devices to monitor respiration can be coupled to the EEG machine. As with the EKG, one channel can be dedicated to respiratory movements.

Skin Artifacts  Biological processes may alter impedance and cause artifacts. Sweat is a common cause (Figure 130.8). Sodium chloride and lactic acid from sweating react with metals of the electrodes and produce large and very slow (usually ~0.5 Hz) baseline sways.

Extraphysiologic Artifacts

Electrode Artifacts  The most common electrode artifact is the electrode “pop.” Morphologically this appears as single or multiple sharp waveforms due to abrupt impedance change. It is identified easily by its characteristic appearance (i.e., abrupt vertical transient that does not modify the background activity) and its usual distribution, which is limited to a single electrode (Figure 130.9). In general, sharp transients that occur at a single electrode (i.e., no field) should be considered artifacts until proved otherwise. At other times, the impedance change is less abrupt,
Figure 130.8  Sweat artifact. Note the very slow (0.5 Hz) sways. The slow frequency is similar to slow rolling eye movements, but the distribution is not (in this case it affects electrodes on the left side of the head).

Figure 130.9  Electrode artifact. This typical electrode artifact is the electrode “pop.” Note the single sharp waveform with abrupt vertical transient that does not modify the background activity, and its distribution, which is limited to a single electrode (P4).
and the artifact may mimic a low-voltage arrhythmic delta wave.

**Alternating Current (60 Hz) Artifact** Adequate grounding on the patient has almost eliminated this type of artifact from power lines. The problem arises when the impedance of one of the active electrodes becomes significantly large between the electrodes and the ground of the amplifier. In this situation, the ground becomes an active electrode that, depending on its location, produces the 60 Hz artifact. The artifact has the exact frequency of 60 Hz and is easily identified by increasing the time base (Figure 130.10).

**Movements in the Environment** Movement of other persons around the patient can generate artifacts, usually of capacitive or electrostatic origin. The artifact produced by respirators varies widely in morphology and frequency. Monitoring the ventilator rate in a separate channel helps to identify this type of artifact. Interference from high-frequency radiation from radio, TV, hospital paging systems, and other electronic devices can overload EEG amplifiers. The cutting or coagulating electrode used in the operating room also generates high-voltage high-frequency signals that interfere with the recording system. Touching or hitting electrodes can produce odd waveforms,

![Figure 130.10](image)

**Figure 130.10** The 60 Hz and muscle (EMG) artifact. The 60 Hz artifact is at electrode O2 (thus seen in channels T6–O2 and P4–O2). Note that its amplitude is perfectly regular and exactly and steadily at 60 Hz. EMG (muscle) artifact, by contrast, is of comparable frequency but affects several electrodes, is variable in amplitude, and variable in frequency (not always nor exactly at 60 Hz).
and, for example, repetitive head movements can produce rhythmic artifacts (Figure 130.11).

**Photic Stimulation**

*Photic stimulation* is performed during routine EEG recordings and can produce some physiologic and some extraphysiologic artifacts, so it is described here separately.

*Photic driving* is a normal physiologic response, which is actually a visual evoked potential. It is the occipital cortex response to flashing lights and is thus seen in the occipital region (electrodes O1 and O2). It is easily identified because it is “time-locked” with (same frequency as) the strobe light (Figure 130.12).

The *photomyoclonic response* is a special type of EMG artifact that occurs during intermittent photic stimulation. Some subjects contract the frontalis and orbicularis muscles. This superficially resembles normal photic driving but is frontal (Figure 130.13), whereas normal photic driving is occipital. As can be shown by spreading the time base, these contractions occur approximately 50–60 m after each flash.

A *photocell (photoelectric) artifact* can also be seen with photic stimulation. This affects one electrode (Figure 130.14) and is easily identified as it disappears if one blocks the light from the electrode in question.

**THE NORMAL EEG**

**Common Patterns of Wakefulness**

*Alpha Rhythm* The alpha rhythm (Figure 130.15) is typically what EEG readers identify first. The normal alpha rhythm has the following characteristics:

- Frequency of 8–12 Hz: Lower limit of normal generally accepted in adults and children older than 8 years is 8 Hz.
- Location: Posterior dominant; occasionally, the maximum may be a little more anterior, and it may be more widespread.
- Morphology: Rhythmic, regular, and waxing and waning.
Figure 130.12  Normal photic driving. Bioccipital rhythmic activity time-locked with (same frequency as) the strobe light (the flash frequency is shown as lines at the bottom). Usually, photic driving is seen at several frequencies, such as shown here. Note that only location differentiates this from (frontal) photomyoclonic (or photomyogenic) response (Figure 130.13).

Figure 130.13  Photomyoclonic (or photomyogenic) response. Bifrontal rhythmic activity time-locked with (same frequency as) the strobe light (the flash frequency is shown as lines at the bottom). Note that only location differentiates this from (occipital) photic driving (Figure 130.12).
Amplitude: Generally 20–100 μV.
Reactivity: Best seen with eyes closed; attenuates with eye opening.

Occasionally the alpha rhythm is of very low amplitude or even not identifiable. This is not in itself abnormal. In addition to amplitude, other characteristics can vary somewhat without being abnormal, including morphology (e.g., spiky), distribution (e.g., widespread), and harmonic frequency (e.g., slow or fast alpha variant).

**Beta Activity** Normal beta activity (Figure 130.16) has the following characteristics:

- Frequency (by definition) greater than 13 Hz, typically 18–25 Hz.
- Location: Mostly frontocentral but somewhat variable; some describe various types according to location and reactivity—generalized, precentral, and posterior.
- Morphology: Usually rhythmic, waxing and waning, and symmetric.
- Amplitude: Usually in the range of 5–20 μV.
- Reactivity: Most common 18–25 Hz beta activity enhanced during stages 1 and 2 sleep and tends to decrease during deeper sleep stages; central beta activity may be reactive (attenuates) to voluntary movements and proprioceptive stimuli; in infants older than 6 months, onset of sleep marked by increased beta activity in central and postcentral regions.

In healthy individuals, beta activity commonly can be mildly different (<35%) in amplitude between the two hemispheres, which may be caused by differences in skull thickness. The amount and voltage of beta activity is enhanced by commonly used sedative medications (benzodiazepines, barbiturates).
Mu Rhythm  Characteristics of the mu rhythm (Figure 130.17) are as follows:

- Frequency of 7–11 Hz: Generally in alpha frequency band (8–12 Hz).
- Location: Centroparietal area.
- Morphology: Arch-like shape or like an “m”; most often asymmetric and asynchronous between the two sides and may be unilateral.
- Amplitude: Generally low to medium and comparable to that of the alpha rhythm.
- Reactivity: mu rhythm attenuates with contralateral extremity movement, the thought of a movement, or tactile stimulation.

Asymmetry, unilaterality, or asynchrony of the mu rhythm is not abnormal unless associated with other abnormalities. Very high voltage mu activity may be recorded in the central regions over skull defects and may become sharp in configuration and thus can be mistaken for epileptiform discharges. When mu rhythm is detected in an EEG, it should be verified by testing its reactivity.

Common Patterns of Sleep

Sleep Architecture  Sleep generally is divided in two broad types: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. On the basis of EEG changes, NREM sleep is divided further into four stages (stage 1, stage 2, stage 3, stage 4). REM sleep is defined not only
Figure 130.16 Normal beta activity. This sample depicts the typical fast (~18–25 Hz) low-amplitude activity in both frontal regions. As shown here, normal amounts of beta activity are moderate and tend to wax and wane.

Figure 130.17 Mu rhythm. This sample depicts the typical m-shaped bicentral bursts at a frequency of 8–12 Hz. If tested, this would react (attenuate) to contralateral movement.
by EEG, but also by EMG and eye movements. NREM and REM sleep occur in alternating cycles, each lasting approximately 90–100 minutes, with a total of 4–6 cycles. In general, in the healthy young adult NREM sleep accounts for 75–90% of sleep time (3–5% stage 1, 50–60% stage 2, and 10–20% stages 3 and 4). REM sleep accounts for 10–25% of sleep time.

Total sleep time in the healthy young adult approximates 5–10 hours. In the full-term newborn, sleep cycles last approximately 60 minutes. The newborn sleeps approximately 16–20 hours per day, with a higher proportion (~50%) of REM sleep.

**Stage 1 (“Drowsiness”)** Slow rolling eye movements (SREMs) (Figure 130.18) are usually the first evidence of drowsiness seen on the EEG. SREMs of drowsiness most often are horizontal but can be vertical or oblique, and their distribution is similar to eye movements in general. However, they are slow (i.e., typically 0.25–0.5 Hz). Because of their frequency, SREMs superficially resemble sweat artifacts but are easily identified by their nonrandom distribution typical of eye movements (e.g., phase reversals at F7 and F8 if horizontal). SREMs disappear in stage 2 and deeper sleep stages.

**Alpha activity dropout** (Figure 130.18) typically occurs together with or nearby SREMs. The alpha rhythm gradually becomes slower, less prominent, fragmented, and disappears.

**Vertex sharp transients** (Figures 130.19 and 130.20), also called vertex waves or V waves, are almost universal. Although they often are grouped together with K-complexes, strictly speaking, vertex sharp transients are distinct from K-complexes because they are briefer in duration, smaller in amplitude, and more focal (i.e., less widespread). Like K-complexes, vertex waves are maximum at the vertex (central midline placement of electrodes (Cz)), so that, depending on the montage, they may be seen on both sides, usually symmetrically, at C3 and C4. Their amplitude is 50–150 μV. They can be contoured sharply and occur in repetitive runs, especially in children. They persist in stage 2 sleep but usually disappear in subsequent stages. Unlike K-complexes, vertex waves do not define stage 2.

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**Figure 130.18** Drowsiness (stage 1 sleep): slow rolling eye movements. Note the very slow (0.3–0.5 Hz) oscillations at F7 and F8. Like rapid eye movements, a negativity at F7 occurs at the same time as a positivity at F8 for an eye movement to the right, and vice versa for an eye movement to the left. Also note the attenuation and slowing of the alpha rhythm (alpha dropout) between the first and second half of the sample. This is the other early finding of stage 1 sleep.
Positiv occipital sharp transients of sleep (POSTS) (Figure 130.19) are seen very commonly on EEG and have been said to be more common during daytime naps than during nocturnal sleep. Most characteristics of POSTS are contained in their name. They have a positive maximum at the occiput, are contoured sharply, and occur in early sleep (stages 1 and 2). Their morphology is best described as “reversed checkmark,” and their amplitude is 50–100 μV. They typically occur in runs of 4–5 Hz and are bisynchronous, although they may be asymmetric. They persist in stage 2 sleep but usually disappear in subsequent stages.

Hypnagogic hypersynchrony (Figure 130.21) is a less common but well recognized normal variant of drowsiness in children aged 3 months to 13 years. This is described as paroxysmal bursts (3–5 Hz) of high-voltage (as high as 350 μV) rhythmic waves, maximally expressed in the prefrontal-central areas.

Stage 2 Sleep spindles (Figure 130.22) normally first appear in infants aged 6–8 weeks and are initially asynchronous, becoming synchronous by the age of 2 years. Sleep spindles have a frequency of 12–16 Hz (typically 14 Hz) and are maximal in the central region (vertex), although they occasionally predominate in the frontal regions. They occur in short bursts of waxing and waning fusiform rhythmic activity. Amplitude is usually 20–100 μV. Less typical or “extreme” spindles (described by Gibbs and Gibbs) are unusually high-voltage (100–400 μV) and prolonged (>20 s) spindles located over the frontal regions.

K-complexes (Figure 130.23) are high amplitude (>100 μV), broad (>200 ms), diphasic, transients often associated
with sleep spindles. Location is frontocentral, with a typical maximum at the midline (central midline electrodes (Cz) or frontal midline electrodes (Fz)). They occur spontaneously or as an arousal response.

**Stage 3/4** Slow-wave sleep (SWS), or delta sleep, is characterized, as the name implies, by delta activity. This typically is generalized and polymorphic or semirhythmic (Figure 130.24). By strict sleep staging criteria on polysomnography, SWS is defined by the presence of such delta activity for more than 20% of the time, and an amplitude criterion of at least 75 \( \mu \text{V} \) often is applied.

The distinction between stages 3 and 4 is only a quantitative one that has to do with the amount of delta activity. Stage 3 is defined by delta activity that occupies 20–50% of the time, whereas in stage 4 delta activity represents greater than 50% of the time. Sleep spindles and K-complexes may persist in stage 3 and even to some degree in stage 4, but they are not prominent.

As mentioned earlier, SWS usually is not seen during routine EEG, which is too brief a recording. However, it is seen during prolonged EEG monitoring. One important clinical aspect of SWS is that certain parasomnias occur specifically out of this stage and must be differentiated from seizures. These “slow-wave sleep parasomnias” include confusional arousals, night terrors (pavor nocturnus), and sleepwalking (somnambulism).

**REM Sleep** REM sleep normally is not seen on routine EEG recordings, because the normal latency to REM sleep (100 min) is well beyond the duration of routine EEG recordings (approximately 20–30 min). The appearance of REM sleep during a routine EEG is referred to as sleep-onset REM period (SOREMP) and is abnormal and warrants an MSLT. While not observed on routine EEG, REM sleep commonly is seen during prolonged (>24 h) EEG monitoring.