Chapter 12 – Electroencephalography, Electromyography, and Electro-Oculography : General Principles and Basic Technology

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INTRODUCTION

Electroencephalography (EEG) has played a central role in the beginnings and evolution of sleep medicine. Many would agree that modern sleep medicine started with the prolonged EEG studies of sleep by Kleitman, culminating in the historic discovery (with Aserinsky) of rapid eye movement (REM) sleep in 1953.[1] This and other seminal early reports emphasized that studying sleep required measurement of electrocerebral activity as well as other physiologic functions such as eye movements and axial electromyography; thus traditional EEG was extended into polysomnography (PSG). As sleep pathology became better defined, the need to measure other physiologic function such as respiratory parameters, limb movements, gastric pH, and penile tumescence, among others, became clear. In the process, EEG received less and less attention.

Early studies of sleep typically devoted 2 channels to monitoring eye movement and 1 channel each for electromyography (EMG) and EEG. This was largely due to the limited amount of channels in the machines available at that time. Increasing numbers of channels were available in subsequent polygraphs, but these were utilized for measuring other physiologic parameters of interest as understanding of sleep pathology grew. Unfortunately, even in modern times EEG recording often remains confined to 2 channels, though 32 channels are often available in contemporary recording systems. We believe that this tendency to neglect cerebral electrophysiology in contemporary polygraphy is unfortunate. Various central nervous system and metabolic disorders may result in a syndrome mimicking excessive daytime somnolence. Unusual nocturnal spells may be seizures. EEG findings during PSG may be the first indication of these medical disorders. Furthermore, EEG findings associated with epilepsy may be confined solely to sleep. Thus, we believe a thorough sampling of electrocerebral activity with multiple channels covering both sides of the scalp should be performed during routine PSG, especially if seizures are suspected. Similarly, multiple EMG channels may provide a more accurate understanding of muscle tone or periodic movments. In summary, the broad availability of multiple polygraphic channels increases the information these studies can provide and so requires a more complete understanding of normal and abnormal EEG, EMG, and electro-oculographic (EOG) patterns.

A complete description of the technical and interpretive issues in EEG, EMG, and EOG is not possible in a single chapter. The reader is referred to several excellent monographs[2–6] for more detail. The discussion presented in this chapter starts with basic technical and safety issues. The polygraphic circuit and differences between analog and digital recording are discussed. Measurement and interpretation of EOG and EMG is then briefly reviewed. The bulk of the chapter is devoted to normal EEG findings in wakefulness and sleep as well as frequently encountered abnormalities. The discussion is limited to findings in humans ages 2 months and older. Several useful sources are available for the reader interested in neonatal wake and sleep EEG.[2,][3,][5,][7]

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ELEMENTARY CONCEPTS OF ELECTRICITY

It is useful to begin with a review of some elementary concepts of electricity. Electricity is largely the study of the concentration and flow of charged particles. A fundamental principle of electricity states that like charges repel and unlike charges attract. Thus, if particles of like charge are allowed to move freely, they will quickly reach a relatively uniform distribution. The flow of charged particles is called *current* (I). Various features of biological systems, such as equilibrium constants of reactions or membrane permeability, often result in a concentration of particles of like charge. This concentration is a store of potential energy that is released when the charged particles are allowed to move and achieve a more uniform distribution. *Voltage* (V) measures how much energy is released when a set amount of charge is allowed to move as current flow. Voltage, also known as *potential difference,* is always measured between two points. Because the concentration of charge may differ at any two points, the potential energy contained in a given concentration of charge can only be measured by relating it to the concentration of charge elsewhere. Charges experience *resistance* (R) as they move through a conducting medium. The resistance is measured in *ohms*. Current, voltage, and resistance are related by *Ohm's law,* which states that I = V/R. The law makes intuitive sense: The higher the voltage (V) between two points, the more “pressure” there is for the charges to move, and so one would expect flow of charge (I) to be higher. Conversely, if there is more resistance (R) to the movement of charge, one would expect the flow of charge (I) to be less.

Concentrations of charges of different polarity are often separated by a poorly conducting medium. This situation can be modeled by an electrical device known as a *capacitor*. Capacitors can be thought of as two conducting plates separated by insulation. The ability of the capacitor to store charge is measured by the capacitance, which equals the amount of charge the device can store for a given voltage. When a capacitor is connected to a source of constant voltage such as a battery, positive charges will flow from the positive pole of the battery to one plate and negative charges will flow from the negative pole of the battery to the other plate. Charges will continue flowing until the mutual repulsion of the accumulated charges on each plate equals the potential difference of the battery. At this point, current flow will cease.

The situation is different when the source of voltage varies with a predictable frequency. Voltages generated by the brain and recorded by the EEG do not stay constant but vary continuously within certain limits. In a circuit with such a voltage source and a capacitor, it can be shown that the current flow at any time equals the capacitance multiplied by the change in voltage with respect to time.* Thus, the capacitor will influence and resist the flow of current in a circuit with a varying voltage. The resistance to current flow exerted by the capacitor is measured by the *capacitative reactance* (X_C). It is clear that the concept of resistance must be expanded to include capacitative reactance in circuits with a voltage source that varies. *Impedance* (Z) is a measure of resistance that includes reactive capacitance and is therefore appropriate in circuits with varying voltages. In these situations, Ohm's law takes the form I = V/Z. Because cerebral voltages vary with time, impedance is the proper measure of how well electrodes and gel transmit brain activity.

* Capacitance (C) is defined as the amount of charge (Q) the capacitor can store for a given voltage (V), or Q/V. Presuming that C is constant, and differentiating with respect to time (t), we find that dC/dt = 0 = -[(Q/V2) (dV/dt)] + [(1/V) (dQ/dt)]. Rearranging, dQ/dt = (Q/V) (dV/dt). Current flow (I) = dQ/dt. Hence, I = C(dV/dt).

PHYSIOLOGIC BASIS OF ELECTROENCEPHALOGRAPHY

An EEG record is essentially a measure of the changes of electrocerebral voltages over a period of time. To interpret an EEG, it is important to understand the source of the voltages recorded at the scalp and how these voltages are organized into normal cerebral rhythms.

Electrocerebral activity measured by EEG does not appear to be caused by individual or summed action potentials. Action potentials are too short (usually <1 msec), and synchronized bursts of action potentials have too limited a distribution, to account for the rhythms seen in a normal EEG. Excitatory and inhibitory postsynaptic potentials, in contrast, last much longer (15–200 msec or more). These synaptic potentials induce more extensive voltage changes in extracellular space. Scalp-recorded EEG activity results from extracellular current flow induced by summated excitatory or inhibitory postsynaptic potentials.

Figure 12-1 illustrates, in a simplified fashion, how synaptic potentials induce voltage changes recorded at the scalp. An excitatory input on a deep dendrite causes positive ions to flow into the pyramidal neuron, resulting in a lack of positive charges, or negativity outside the neuron. Everywhere else, including at the superficial dendrite, positive ions flow out of the cell into the extracellular space to complete the current loop. This results in a relative positivity in the superficial extracellular space. Because the superficial dendrite and surrounding extracellular space are closer to the scalp electrode, a positive deflection is recorded. The separation of superficial positive and deep negative charges allows one to view the pyramidal neuron as a dipole. This permits a more complete analysis of how synaptic potentials result in scalp EEG changes.[8–10]

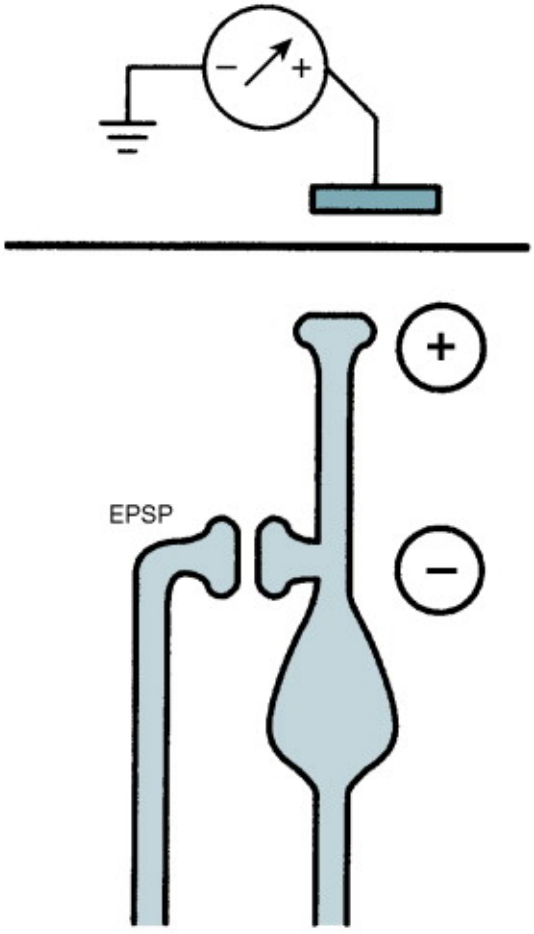

FIGURE 12-1 Scalp EEG voltage recordings resulting from an excitatory input on a deep synapse. (EPSP, excitatory postsynaptic potential.)
(Modified from Kandel ER, Schwartz JH [eds]. Principles of Neural Science, 4th ed. Amsterdam: Elsevier, 2000.)

EEG voltage recordings are rhythmic (i.e., they are regularly recurring waveforms of similar shape and duration). It is important to understand how voltage changes induced by individual neurons are organized into the widely distributed

rhythms recorded with EEG. The dominant theory of EEG rhythmicity was advanced by Andersen and Andersson[11] and is based on studies of barbiturate-induced spindle activity. These investigators recorded synchronous rhythmic spindles from the cerebral cortex and thalamus. Neither removal of the cerebral cortex nor transection of the brain stem below the thalamus eliminated thalamic spindles. Ablation of the entire thalamus, however, abolished spindle activity. These findings led to the proposal that rhythmic oscillations of thalamic neurons induced synchronous synaptic excitatory or inhibitory potentials over broad areas of the cortex, and thus the rhythmic voltage changes recorded with scalp EEG. Diffuse thalamocortical neuronal projections were known to exist and could mediate this thalamic influence. This model was expanded to explain most EEG rhythmic activity.

More recent work has emphasized the fact that barbiturate-induced spindles differ significantly from other cerebral rhythms.[12] The role of the thalamus in synchronizing barbiturate spindle activity over broad areas of cortex may not be relevant to other EEG rhythms. Neurons in other brain structures, including the inferior olive, hippocampus, and temporal neocortex, exhibit oscillatory behavior and may play a role in generating EEG rhythms.[13] Although widespread subcortical influences probably play an important role in organizing EEG rhythms, it is premature to conclude that all EEG rhythms are induced by oscillations of thalamic neurons.

Cerebral activity recorded at the scalp has approximately one-tenth the voltage of activity simultaneously recorded at the cortical surface. This attenuation is largely due to the cerebrospinal fluid, dura, and skull overlying the cortical surface. The area and location of the cortex generating the activity also play a role.

COMPONENTS OF THE POLYGRAPHIC CIRCUIT

Voltages and current flows generated by the cortex, eyes, and heart during PSG studies are exceedingly small. The function of the polygraph is to transform these tiny voltages into an interpretable record. The major components necessary to accomplish this are illustrated in Figure 12-2. Two types of polygraphic machines are in use: analog polygraphs based on solid-state circuitry and digital polygraphs based on digital circuits, computers, and, increasingly, network technology (see Fig. 12-2). As one may suspect, analog polygraphs are becoming replaced by more contemporary digital machines. However, study of signal flow through analog systems remains important because it provides a fundamental understanding of how brain signals need to be modified so that clinical interpretation is possible. To start with, we discuss electrodes and the scalp-electrode interface. We then discuss the components of the analog polygraph. Finally we discuss how the functions of the polygraphic circuit are carried out by the digital polygraph. The discussion is based on idealized systems rather than polygraphs provided by any specific vendor.

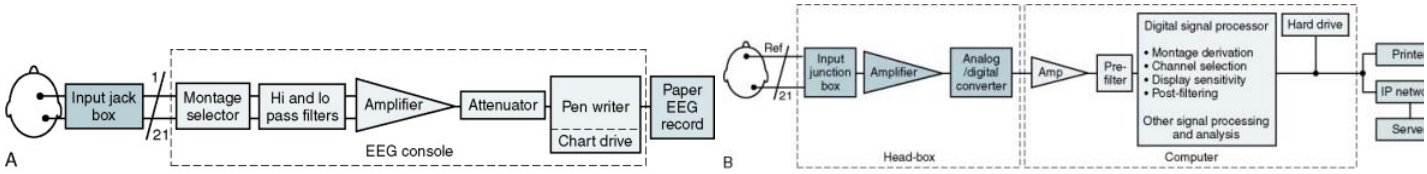

FIGURE 12-2 Computer-generated components of an analog **(A)** and a digital **(B)** polygraph.
(Courtesy of Dr. Sidney Diamond.)

Electrodes and the Scalp-Electrode Interface

Electrodes and conducting gel transmit biological voltages from skin or muscle to the polygraphic circuit. Various types of electrodes have been designed.[4,][14] Disk electrodes are preferred for recording EEG, EOG, and electrocardiography, and may be used to record EMG as well. These are typically made of chlorided silver or noble metals such as gold or platinum.

The critical component of the conducting gel is an electrolyte, usually sodium chloride, that easily dissociates into its ionic components. The anions and cations establish a layer of positive and negative charges between the scalp and recording electrode. This charged double layer allows transmission of scalp voltage changes to the electrode and the rest of the polygraphic circuit.

The electrode-electrolyte interface is the most critical link in the polygraphic circuit. Most artifact originates here; consequently, careful technique in electrode application largely determines the quality of the recording. The impedance in any electrode pair should not exceed 10 kohm. High impedance can decrease the amount of signal the electrode presents to the amplifier. Methods to achieve low impedance are described in Chapter 11. In addition, the impedance in the two electrode inputs into the amplifier should not differ by more than 10 kohm. Higher values will degrade the ability of a differential amplifier to eliminate environmental noise and will increase artifact (see *Artifacts* later). Impedance varies with composition and surface area of the electrode as well as with the surface area of the conducting gel beneath the electrode. Thus, these factors should be held constant in an electrode pair attached to the same amplifier. For example, a disk electrode and needle electrode have different surface areas, and conducting gel is not used with needle electrodes. Therefore, impedances of the two electrodes will be significantly different. If the two electrodes are attached to the same amplifier, environmental artifact is likely to contaminate the recording.

The Analog Polygraphic Circuit

Electrodes are attached to electrode wires, which conduct the EEG signal to the electrode box or jackbox. The electrode wires terminate in a pin that is plugged into a receptacle in the electrode box known as a *jack*. The jacks are usually numbered or identified according to the International 10-20 System. Wires from each of the jacks run together in a shielded conductor cable to the polygraph. Here, wires from each of the jacks are connected to a specific point on a multiple contact switch known as the electrode selector. The selector contains rows of switches, arranged in pairs corresponding to the two inputs of an amplifier. Depressing the switches allows the technician to select which two electrodes will contribute signal to each amplifier.

The amplifiers used in both analog and digital polygraphic recording have several important features. *Differential* amplifiers are usually used. These amplify the *difference* in voltage between the two amplifier inputs. Figure 12-3 provides an illustration. Let us assume that T3 is connected to input 1 of an amplifier and C3 is connected to input 2 of the same amplifier. The amplifier would determine the difference between the two inputs (5 μV) and the galvanometer pen would register a deflection of 5 units. The actual amount of the deflection in millimeters would depend on the sensitivity used (see next paragraph). The fact that the differential amplifier amplifies the difference between electrode inputs rather than the absolute voltage at any electrode is a useful feature, because environmental noise, which is likely to be the same at the two electrodes, is "subtracted out" and therefore does not contaminate the recording. The *common mode rejection ratio* measures the ability of the amplifier to suppress a signal, such as noise, that is present simultaneously at both electrodes. This ratio should exceed 1000:1; most amplifiers currently in service have values that exceed 10,000:1.

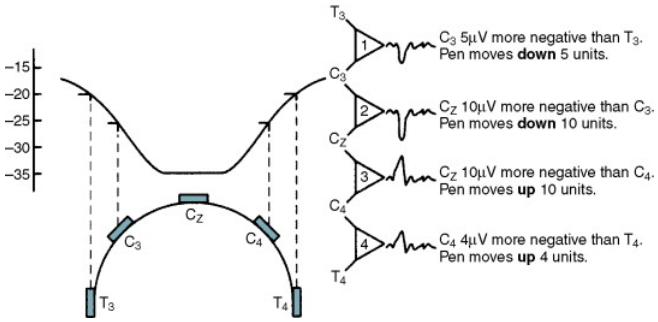

FIGURE 12-3 Scalp voltage distribution of a vertex wave. A plot of hypothetical absolute voltages at various electrode positions is shown on the left, and resulting EEG tracing with explanation is shown on the right. A transverse bipolar chain is used with amplifiers connected from left to right. The same electrode is connected to input 2 of an amplifier and input 1 of the next amplifier in the chain.

A differential amplifier multiplies the small difference in cerebral voltages by a constant, referred to as *gain*. This multiplication is necessary because the recording galvanometer pen requires voltages much higher than those generated by the brain to generate an EEG record. Analog-to-digital converters also require higher voltages to perform digitization. Amplifiers can faithfully amplify input voltages only within a certain range known as the *dynamic range*. Input voltages below the lower limit of the dynamic range are lost in noise; voltages above the upper limit result in a distorted EEG output. Flexible control of amplification within the dynamic range is achieved by manipulating the sensitivity switch. The sensitivity switch is connected to a series of voltage dividers that attenuate the amplified cerebral voltages sufficiently for the EEG record to be interpretable. *Sensitivity* is defined as the amount of voltage necessary to produce a set deflection of the pen. The usual units are microvolts per millimeter or millivolts per centimeter. One of the technologist's most important tasks during analog recording is to maintain sensitivity settings low enough for the input voltage to result in a pen deflection of sufficient amplitude to be detectable. However, the sensitivity cannot be so low that the amplitude of pen deflection interferes with or "blocks" pen movements in adjacent channels. Because the voltage of electrocerebral activity varies during the study, sensitivity settings may need to be adjusted to maintain an appropriate amplitude of recorded EEG activity. Because amplitude of various waveforms is an important consideration in scoring sleep stages, these adjustments must be carefully documented.

Whereas the sensitivity settings determine the *amplitude* of pen deflection, the polarity of the cerebral activity determines the *direction* of the pen deflection. The differential amplifier compares polarity at the two electrodes. The resulting pen deflection is determined according to the *polarity convention*. The pen moves up if input 1 is negative relative to input 2, or if input 2 is positive relative to input 1. The pen moves down if input 1 is positive relative to input 2, or if input 2 is negative relative to input 1. It follows that *phase reversals* of EEG waveforms can be used to roughly localize the scalp distribution of those waveforms. The scalp voltage distribution of a typical vertex wave is illustrated in Figure 12-3, where hypothetical absolute voltages at several electrode positions are shown. The electrodes are linked in serial pairs from left to right. When C3 is connected to input 1 and Cz is connected to input 2 in amplifier 2, the amplifier determines that the difference between the two electrodes is 10 μV. Input 2 is more negative than input 1. Thus, the galvanometer pen recording from this amplifier registers a downward deflection of 10 units. In amplifier 3, Cz is connected to input 1 and C4 to input 2. The amplifier determines that the difference in voltages is also 10 μV. However, input 1 is now more negative than input 2. Consequently, the galvanometer pen recording from this amplifier registers an upward deflection of 10 units. The phase reversal in the adjacent channels marks the electrode where the vertex wave is most negative. This is the electrode shared by both amplifiers, Cz. Because most cerebral activity is negative at the scalp, phase reversals with the pen deflections pointing toward each other (see Fig. 12-3) are encountered most commonly. Positive cerebral activity would result in a phase reversal with the pen deflections pointing away from each other at the electrode that was most positive. Note that localization by phase reversal is accurate only when electrodes spaced at relatively short distances are serially linked in adjacent amplifiers. This is known as a *bipolar montage*.

After voltages at the two inputs are subtracted and amplified, the result is passed through a series of filters. The goal of filtering is to attenuate voltages occurring at undesirable frequencies (e.g., environmental noise) without disturbing frequencies found in the biological signal of interest. A frequency-response curve measures the ability of a filter to attenuate various frequencies. Frequency-response curves for two types of analog filter included in many polygraphs are presented in Figures 12-4 and 12-5. A *high-pass filter* (also known as a *low-frequency filter*) allows higher frequency activity to pass unchanged while progressively attenuating lower frequencies (see Fig. 12-4). A *low-pass filter* (also known as a *high-frequency filter*) allows lower frequencies to pass unchanged while progressively attenuating higher frequencies (see Fig. 12-5). Analog filters are defined by *cutoff frequency* and *roll-off*. The filter with a given cutoff frequency will attenuate voltage of that frequency by 20%* (e.g., the high-pass filter in Figure 12-4 has a cutoff frequency of 1 Hz, so a 100-μV, 1-Hz wave passed through this filter will have an amplitude of 80 μV). Attenuation of frequencies above the

cutoff frequency is more or less linear for the high-pass filter. Attenuation of frequencies below the cutoff frequency is progressively more severe as lower frequencies are encountered. This progressively more severe attenuation is defined by the filter's roll-off. Roll-off for most analog EEG filters is -6 dB per octave. For the high-pass filter, this means that the voltage of activity is decreased by half for every halving of the frequency.

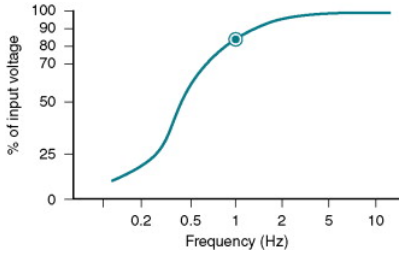

FIGURE 12-4 Frequency-response curve of a hypothetical high-pass filter with a cutoff frequency of 1 Hz and a roll-off of -6 dB per octave.
(Modified with permission from Tyner F, Kott J, Mayer W Jr. Fundamentals of EEG Technology, Vol 1. New York: Raven, 1983.)

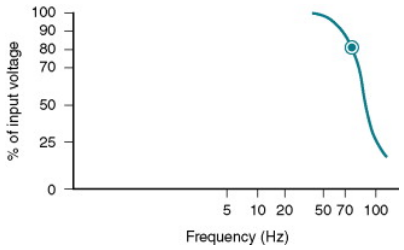

FIGURE 12-5 Frequency-response curve of a hypothetical low-pass filter with a cutoff frequency of 70 Hz and a roll-off of -6 dB per octave.
(Modified with permission from Tyner F, Kott J, Mayer W Jr. Fundamentals of EEG Technology, Vol 1. New York: Raven, 1983.)

The *60-Hz* or *notch* filter is also present in most polygraphic amplifiers.[†] This filter is designed to attenuate mains frequency very harshly while attenuating activity of surrounding frequencies less extensively.[4] Because electrical mains are ubiquitous, 60-Hz artifact may easily contaminate an EEG recording. The notch filter should be used sparingly for at least two reasons. First, some biological signals of interest to the polysomnographer have waveforms with important components in the range of 40–80 Hz. Examples include myogenic activity and epileptiform spikes, both of which may be significantly attenuated by the notch filter. For example, use of the notch filter in the chin EMG channel may result in a false impression that tonic EMG has significantly decreased. In addition, the capability of the differential amplifier to reject common signals (see earlier) should be sufficient to suppress 60-Hz artifact in most cases. Thus, the appearance of 60 Hz usually signals a problem somewhere in the polygraphic circuit that needs to be resolved. Most often the culprit is high impedance at the electrode-scalp interface. Less frequently, defects in the amplifier or grounding of the polygraph are responsible. In these cases, addressing the cause of the 60 Hz rather than using the notch filter is the appropriate course. There are circumstances in which a nearby source of 60 Hz (e.g., a critical piece of medical equipment that cannot be disconnected) renders the EEG uninterpretable. Use of the 60-Hz filter may be justified in these circumstances but must be clearly documented.

A writer unit transforms the amplified and filtered signal into a written record. The writer unit consists of an oscillograph and chart drive. A *galvanometer pen unit* is a widely used oscillograph (Fig. 12-6). A specially designed coil of wire and a pen stylus are mounted on a rod. The coil of wire is placed between the two poles of a permanent magnet. Current flow from the amplifier enters the coil and induces a magnetic field. The induced magnetic field interacts with the field of the permanent magnet, resulting in a deflection of the pen stylus on the paper. The amount of deflection is proportional to the magnetic field, which is proportional to the current from the amplifier, which in turn is proportional to the biological signal. A spring attached to the rod returns the pen stylus back to baseline after the current responsible for the deflection has ceased. This spring, together with the friction of the pen stylus against the paper and the inertia of the galvanometer, are collectively known as *damping* and resist the pen movement. Very rapid signal changes (high-frequency signal) require very rapid galvanometer movement, increasing disproportionately the amount of energy necessary to overcome damping. Because more energy is required to write out high-frequency signals, the galvanometer pen unit, in effect, acts as a high-frequency filter. Galvanometer pen writer units usually do not faithfully reproduce signals with frequencies higher than 80–90 Hz.

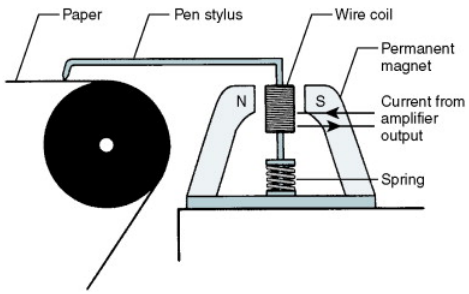

FIGURE 12-6 Hypothetical galvanometer pen writer unit (not to scale).
(Modified with permission from Tyner F, Kott J, Mayer W Jr. Fundamentals of EEG Technology, Vol 1. New York: Raven, 1983.)

The *chart drive* pulls the paper below the pens at a constant speed to provide a continuous record of pen deflections (voltage changes) over time. Paper speeds slower than 10 mm/sec save paper but cannot be recommended because resolution of faster waveforms necessary for scoring sleep stages is impossible. When suspicious waveforms such as epileptiform spikes are noted, increasing paper speed to 30 mm/sec can aid interpretation.

Components of the Digital Polygraphic Circuit

Modern digital communications technology has allowed miniaturization of what had been much bulkier (if somewhat hardier) solid state components. It has also resulted in some reorganization of the functions of the polygraphic circuit. Digital polygraphs perform all of the functions of the analog polygraphic circuit. However, the ability to digitize the EEG signals allows easier manipulation, transmission, display, and storage, conferring some distinct advantages.

Many contemporary systems have folded jackbox inputs, amplification, and sampling/analog-to-digital conversion into one box, approximately the size of the jackbox in analog systems (see Fig. 12-2B). In addition to inputs for the standard 10-20 scalp electrode postions, electrode inputs for digital systems always include one or more additional inputs for reference electrodes. A location between Fpz and Fz is often chosen for the reference electrode, but any location on the scalp that is relatively noise free and where firm attachment to the scalp is possible can be used. Reference electrodes are necessary because digital EEG recording is always referential. In other words, signal recorded from any given electrode is presented to input 1 of the differential amplifier assigned to that electrode, while signal recorded from the reference electrode is always presented to input 2. Referential recording allows easy re-montaging later in the polygraphic circuit. This contrasts with analog EEG machines, in which signal from each electrode is presented to the montage selector switch; in the latter situation, the technician decides on the montage at the time of recording and changing montages later is not possible. However, referential recording can cause significant confusion if the reference electrode becomes disconnected.

The signal (still an analog signal) is next presented to an amplifier assigned to that particular input. The principles of differential amplifiers and common mode rejection described previously also pertain to digital machines. Amplifiers are smaller and typically are part of the box containing the inputs.*

Amplified inputs are next presented to the *analog-to-digital converter* (ADC). The ADC is the heart of the digital EEG machine. Conceptually, the ADC has several parts: a clock, a voltmeter, and memory. Simply put, the ADC assesses (samples) the voltage (amplitude) of the continuous analog EEG, creates a numeric value corresponding to this voltage, and stores this value in memory. The ADC then repeats this process at a uniform interval (intersample interval). In this manner, a continuous (analog) EEG signal is converted into a series of numeric values representing the voltage of the signal at serial moments in time. Thus the signal is commonly referred to as “digitized” or a “digital signal.”

For the digitized EEG signal to be clinically useful, it has to be a faithful representation of the “real” analog signal. Two features of the ADC determine how accurately digitized signal reflects the original analog waveform: (1) the sampling rate of the ADC (“horizontal resolution”), and (2) the number of bits in the ADC (“vertical resolution”). The number of times the EEG amplitude is sampled in a period of time is called the *sampling rate*. Intuitively, the more often the ADC samples an analog waveform, the more accurately the digitized output reflects the analog waveform. For example, a

sampling rate of 256 per second means that the voltage of the analog waveform is sampled (assigned a numerical value) every 1/256 of a second (the intersample interval). This intersample interval indicates the horizontal (time) resolution and is a typical value for contemporary polygraphs. If the digitized signal is to reflect the analog signal faithfully, the sampling rate must be at least two times the highest frequency in the analog signal. Frequencies in the analog signal exceeding half the sampling rate will appear in the digitized signal at a lower frequency than in the analog signal. This is known as *aliasing* because the faster frequencies in the analog signal appear under the "alias" of a lower frequency in the digitized signal (see Ebersole and Pedley,[2] page 47, for further discussion).

The number of discrete numeric levels that the voltage of the analog signal can potentially be assigned indicates the vertical resolution. Intuitively, the more numeric levels the ADC has available to assign a voltage reading of an analog waveform at any point in time, the more accurately the digitized signal represents the "real" analog waveform. Vertical resolution of the analog-to-digital converter is measured in "bits" or powers of 2. For example, a 10-bit converter can assign 2^{10}, or 1024, levels to the voltage of a signal at any point in time. Vertical resolution of the converter should be higher than the noise level of the amplifier so that all signals exceeding noise level can be represented. Because noise level of contemporary amplifiers is approximately 1 μV, a 10-bit converter is usually adequate for EEG signals. A 10-bit converter would allow amplitude at any point in an analog signal to be assigned a value in 1-μV steps from -511 to +512 μV ($2^{10}/2$ - 1 level assigned to 0 μV). This range encompasses the vast majority of EEG voltages recorded from the scalp. This is usually adequate for EMG and EOG signals as well. Contemporary digital polygraphic systems typically utilize ADC with vertical resolution ranging from 16 to 22 bits.

The digitized signal is now passed to a computer for storage in memory and further manipulation by a software program. Sofware programs perform many of the functions of the solid state components of the analog polygraph (see Fig. 12-2B).

Montage selection is performed by software manipulation of the digitized signal. Because the analog EEG signal is recorded referentially (see earlier), montage selection can occur by simple subtraction of the digitized outputs of amplifiers corresponding to the individual electrodes. For example, a patient undergoing a sleep study may have a full complement of electrodes attached. The amplifiers in the digital headbox amplify the signal from each of these relative to the reference electrode (Fp1-R, T3-R, O1-R, C3-R, A2-R, etc.), and this signal is digitized and recorded in computer memory. The interpreter desires to display C3-A2 and sends the appropriate command to the software. The software subtracts C3-R from A2-R and the screen displays C3-A2. This recording displays unusual activity, and there is concern that this may represent a seizure. The interpreter requests the software to display Fp1-T3, T3-O1, Fp1-C3, and C3-O1. Digitized referential output from these amplifiers was being recorded to the computer hard drive throughout the study but was not being displayed. The interpreter can now use this information to address concerns that the unusual EEG activity was an epileptic seizure. In a similar manner, a variety of montages can be accessed by the interpreter to address other concerns provided that a broad range of information is recorded referentially throughout the study.

Filtering is another function performed by software on the digitized EEG signal. Digital filters are computer algorithms that transform a digitized EEG by filtering out designated frequencies. Digital filtering can be performed in the frequency domain by computing the Fourier transform of a segment of an EEG, replacing coefficients at the frequency one wishes to eliminate by zero, and then reconstituting the EEG by computing the inverse Fourier transform.[15] (See further discussion of Fourier analysis later.) Digital filtering can also be performed in the time domain by using a moving average method.[15] Such finite impulse response[12] filters are increasingly used in digital EEG machines and allow filtering without phase distortion, an advantage over traditional analog filters.

Software can manipulate and analyze digitized EEG signal in more sophisticated ways as well. Spectral analysis is a commonly employed technique relying on the Fourier theorem and forms the basis of sleep stage scoring software. Pitfalls and limitations of this technique are discussed later. Algorithms designed to detect seizures and epileptiform abnormalities are also commercially available.[16] The clinical utility of these is variable, and they do not replace a thorough analysis of the original record by a qualified interpreter.

Modified and organized digital signal can be directed from computer memory to a variety of destinations. Polygraphic data must be presented to the interpreter for visual inspection. Polygraphic data in digital systems is typically displayed on monitors. Monitor resolution must be sufficient so that the degree of resolution provided by the ADC is not significantly compromised. For example, if 1024 pixels (a common horizontal resolution in "off-the-shelf" computer monitors) are available to display 30 seconds of EEG, at most 1024/30 or 34 pixels can be devoted to display 1 second of EEG. This is far less than the 256 samples/sec horizontal resolution provided by ADCs typically used in contemporary polygraphic systems. While this degree of horizontal resolution may be adequate for sleep stage scoring, it is not sufficient for analysis of epileptiform activity or electrographic seizures. Changing the time base of the display so that 10-second epochs are displayed on the monitor will triple the horizontal resolution of the monitor in the previous example and bring it more into line with the resolution provided by the ADC. Similar considerations pertain to the vertical resolution of the monitor, though this is of less concern in polygraphy, in which fewer channels of polygraphic recording are typically presented at any time. In general, larger monitors with higher monitor resolution better reflect all the information present in the digitized signal.

Digitized polygraphic data can be transmitted to a printer to generate a "hard copy" polygraphic tracing of selected epochs. Printing an entire polygraph is rarely necessary with digital systems. Digitized data in computer memory must ultimately be transmitted to peripheral devices for storage. A variety of digital storage media are available, all of which are less expensive and more convenient than the paper and microfilm required for analog EEG. After security and privacy issues are addressed, digitized polygraphic data can be transmitted via network or the Internet to other computers in the clinic, the interpreter's home, or another continent.

Advantages and Limitations of Digital Recording Systems

The advantages of digital recording are leading to the obsolescence of analog polygraphic systems. Advantages include decreased size and weight, the ability to record vast amounts of data cheaply and efficiently, and the ability to re-montage digitized data post hoc as needed. Digitized polysomnograms can be analyzed, transmitted, and stored more cheaply, quickly, and efficiently than analog studies. For these reasons, digital recording will continue to replace analog polygraphic systems over time.

The reliance of digital polygraphic systems on referential recording (see earlier) leads to a vulnerability that is important to remember. If the reference electrode becomes detached, the EEG signal from the recording electrode may be overwhelmed by the environmental noise from the open reference input. When inputs from individual channels are subtracted to display a montage, the record may appear identical to a low-voltage EEG. An unsuspecting technician may think nothing is amiss when in fact no EEG signal at all is being transmitted to the digital headbox and potentially clinically important activity is being missed. Checking impedance of the reference electrode and displaying all electrodes to reference at the beginning and end of the study will address this weakness. In general, it is important to remember that the analog electrophysiologic signal is closer to the clinical phenomena that interest the practitioner of sleep medicine than the digital signal interpreted on the monitor. Understanding how the original analog signal is transformed is therefore important.

* In electrophysiology, a widely used convention dictates that voltage at the cutoff frequency is attenuated by 20%. In electrical engineering, voltage at the cutoff frequency is attenuated by approximately 30%.

† Because mains frequency is 50 Hz, a 50-Hz notch filter is widely available.

* Amplifiers in contemporary systems (and in most analog systems) perform amplification in multiple stages. Usually the last (and most significant) stage incorporates analog filters. These attenuate frequencies that can potentially damage the analog-to-digital converter but that are not physiologically relevant. Analog filters at this stage are "wide open"; that is, they do not attenuate clinically relevant activity. They cannot be modified by technicians or interpreters. Filtering of clinically relevant material is performed later in the polygraphic circuit (see later).

SPECTRAL ANALYSIS

Spectral analysis is probably the most widely used computerized analysis of a digitized EEG.[15,][17,][18] Spectral analysis is based on the Fourier theorem, which states that any waveform can be decomposed into a sum of sine waves at different frequencies with different amplitudes and different phase relationships. When summed, these waves reconstitute the original waveform. The Fourier transformation is a mathematical operation that provides the frequency, amplitude, and phase parameters of each of these component sine waves. Fourier coefficients represent the amplitude and phase relationship at each of the component sine wave frequencies. Squaring and summing the Fourier coefficients at each frequency provides the power at that frequency. A plot of power at each of the component frequencies is called the *power spectrum*. The power spectrum allows determination of relative amounts of given frequencies in the waveform over the time segment analyzed.

The fast Fourier transform algorithm[19] allows real-time spectral analysis with contemporary personal computers. Commercially available software packages offer straightforward presentation of the power contained in the traditional frequency bands during a set period of EEG. This allows detection and quantification of frequencies not detected with visual inspection. However, there are many potential pitfalls.[17,][18] Theoretically, the power spectrum is a faithful representation of the original signal only if the original signal is stationary (has stable statistical properties). The EEG signal is clearly not stationary over long periods, although it appears reasonably stationary over brief epochs.[20] In practice this means that the EEG segment selected for analysis should not include obvious changes such as those due to alerting or drowsiness. In addition, the Fourier theorem assumes that an infinitely long sample is available for analysis. Because even long samples are clinically impractical, tapering or “windowing” of the end points of the sample is necessary to attenuate the spurious frequencies (leakage) arising from the segmentation of the signal. Windowing is never completely successful—some leakage is unavoidable. This may affect clinical interpretation when power is displayed in the traditional frequency bands; for example, a reasonable amount of alpha power may leak to the theta or beta bands. Nonsinusoidal rhythms such as “spiky alpha” are common in routine EEG. Fourier analysis of a nonsinusoidal rhythm of a set frequency often shows a large peak at that frequency with smaller peaks at harmonics of the frequency. These smaller, higher frequency peaks may lead the interpreter to conclude that cerebral activity at the higher frequency is actually present. The most common pitfall in interpreting power spectra is artifact. Artifact is ubiquitous, often subtle, and can take an almost infinite variety of forms. The computer cannot separate artifact from EEG and includes artifact in the computation of the power spectrum. This can lead to significant misinterpretation. Artifact is much more difficult to recognize in the power spectrum than in the unprocessed EEG. It is therefore very important to review an EEG before spectral analysis or interpretation of the power spectrum to prevent analysis of segments contaminated by artifact.

Despite these limitations, spectral analysis can play a useful role in the operating room and in routine scoring of sleep studies (see Chapter 18). A basic understanding of the principles of signal processing and thorough experience in the appearance of various cerebral activities after spectral analysis is necessary. Unprocessed “real” physiologic signal must always be reviewed. Spectral analysis has not demonstrated any consistent clinical utility in routine EEG despite almost 2 decades of active research. Because the potential for misinterpretation and abuse is high, the major neurologic and neurophysiologic professional organizations have taken strong positions against the use of spectral analysis during routine EEG.[21,][22]

Expert CONSULT

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

ELECTRICAL SAFETY

Contemporary polygraphic and EEG studies are very safe procedures. Nevertheless, the possibility of electrical injury exists whenever a patient is connected to an electrical apparatus. Thus, technicians performing studies and physicians supervising sleep laboratories must understand the basic principles of electrical safety.

Electrical injury is caused by excessive current flow through biological tissue. Such electrical injury includes burns, seizures, and irreversible damage to nervous tissue. When excessive current flows through the heart, potentially fatal arrhythmias may occur. The amount of current necessary to induce ventricular fibrillation is dependent on skin impedance, the mass of tissue the current must traverse before reaching the heart, the health of the heart, and the general health of the patient, among other factors. In a healthy adult with dry intact skin, 100–300 mA delivered at 60 Hz will induce fibrillation (macroshock).[4] Smaller amounts of current (microshock) will induce fibrillation in electrically susceptible patients. These include patients with wet skin or wounds, as well as patients with pacemaker electrodes inserted in the ventricular myocardium. Dry intact skin offers high impedance to current flow, as technicians well appreciate. Increased skin moisture or skin interruption significantly reduces this impedance, allowing current to flow toward the heart more readily. Pacemaker wires allow the current to flow directly into the vulnerable myocardium rather than through the high impedance offered by the chest wall and pleural cavities. When a 60-Hz current is applied directly to the heart, intensities as low as 100 μA can result in fibrillation, although higher values are necessary in most cases.[23–25]

Current flow requires a source of current and the formation of a complete circuit. Thus, electrical safety has two goals: (1) the polygraph must not become a source of excessive current, and (2) the polygraph and patient (or technician) must not form a complete circuit through which excessive current may flow and cause electrical injury. Proper maintenance, proper grounding, and use of isolation devices accomplish these goals.

The power unit of the polygraph is a potential source of excessive current. A fault in the power unit may result in a short circuit that would allow a *fault current* to flow to the polygraph chassis (Fig. 12-7A). If the machine ground were disabled and the patient were touching a pipe or some other conducting substance, current would flow through the electrodes and the patient to the pipe, possibly causing electrical injury. Current would also flow through a technician touching the polygraph and a conducting substance. To guard against this possibility, the chassis of the polygraph is connected to the building ground through a three-pronged outlet (Fig. 12-7B). Should a current-bearing element contact the chassis, the current would be shunted through machine ground to the building ground because this path has the least resistance. The sudden high-current flow would blow a fuse in the power unit or open a circuit breaker, stopping further current flow. A brief period is necessary for the excessive current to blow the fuse and, if the patient is touching a conducting substance during this period, current will still flow through the patient. The duration of the current flow would be briefer, however, and the danger to the patient decreased.

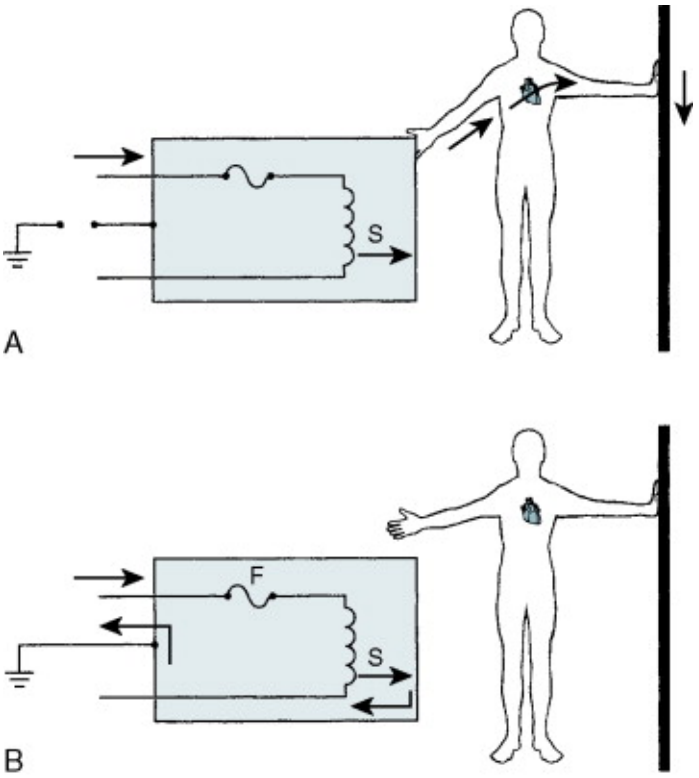

FIGURE 12-7 **(A)** Technician touches polygraph chassis and water pipe. Polygraph chassis is ungrounded because of interruption in ground wire. A shor circuit (S) in the power supply unit allows current to flow to the chassis, through the technician's heart, and to the water pipe. *Arrows* trace path of current flow. **(B)** A short circuit in the polygraph with an intact chassis ground. A short circuit (S) in the power supply allows current to flow to the chassis. The low-resistance chassis ground allows unimpeded flow of current to the building ground. The current surge blows a fuse (F), quickly stopping further current flow. Bystanders are safe unless they touch the chassis at the moment of the short circuit.

It follows that the connection between the polygraph and building ground must not be compromised. Electrical outlets powering appliances connected to patients must have documented secure connection to building ground. Technicians should ensure good contact between the ground pin of the power cord and the outlet. Three- and two-pronged adapters do not provide secure contact with building ground and must not be used. Resistance of the ground circuit in the polygraph should be checked periodically to detect interruptions. Fuses should never be defeated (i.e., short-circuited). Repeatedly blown fuses may indicate fault currents and potential danger to the technician and patient. Finally, regular maintenance may prevent potentially dangerous fault currents.

Even in the absence of a fault, the complicated circuitry of the polygraph generates lower intensity currents known as *leakage currents. Stray capacitance* is a major source of leakage current. Any circuit with current flow that is insulated from other conducting substances can be viewed as a capacitor. The current-carrying circuit can be considered one plate of a capacitor, the insulation and surrounding space can be considered the dielectric, and the other conducting substances can be considered the other plate of the capacitor. Alternating current flowing through any insulated circuit will therefore generate currents in other conducting substances in the area. One pertinent example is the power cord of the polygraph. Alternating current flow in the insulated “hot” wire of the power cord will induce a lower amount of current flow in the neutral and ground wires. Although leakage currents are much smaller than fault currents, they can cause injury in electrically susceptible patients. Acceptable limits for leakage currents have been defined.[4,][26] Adequate grounding protects both patient and technician in this circumstance. The leakage current is shunted to the low-resistance machine ground and then to the building ground. Extension cords increase stray capacitance, and thus leakage currents and should never be used during PSG. Isolation jackboxes that limit the amount of possible current flow through electrodes, and therefore prevent currents generated from the machine from reaching the patient, are widely used. These offer additional protection in electrically susceptible patients and should be employed wherever possible.

Current flow can also occur when machinery attached to the patient draws power from different outlets or when multiple grounds are attached to the patient. The voltage of the ground contact at different outlets may be quite different, resulting in current flow. Multiple grounds can result in a *ground loop,* which can act as a secondary coil of a transformer and generate current flow. A ground loop also acts as an antenna that will pick up ubiquitous environmental electromagnetic radiation and will increase artifact. These potentially dangerous situations can be avoided by plugging in all machinery attached to the patient to the same outlet cluster and using only one patient ground.

ELECTRO-OCULOGRAPHY

The electrical field generated by the eye approximates a simple dipole (Fig. 12-8A) with a posterior negativity centered at the retina and a relative positivity probably centered at the cornea. Eye movements change the orientation of this dipole. Polygraphic recording from strategically placed electrodes can detect these changes and can therefore be used to monitor eye movements. The Rechtschaffen and Kales (R&K) standard sleep scoring manual[27] (see also Chapter 18) recommends that an EOG use at least 2 channels (Fig. 12-8B). One electrode is placed 1 cm superior and lateral to the outer canthus of one eye. This electrode is input 1 to an amplifier; input 2 to this amplifier is an electrode attached to one ear or the mastoid. Another electrode is placed 1 cm inferior and lateral to the outer canthus of the other eye. This electrode forms input 1 to a second amplifier; input 2 to this amplifier is attached to the same ear as input 2 of the first amplifier. This placement scheme will detect conjugate horizontal and vertical eye movements. For example, when the eyes look to the right (see Fig. 12-8B), the cornea of the right eye approaches electrode A and electrode A becomes positive relative to the inactive ear. According to the polarity convention, amplifier 1 will register a downward deflection. Simultaneously, the retina of the left eye approaches electrode B. Consequently, electrode B becomes negative relative to the inactive ear and amplifier 2 registers an upward deflection. The out-of-phase deflections in the two adjacent channels indicate that a conjugate eye movement has occurred. Similarly, an upward eye movement results in an downward deflection in amplifier 1 and a upward deflection in amplifier 2. Eye blinks will produce an identical pattern because eye closure results in an upward rotation of the eyeball (Bell's phenomenon) (see also Chapter 11).

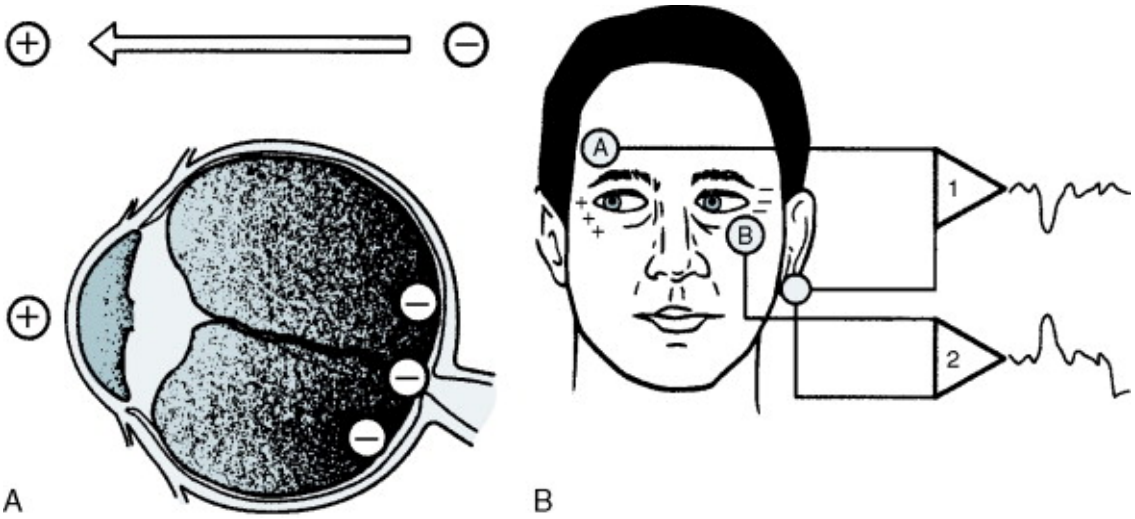

FIGURE 12-8 **(A)** The voltage field generated by the eye can be represented by a simple dipole, the cornea being positive and the retina negative. **(B)** Use of two polygraphic channels to detect conjugate eye movements according to the scheme suggested in the EOG sleep scoring manual.[18] Eye movements result in out-of-phase potentials in the 2 channels.

Some laboratories attach electrodes to both ears and refer the periocular electrodes to the contralateral ear (e.g., right upper canthus to left ear and left lower canthus to right ear). This minor change has several advantages. The longer interelectrode distances increase the amplitude of the deflections. The amplitude of the deflections generated by the movement of each eye is more likely to be equal because the interelectrode distances are equal. Finally, if one of the ear electrodes comes off during the study, the technician can refer both periocular electrodes to the remaining ear electrode and avoid waking the patient. Whereas these montages detect both horizontal and vertical eye movements, they cannot distinguish between them. This can be easily accomplished by recording inputs from supraorbital and infraorbital electrodes with a third amplifier.[4,][14]

Several varieties of eye movements are recorded during routine PSG. Although the patient is awake, saccadic eye movements as well as eye blinks are noted. Saccadic eye movements are rapid and can point in any direction. Eye blinks produce the same EOG pattern as vertical eye movement. One of the first signs of drowsiness is the cessation of any eye movements. Somewhat later in drowsiness, slow eye movements are seen. These usually have a frequency of less than 0.5 Hz,[28] are most consistently recorded in the horizontal axis, gradually increase in amplitude as background alpha activity drops out, and usually disappear in stage 2 sleep. REMs occur during REM sleep. Movements along the horizontal axis are the most common, although oblique and vertical movements occur as well. REMs typically occur in bursts and may be preceded by characteristic sawtooth waves on the EEG. There is no widely accepted definition of REMs that would serve to distinguish these from slow eye movements. Parameters useful for computerized quantification of REMs have been reported,[29] but these are not directly applicable to visual scoring. Radtke[30] has suggested a reasonable,

clinically applicable definition for REM, namely that the duration of the initial pen deflection is less than 200 msec and that the duration of the entire waveform is less than 1 second.

In a study of drowsiness in normal subjects, Santamaria and Chiappa[28] recorded eye movements with a sensitive motion transducer attached over the globe as well as with the traditional EOG. They found two types of eye movements not previously reported. What were named *small fast irregular eye movements* were found in 60% of normal subjects in early drowsiness, before the occurrence of slow eye movements. They did not appear in the routine EOG channels. What were called *small fast rhythmic eye movements* were found in 30% of normal subjects, usually associated with the traditional slow eye movements. These occasionally appeared in the routine EOG channels, although usually with a very low amplitude. If confirmed, these findings could be useful for determination of early stages of drowsiness.

ELECTROMYOGRAPHIC RECORDINGS IN SLEEP DISORDERS

EMG activities are important physiologic characteristics that need to be recorded for diagnosis and classification of a variety of sleep disorders. An EMG represents electrical activities of muscle fibers resulting from depolarization of the muscles after transmission of nerve impulses along the nerves and neuromuscular junctions.[6] An EMG could represent tonic, phasic, and rhythmic activity. Physiologically, there is a fundamental tone in the muscles, at least throughout the period of wakefulness and non-REM (NREM) sleep, but it is markedly diminished or absent in major muscle groups during REM sleep. Maintenance of muscle tone is a complex physiologic phenomenon that depends on suprasegmental, segmental, and peripheral afferent mechanisms.[31] Tone, therefore, may be influenced by a variety of extrinsic and intrinsic stimuli. After a nerve impulse, the resting muscle membrane potential is altered, and when it reaches a threshold level, depolarization of the muscle results from a change in the external and internal ionic balance and muscle calcium channel alterations.[6] The threshold depolarization causes an action potential to develop in the muscle. A compound muscle action potential represents summation of the action currents in many muscle fibers. Surface EMG recordings are of many muscle fibers, bundles, and groups; needle EMGs record approximately 15–20 muscle fibers near the needle tip.[6] Phasic EMG represents activities related to some physiologic alterations, either spontaneous or induced. Examples of phasic EMGs are EMG activities phasically related to inspiratory bursts and myoclonic muscle bursts that occur spontaneously or in response to some stimuli. If there are rhythmic activities (e.g., tremor), EMG bursts have a rhythm. In sleep disorders medicine, the tonic and phasic EMG bursts are usually the most important ones. As sometimes happens, however, rhythmic EMG bursts are noted in certain sleep disorders, such as patients with restless legs syndrome or other sleep-related movement disorders (see Chapter 28).

Method of Recording

EMG recordings from submental muscles using surface electrodes are routinely performed in PSG and multiple sleep latency tests. Electrodes placed in this area record mostly the activities of the mylohyoid and the anterior belly of the digastric muscles, which are innervated by the motor Vth cranial nerve. The electrodes also record some activities from the genioglossus and hyoglossus muscles (innervated by the XIIth cranial nerve) by volume conduction. This recording is important for identifying the presence or absence of muscle tone for sleep stage scoring.

In a patient suspected of restless legs syndrome, tibialis anterior muscles must be recorded, preferably bilaterally, because sometimes the periodic movements of legs alternate between the two legs. Ideally, the recording should also include 1 or 2 EMG channels from the upper limb muscles, as occasionally movements are noted in the upper limbs.

To understand the pathophysiology of sleep apnea, it is important to record the respiratory muscle activities (see also Chapter 14). These should include not only the intercostal muscles but also the diaphragmatic muscle, a variety of upper airway muscles, and the facial muscles. The true diaphragmatic activities are typically recorded by intraesophageal bipolar electrodes, which can also quantitate the diaphragmatic EMG activity.[32–34] This technique as well as esophageal pressure recording by inserting an esophageal balloon transnasally (which provides respiratory muscle mechanical activity) are invasive and uncomfortable. The noninvasive technique of placing surface electrodes to the right or left of the umbilicus or over the anterior costal margin may also pick up diaphragmatic activity, but the admixture of intercostal muscle activity makes this noninvasive technique unreliable for quantitative assessment of diaphragmatic EMG.[35] The intercostal EMG recorded from the seventh to the ninth intercostal space[36] with active electrodes on the anterior axillary line and the reference electrodes on the midaxillary line may record some diaphragmatic muscle activity in addition to the intercostal activity. Sharp et al.[37] compared data from chest wall surface electrodes (surface electrodes over the sixth and seventh intercostal space just above the right costal margin) with simultaneous data obtained from a swallowed bipolar electrode double-balloon catheter similar to that described by Onal and coworkers.[38] After performing power spectral analysis of diaphragmatic EMGs from surface electrodes and esophageal electrodes, Sharp et al.[37] concluded that thoracic surface recordings of the diaphragmatic EMG do not accurately reflect frequency information. Esophageal balloon manometry is important for a definite diagnosis of upper airway resistance syndrome.[39] In this condition, a narrowed upper airway results in increased work necessary to move air through a constricted airway but does not cause apneas or hypopneas.[39,][40] Esophageal balloon manometry in this condition documents abnormally increased negative intrathoracic pressure during inspiration associated with repeated arousals and fragmentation of sleep that are responsible for daytime hypersomnolence. Esophageal balloon manometry has been superseded by the use of a thinner and better tolerated water-filled catheter connected to a transducer.[40,][41]

An important muscle for recording respiratory activity is the alae nasi muscle.[34] This muscle picks up not only inspiratory activity but also some expiratory activity. Many upper airway muscles are accessory muscles of respiration. All the facial muscles, including the masseter muscles, show inspiratory bursts during EMG recordings.[36] To show the decrease of tone in the genioglossus and other oropharyngeal and laryngeal muscles, it is important to record EMGs from them. Intramuscular electrodes in humans are typically used to record inspiratory-related genioglossal muscle activity.[42–44] For many of these upper airway muscles, however, recordings can be made in a noninvasive manner by means of intraoral surface electrodes.[36,][45] For some laryngeal muscles, an invasive technique using fine-wire electrodes is required.[42] To

record the muscle activity from an individual muscle only, wire electrodes must be inserted into that particular muscle only.

For patients with suspected REM sleep behavior disorder (RBD), multiple muscle EMGs from all four limbs are essential—there is often dissociation in the activities between upper and lower limb muscles with these patients. Hence, if upper limb EMGs are not included in the recording, REM sleep without atonia may be missed in some cases.[46] Polysomnographic documentation of REM sleep without muscle atonia associated with excessive tonic and phasic EMG activities is a requirement in the second edition of the International Classification of Sleep Disorders for diagnosis of RBD.[47] This is, however, a qualitative documentation and there is no standardized, generally acceptable quantitative scoring method available.[48–51] A quantitative standardized scoring method is essential to determine the severity of RBD, monitor the effectiveness of therapy, and understand the pathophysiology of RBD.

In patients with nocturnal paroxysmal dystonia, which is now thought to be a form of nocturnal frontal lobe epilepsy, multiple muscle EMGs including all four limbs are required. In this condition, the patient displays dystonic-choreoathetoid movements; surface EMGs in addition to the video recordings are necessary to record these activities.

Clinical Significance of EMG Recording

EMG shows decreasing tone from wakefulness through stages 1–4 of NREM sleep. In REM sleep, the EMG tone is markedly diminished or absent. It is important to use the appropriate filters and very high gain at the beginning of the recording to appreciate the decreasing muscle tone during REM sleep. In certain pathologic conditions (e.g., RBD), the EMG tone may persist or phasic muscle bursts may be seen repeatedly during REM sleep. This may also happen in patients being treated for narcolepsy-cataplexy syndrome, and may represent a medication (e.g., selective serotonin reuptake inhibitors or tricyclic antidepressants being used to treat cataplexy) side effect.

EMG recordings of the tibialis anterior muscles are essential for the diagnosis of periodic limb movements in sleep (PLMS), which are seen in most of the cases of restless legs syndrome, a variety of other sleep disorders, and normal individuals, particularly older ones. Characteristics of the EMG bursts in PLMS are described in Chapter 28. In upper airway obstructive apnea, the EMG of the upper airway muscles shows marked decrease of tone during the apneic episodes, whereas the diaphragmatic and intercostal muscle activity persists.[36] During REM sleep, however, intercostal and even diaphragmatic EMGs show marked diminution of the tonic activity.[52]

In certain neurodegenerative diseases such as multiple system atrophy (Shy-Drager syndrome), laryngeal EMG recording may be important to detect vocal cord paralysis causing upper airway obstructive apnea.[53]

Multiple muscle EMG recordings are also important in patients with restless legs syndrome because of the presence of a variety of EMG activity types in these patients, including PLMS (predominantly dystonic), myoclonic bursts during both wakefulness and sleep, and a mixture of myoclonic and dystonic EMG bursts (Fig. 12-9) (see Chapter 28). Restless legs syndrome patients often have PLMS in the agonist and antagonist muscles (see Fig. 12-9), occasionally in the arms and rarely in the submental muscles (Fig. 12-10).[54]

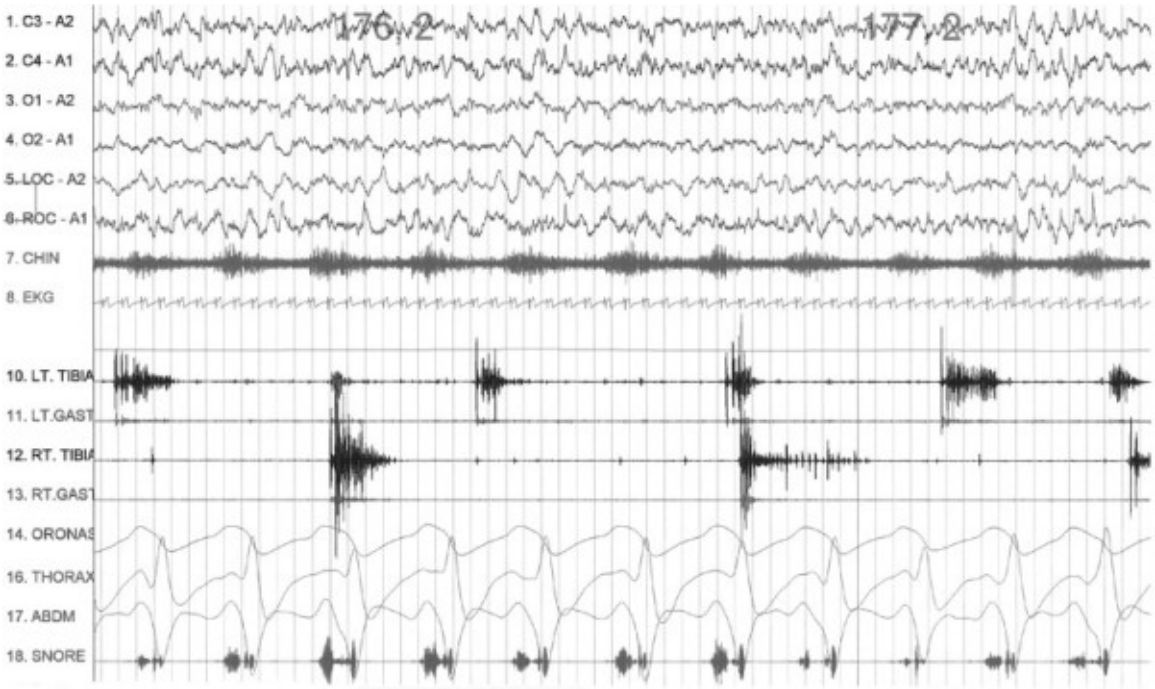

FIGURE 12-9 Polysomnographic (PSG) recording showing periodic limb movements in sleep (PLMS) characterized by dystonic and dystonic-myoclonic electromyographic (EMG) bursts in left (LT) and right (RT) tibialis (TIBIA) and gastrocnemius (GAST) muscles during stage 2 (N2) non–rapid eye movemen (NREM) sleep in an adult patient with restless legs syndrome (RLS). Top 4 channels show electroencephalograms (EEG) using international nomenclature. (A1, left ear; A2, right ear; ABDM, abdominal respiratory effort; CHIN, submental EMG; EKG, electrocardiogram; LOC, left electro-oculogram; ORONAS,

oronasal airflow; ROC, right electro-oculogram; THORAX, chest respiratory effort.)

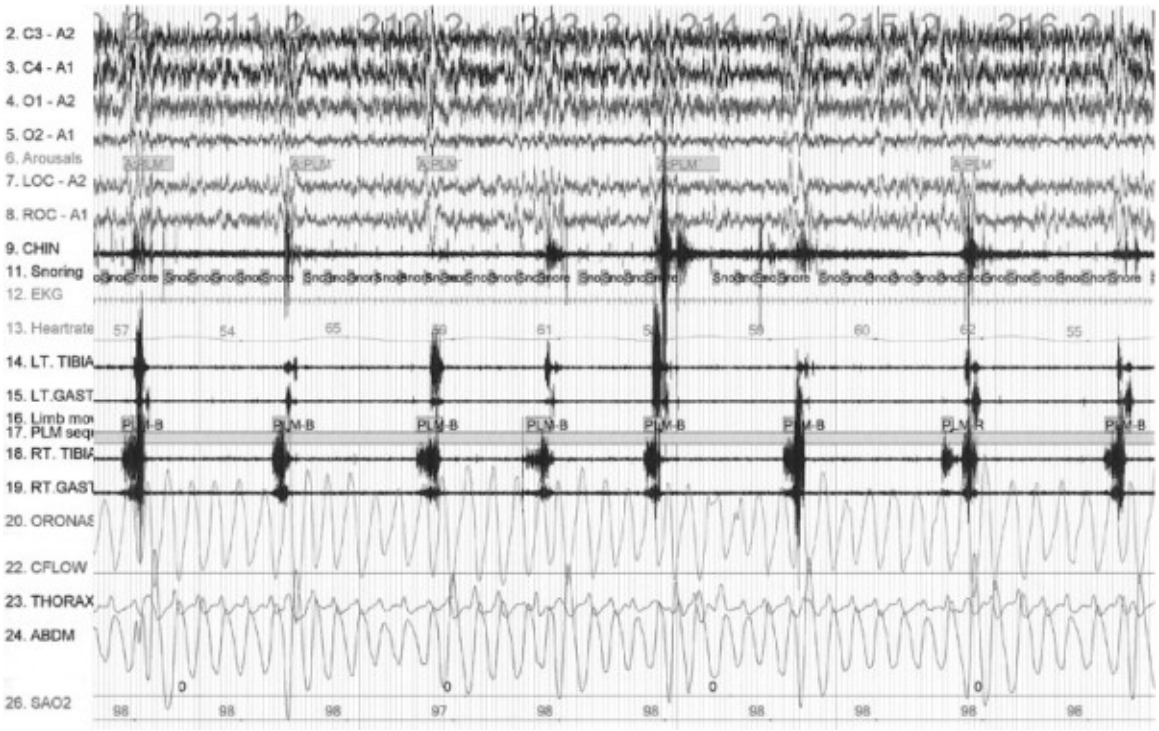

FIGURE 12-10 Overnight PSG recording from an adult patient with RLS showing PLMS in tibialis and gastrocnemius muscles bilaterally synchronous wit EMG bursts in submental (CHIN) muscles. (A:PLM, arousal with PLMS; Limb mov, limb movements; PLM seq, PLMS sequence; PLM-B, PLMS in right and left limbs; PLM-R, PLMS in right limb seen independently; SaO_2, oxygen saturation (%) by finger oximetry.) For description of the montage, see Figure 12-9

Multiple muscle EMGs may also be required for evaluation of propriospinal myoclonus at sleep onset, hyponic jerks, rare parasomnias such as sleep-related faciomandibular myoclonus,[55] and other sleep-related movement disorders (e.g., rhythmic movement disorder).

EMG recordings are needed to score excessive fragmentary myoclonus, hypnagogic foot tremor, and alternating leg muscle activation.[56] For research purposes, multiple muscle EMGs, including those of cranially innervated muscles, are necessary to understand the pathophysiology of post-polio syndrome,[57,][58] neuroleptic-induced akathisia,[59] and tardive dyskinesias.[60,][61]

ELECTROENCEPHALOGRAPHY

An EEG is recorded in sleep studies mostly to assist scoring of sleep stages. Obviously, EEGs can provide other useful information as well. Routine diagnostic EEGs usually sample at most 1 hour of electrocerebral activity. The PSG records EEG activity for much longer periods, increasing the likelihood that abnormalities will be recorded. Consequently, the polysomnographer must be familiar with the broad range of normal EEG findings and the abnormalities encountered in the various age groups. The following is at best an incomplete review of some of the major patterns encountered in routine EEG.

Normal Waking Rhythms

Individual waves recorded on the EEG can be characterized by their frequency (i.e., how many of that wave would be required to occupy a given period of time, typically 1 second). Frequency of EEG activity has been divided into four bands, assigned the Greek letters beta, alpha, theta, and delta. Scoring of sleep is largely based on the amplitude and frequency of EEG waves. Human electrocerebral activity is better characterized, however, by the broader concept of EEG *rhythms*. EEG rhythms can be defined as sustained periods of electrocerebral activity of similar frequency with a stereotyped distribution, reactivity, symmetry, and synchrony, and are associated with specific physiologic states. These rhythms have also been assigned Greek letters that correspond, in part, to the letters assigned to frequency.

Several rhythms characterize the awake adult EEG. The most obvious is the *alpha rhythm*. Alpha rhythm frequency varies between 8 and 13 Hz. This rhythm is distributed over the parieto-occipital regions bilaterally. A normal alpha rhythm is synchronous and symmetric over the two hemispheres. Frequency of the rhythm should not vary by more than 1 Hz, and amplitude should not vary by more than 50%. The alpha rhythm is best seen during quiet alertness with eyes closed. Various maneuvers cause alpha rhythm to react or decrease in amplitude. The most effective is opening the eyes, but any sort of intense stimulation produces some degree of amplitude attenuation. Up to 10% of normal adults will show no alpha rhythm during quiet wakefulness. The low-voltage EEG in these subjects is characterized by poorly sustained beta and theta frequencies with amplitudes between 10 and 20 μV. Occasionally, hyperventilation elicits a typical alpha rhythm in such patients. Low-voltage EEGs have not been recorded in normal subjects less than 10 years old.

Beta rhythms are present in virtually all adults, although they are usually less striking than alpha rhythms. Frequency of the beta rhythm is by definition above 13 Hz but typically ranges between 18 and 25 Hz. There are probably at least two beta rhythms, one distributed over the frontal and central regions and the other with a more diffuse distribution. Beta rhythms are present during wakefulness and drowsiness. They may appear more persistent during drowsiness, drop out during deeper sleep, and reappear during REM sleep. Amplitude over the two hemispheres should not vary by more than 50%. Amplitude of beta activity is less than 20 μV in 98% of normal drug-free subjects. A persistent beta rhythm with higher amplitude suggests use of sedative-hypnotic medications because most such medications increase amplitude of beta activity.

Mu rhythms are recorded in approximately 20% of routine daytime EEGs and are most common in young adults. This rhythm consists of brief trains of 7- to 11-Hz waves over the central regions, often with phase reversals over C3 or C4. The waves have a wicket or arciform shape (Fig. 12-11). Mu may occur synchronously or independently over the two hemispheres. This rhythm shows a characteristic reactivity. Active or passive movement of the limbs or even an intention to move a limb attenuates mu activity. Mu is seen during wakefulness and may become more prominent during stages 1 and 2 of NREM sleep. It typically disappears in slow-wave sleep and may reappear during REM sleep.[62] Direct EEG recordings from the human motor cortex have demonstrated superharmonics of mu activity that react to limb movement.[63] This has led to the conclusion that mu is a “ubiquitous rhythm of the sensorimotor cortex at rest.”[64]

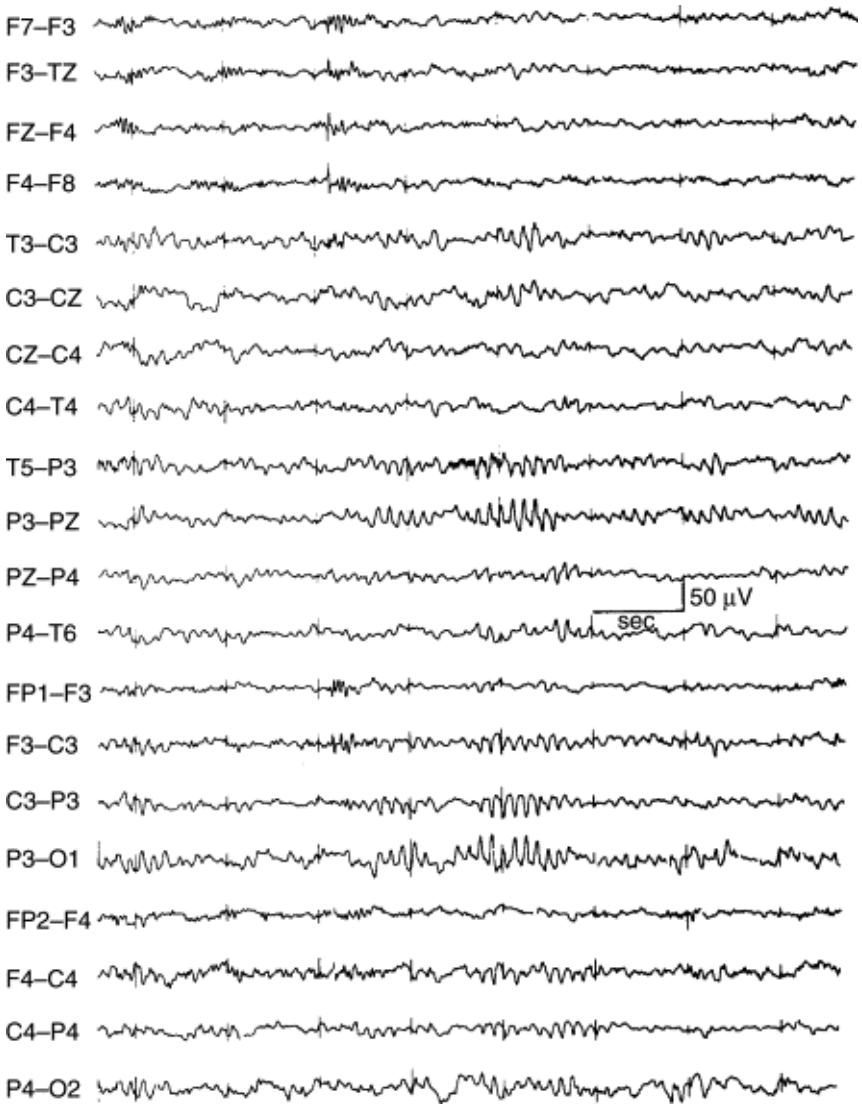

FIGURE 12-11 Mu rhythm in the left parietal region (P3). Note phase reversal of the 7- to 8-Hz comblike rhythm at P3 with spread of activity to C3.

Lambda rhythm is present in approximately 75% of young adults and becomes somewhat less common as individuals age. Lambda consists of a diphasic or triphasic waveform with the most prominent phase being a positivity at O1 or O2. The lambda rhythm is elicited by saccadic eye movements and appears to be an evoked response. It is present only during wakefulness with eyes open.

The EEG in infants and children undergoes significant evolution with increasing age. This paragraph emphasizes a few major points; the reader is referred to other sources for details.[65–67] A sustained parieto-occipital, alpha-type rhythm is not seen until approximately 3 months of age. At that time, reactive 3-Hz waves are recorded during wakefulness. Frequency of the parieto-occipital rhythm increases rapidly over the next several years, reaching adult values in most children by 3 years of age. *Polyphasic slow waves* (slow waves of youth) are found in the occipital regions bilaterally after 2 years in as many as 10% of normal subjects. Prevalence is highest at approximately age 10 and gradually decreases afterward. These waveforms rarely occupy more than 25% of the record, and they do not significantly exceed the amplitude of other background rhythms. Polyphasic slow waves react to eye opening in the same manner as the alpha rhythm; these waves are in fact considered a variant of the alpha rhythm. Greater amounts of random frontocentral theta activity are seen in children than in adults, but this decreases as the child ages. Brief runs of more sustained low-amplitude (15-μV) frontal 6- to 7-Hz waves are seen in as many as 35% of adolescents. This rhythm is present during quiet wakefulness with eyes open and may be related to affective arousal.

In the elderly, frequency of the alpha rhythm slows somewhat from a population mean of approximately 10.5 Hz to approximately 9 Hz. However, an alpha rhythm with a dominant frequency of less than 8 Hz is abnormal in adults. Focal temporal theta activity is seen in as many as 35% of asymptomatic individuals older than 50 years. Such activity is more commonly noted over the left temporal regions and should probably occupy no more than 5% of the tracing. Slower frequency or more persistent temporal slowing is considered abnormal by the authors. The exact point at which temporal slowing in the elderly can be considered unequivocally abnormal, however, remains controversial.[68–70]

Sleep Electroencephalography in Adults

The R&K standard scoring manual[27] divides sleep into four stages (see the American Academy of Sleep Medicine modification in Chapters 2 and 18). EEG findings in each of these stages are discussed in this section.

Drowsiness or *stage 1 NREM sleep (N1)* is used to designate the transition between wakefulness and stage 2 sleep and beyond. Stage 1 sleep is also briefly seen after arousals from other sleep stages and often transiently precedes and follows periods of REM sleep. Because this is a transitional sleep stage, it occupies a relatively small percentage of a normal night's sleep, generally less than 5%.[71]

A number of studies have emphasized the fact that EEG activity recorded during the transition between wakefulness and stage 2 NREM (N2) sleep is variable and complex. EEG and physiologic changes that are inconsistent with wakefulness clearly occur before stage 1 as defined in the R&K sleep scoring manual. Santamaria and Chiappa[28] identified several phases of drowsiness and more than 20 distinct EEG patterns in a careful study of 55 normal adults. The patterns of drowsiness varied in different subjects and varied in the same subject at different times. An early phase was characterized by changes in the alpha rhythm that persisted throughout this phase. Alpha may shift from its characteristic parieto-occipital distribution to the frontocentral or temporal regions. Amplitude of alpha activity may either increase or decrease, and frequency of the alpha rhythm may slow. Slower theta and delta frequencies may be superimposed in the central or temporal regions. These may have a paroxysmal or sharpish character and be confused with epileptiform potentials. Paroxysmal theta bursts may predominate in one temporal region and be misinterpreted as the temporal sharp waves often associated with complex partial seizures. Criteria for distinguishing these benign potentials from genuine focal epileptiform discharges have been outlined.[72]

As the alpha rhythm disappears, bursts of frontocentral beta and generalized delta slowing may appear. Frankly paroxysmal but nonetheless benign patterns are occasionally seen in normal adults at this time as well. *Benign epileptiform transients of sleep* (BETS) are seen in 5–24% of normal subjects.[28,][73] These are spiky, often diphasic transients with a broad field of distribution, usually involving both hemispheres. They typically shift from side to side and become less frequent during deeper sleep stages. Although BETS may superficially resemble epileptiform spikes, they are not associated with seizure disorders. White and coworkers[73] have outlined useful criteria distinguishing BETS from genuine epileptiform discharges. Less frequently, paroxysms of 6-Hz spike-and-wave discharge (Fig. 12-12) may be noted in either the frontal or temporal regions. The spike component usually has a relatively low amplitude, whereas the following slow wave is more prominent. Paroxysms of such activity rarely last longer than 3 seconds,[74] have an evanescent quality, and are less common during deeper sleep. Despite their paroxysmal quality, they are not associated with seizures either.[74]

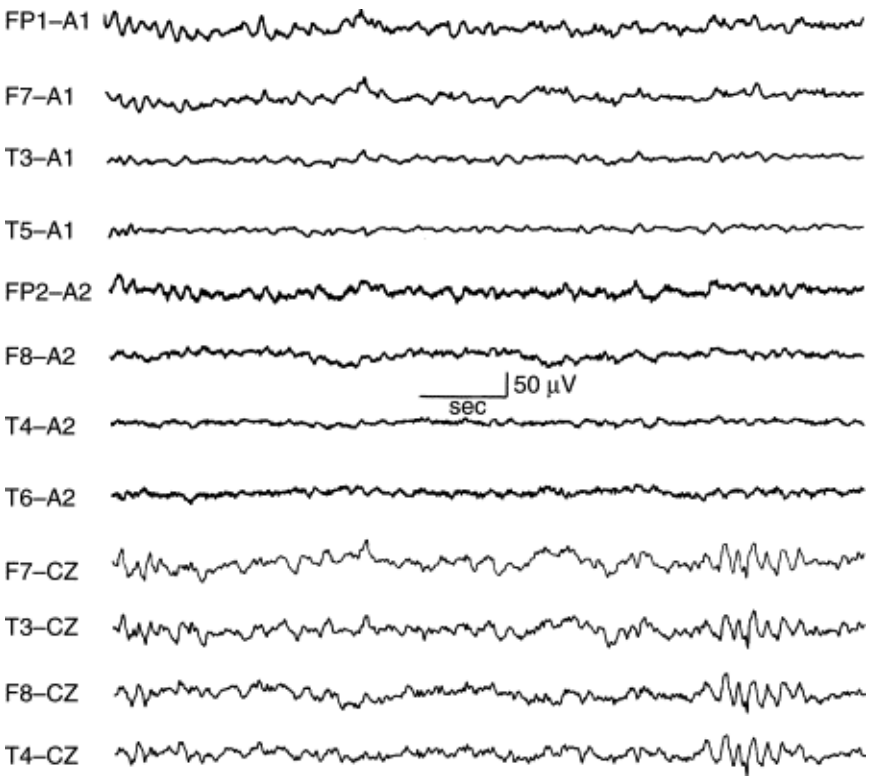

FIGURE 12-12 Spike-and-wave discharge of 6 Hz (phantom spike and wave) seen in the last 4 channels.

Eventually, the alpha rhythm disappears altogether. *Vertex waves* are now frequently present. These are high-voltage sharp transients, surface negative, followed by a lower voltage, surface-positive component. They have maximal voltage at the Cz electrode. Mild asymmetry between the two hemispheres and extension of the field to Fz, or less frequently Pz, is not uncommon. Vertex waves occur spontaneously or in response to stimuli that are insufficient to fully arouse the

subject. *Positive occipital sharp waves of sleep* (POSTS) appear in post–transitional stage 1, although these potentials are more common in deeper sleep stages (Fig. 12-13). These are diphasic or triphasic sharp waves with a predominant positive phase at the occipital electrodes. They have a triangular appearance similar to lambda waves. POSTS are noted synchronously over the two hemispheres and may occur singly or in runs. Occasional shifting amplitude asymmetry is noted in normal controls; however, persistent significant asymmetry should raise suspicion of a posterior lesion. Because these potentials have a paroxysmal sharpish appearance, they may be confused with epileptiform discharges.

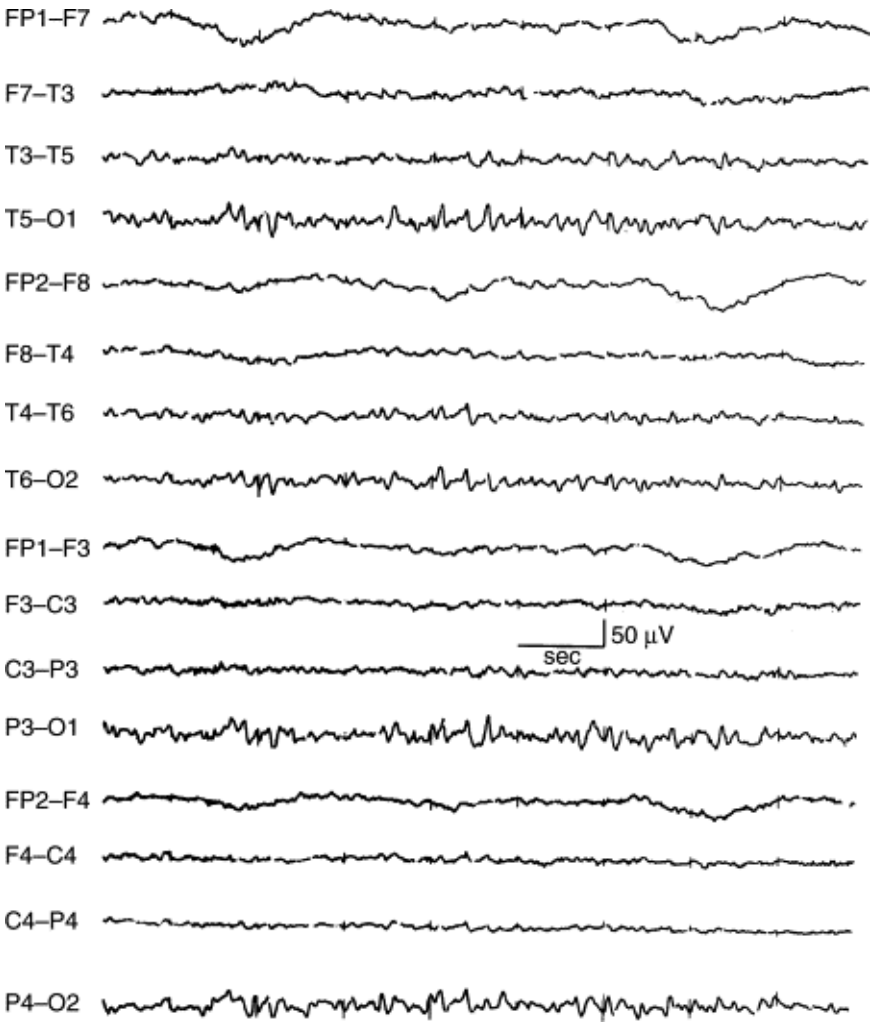

FIGURE 12-13 Positive occipital sharp transients (T5-O1, T6-O2, P3-O1, P4-O2 channels) during stage 1 NREM sleep.

To summarize, drowsiness, or stage 1 (N1), is a transitional state with many shifting and variable EEG patterns. Some of these resemble abnormal patterns, and determining whether an individual potential is normal may be difficult with the limited EEG montages typically used in PSG. It is important not to overinterpret. If there is uncertainty, routine EEG with a full complement of electrodes and multiple montages often clarifies the issue.

The R&K standard sleep scoring manual defines *stage 2 NREM sleep (N2)* by the presence of sleep spindles of at least 0.5-second duration or K complexes, as well as the absence of the features of slow-wave sleep (N3). Stage 2 sleep (see Fig. 2-3) comprises the bulk of a normal night's sleep (approximately 50% in normal adults).[71] Sleep spindles consist of a sequence of 12- to 14-Hz sinusoidal waves typically lasting a second or more. Voltage is usually maximal over the central regions. There is a high degree of symmetry and synchrony between the two hemispheres in normal subjects older than 1 year. Some investigators have proposed a classification of spindles based on topography and frequency,[64,][75,][76] but it is not clear that this has clinical utility at this time.

K complexes have been defined differently by sleep disorder specialists and electroencephalographers, which may confuse those trained in both disciplines. The R&K sleep scoring manual[27] defines the K complex as a well-delineated negative sharp wave followed by a positive component. The K complex must exceed 0.5 seconds in duration and may or may not be accompanied by sleep spindles. Vertex waves are not specifically defined in the manual or the sleep disorders glossary of terms.[77] Most polysomnographers accept that vertex waves have a duration of less than 0.5 seconds and distinguish vertex waves from K complexes on the basis of duration, although this may be difficult at the slow paper speeds often used. This distinction is important when scoring sleep because K complexes, even without spindles, are sufficient for scoring stage 2 sleep, whereas vertex waves alone do not allow the scoring of stage 2 sleep. Glossaries of EEG terminology,[78] in contrast, insist that K complexes always have associated sleep spindles and do not specify a duration.

The EEG in *stages 3 and 4 NREM sleep (N3)* (see Figs. 2-4 and 2-5) is marked by high-amplitude slow waves. The R&K sleep scoring manual[27] requires that more than 20% of any epoch be occupied by slow waves slower than 2 Hz and greater than 75 μV for stage 3 and that more than 50% of any epoch be occupied by slow waves with these characteristics for stage 4. Computerized analyses indicate that sleep spindles, vertex waves, and POSTS[79,][80] are abundant in stages 3 and 4, although they may be less discernible to the interpreter's eye because of the abundant slow activity.

During *REM sleep* (see Fig. 2-6), the background EEG is characterized by low-voltage, mixed-frequency activity similar to early stage 1. Alpha frequencies are often present and may be more persistent than in stage 1. The alpha frequencies are usually 1–2 Hz less than the subject's waking rhythm.[81] Vertex waves, sleep spindles, and K complexes are absent. Characteristic *sawtooth waves* are frequently recorded. These are 2- to 3-Hz sharply contoured triangular waves, usually occurring serially for several seconds with highest amplitude over the Cz and Fz electrodes. A series of sawtooth waves typically precedes a burst of REM.[82,][83] REM sleep occupies 20–25% of a night's sleep in a normal subject.[71] Brief periods of stage 1 sleep typically precede and follow a period of REM sleep. Detailed rules for demarcating onset and termination of REM sleep in these and other circumstances have been outlined in the R&K sleep scoring manual.[27]

Various *atypical PSG patterns* have been described. These usually occur in various sleep pathologies or when sleep has been significantly disrupted in normal individuals. *Alpha-delta sleep* (see Fig. 33-1) is characterized by persistence of alpha activity during stages 3 and 4. Excessive alpha intrusion may be seen in stage 2 as well, and the abundance of spindles appears to be decreased. This pattern appears to be associated with nonrestorative sleep and is seen in a variety of conditions.[84,][85] It may signal the fibromyalgia syndrome. Moldovsky and Scarisbrick[86] have elicited this EEG pattern in normal subjects by selectively depriving them of stage 4 sleep. Deprivation of stage 4 sleep also elicited complaints of diffuse arthralgias, myalgias, and fatigability, similar to the complaints of the fibromyalgia syndrome. *REM-spindle sleep* (Fig. 12-14) is characterized by the intrusion of sleep spindles into portions of the PSG that otherwise meet all criteria for REM sleep. This pattern may be seen in 1–7% of normal subjects[87] but is more common when sleep is disrupted and after the first night of continuous positive airway pressure treatment. Broughton[88] reviewed other atypical patterns that occasionally occur during REM sleep.

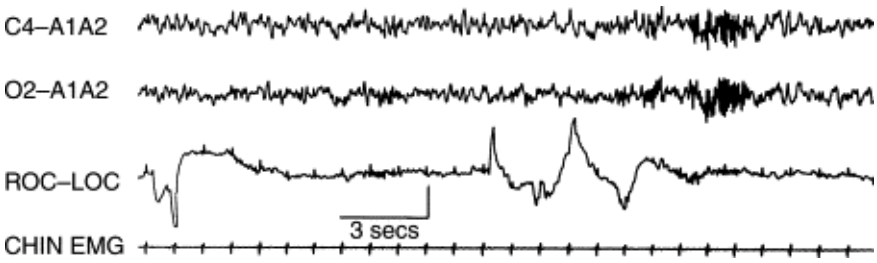

FIGURE 12-14 REM-spindle sleep. Note intrusion of sleep spindles in the EEG channels of a portion of a polysomnography that meet all criteria for REM sleep. REM is present in the eye channel—outer canthus of the right and left eye (ROC-LOC)—and atonia in the chin electromyography (CHIN EMG). Calibration (*vertical bar*) is 50 μV for the top 3 channels and 20 μV for the bottom channel.

Normal Sleep Electroencephalography in Pediatrics

The transition from neonatal to infantile EEG sleep patterns occurs between 1 and 3 months. Even after this period, there is a great deal of change in the EEG patterns until the adult patterns are reached. The major points are emphasized in the following paragraphs; the reader is referred elsewhere for more detailed discussion (see also Chapters 2 and 38).[65–67,][89]

Drowsiness in the pediatric age group differs from the adult patterns in several ways. Before 8 months of age, drowsiness is marked by a progressive slowing of EEG frequencies until delta waves predominate. After 8 months, the onset of drowsiness is marked by long runs of continuous, generalized, high-voltage, rhythmic theta or delta rhythms that have been called *hypnagogic hypersynchrony*. Three types have been described in normal subjects.[66,][67,][89] In the most common type, the rhythmic slow waves have highest amplitude in the frontal and central regions. The continuous rhythmic slowing may persist for several minutes. Less commonly, amplitude is highest in the parieto-occipital regions. Finally, a paroxysmal type occurs in approximately 10% of normal children. With this pattern, the alpha rhythm is gradually replaced by mixed frequencies. Diffuse bursts of 2- to 5-Hz slow waves, a few seconds in duration, then appear intermittently. Occasionally, random, poorly developed, sharpish waveforms are noted amid the slow waves. These may be random, superimposed alpha transients and should not be confused with epileptiform spike-and-wave discharge. The first two types of hypnagogic hypersynchrony are rarely recorded after age 10. The paroxysmal type persists into the mid-teens or, rarely, into adulthood.[66,][67] In infancy and early childhood, 20- to 25-Hz beta activity is also a prominent feature of drowsiness. The beta rhythm may have maximum voltage anteriorly or posteriorly or have a diffuse distribution. The amplitude may reach 60 μV. This pattern appears at 6 months and is seen most frequently from 12 to 18 months. Prevalence decreases subsequently, and prominent beta activity during drowsiness is rarely seen after 7 years.[89]

Vertex waves and K complexes appear at age 6 months. These potentials are rather blunt and may reach amplitudes exceeding 200 μV in infancy and early childhood. By age 5, both vertex waves and K complexes have an increasingly spiky configuration. Mild asymmetry is quite common. They may occur repetitively in brief bursts.

Sleep spindles appear at approximately 3 months of age. Between 3 and 9 months of age, spindles occur in wicket-like trains often exceeding several seconds. These potentials are common at this age, often occupying as much as 15% of stage 2 (N2) sleep. Asynchrony between the hemispheres is the rule, with only half of the trains demonstrating interhemispheric synchrony at 6 months.[90,][91] Interhemispheric synchrony increases to 70% by 12 months, and the duration and frequency of the spindle bursts gradually decrease. By 2 years of age, virtually all spindle trains are synchronous; however, spindles are much less frequent, occupying only 0.5% of stage 2 sleep.[90,][91] Spindles remain infrequent until approximately 5 years of age.[91]

Stages 3 and 4 (N3) sleep are marked by high-amplitude, slow activity, as in adults. However, amplitude of the slow activity is usually higher. An occipitofrontal gradient is often present, with the very-high-amplitude, slower frequencies predominating posteriorly and lower amplitude, faster frequencies predominating anteriorly.[92] This gradient becomes less striking with age, so that by 5 years, the slow waves are distributed more diffusely.

The EEG during REM sleep in infants and children is characterized by a greater amount of slow activity than is seen in adults. The mature desynchronized EEG with scattered alpha rhythms emerges during the mid-teens.[93] The percentage of a normal night's sleep occupied by REM gradually decreases from 40% at ages 3–5 months to 30% at ages 12–24 months and then gradually assumes adult values after puberty.[94] REM onset latency gradually lengthens over the first year of life as well.

Abnormal Electroencephalographs

Many abnormal EEG patterns have been described. Only frequently encountered abnormalities are discussed in this section. *Diffuse slowing of background activity* (Fig. 12-15) is probably the most commonly recorded EEG abnormality. It can take several forms. One may see slowing of the parieto-occipital, alpha-type rhythm to a frequency below that allowable for the patient's age. Alternatively, frequency of the alpha-type rhythm may be normal but excessive, and diffuse theta and delta activity may be recorded. Finally, one may see both a slowing of the alpha-type rhythm and excessive, diffuse slower frequencies. Before concluding that an EEG has excessive slowing of background frequencies, the polysomnographer must consider the patient's age and state of alertness. More diffuse theta activity is seen in normal children than is acceptable for adults. Frequency of background rhythms must be assessed while the patient is clearly awake. As noted earlier, both slowing of alpha-type rhythms and diffuse slower frequencies are commonly found in drowsiness in normal subjects. Consequently, the polysomnographer must be certain that the background frequencies are slow during wakefulness. Unfortunately, diffuse slowing of background frequencies is a very nonspecific pattern. It is commonly interpreted as being consistent with a variety of diffuse encephalopathies, including toxic, metabolic, and degenerative encephalopathies, among others.

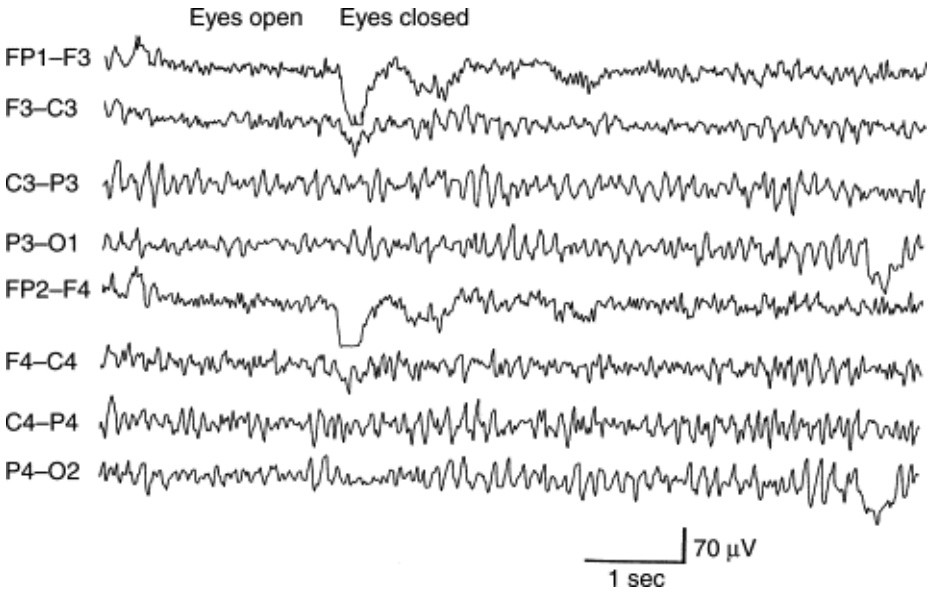

FIGURE 12-15 Diffuse slowing in a 67-year-old patient with dementia. Activity of 6–7 Hz predominates over the parieto-occipital regions. Although it is reactive to eye closure, the frequency of this rhythm is abnormally slow. Calibration: vertical bar = 70 µV, horizontal bar = 1 sec.
(Reproduced with permission from Emerson RE, Walczak TS, Pedley TA. EEG and evoked potentials. In LP Rowland [ed], Merritt's Textbook of Neurology. Philadelphia: Lippincott Williams & Wilkins, 2000:64.)

Focal slowing (Fig. 12-16) means that slow frequencies predominate over one region of the brain. Electrocerebral activity elsewhere is normal, or generalized slowing is present but is relatively mild. In experimental models, focal slowing is produced by focal white matter lesions, even when the cerebral cortex remains intact.[95] Focal cerebral lesions often involve both white matter and cortex, however, so the usefulness of this distinction is blurred in practice. A structural lesion must always be suspected when persistent focal slowing is recorded. Not all patients with focal slowing, however, will have neuroradiologically demonstrable lesions.[96] Patients with transient ischemic attacks or focal epilepsy often have

focal EEG slowing even when complete neuroimaging evaluations are normal. In epilepsy patients, this slowing may be due to ongoing local inhibitory phenomena or may be a transient postictal finding.

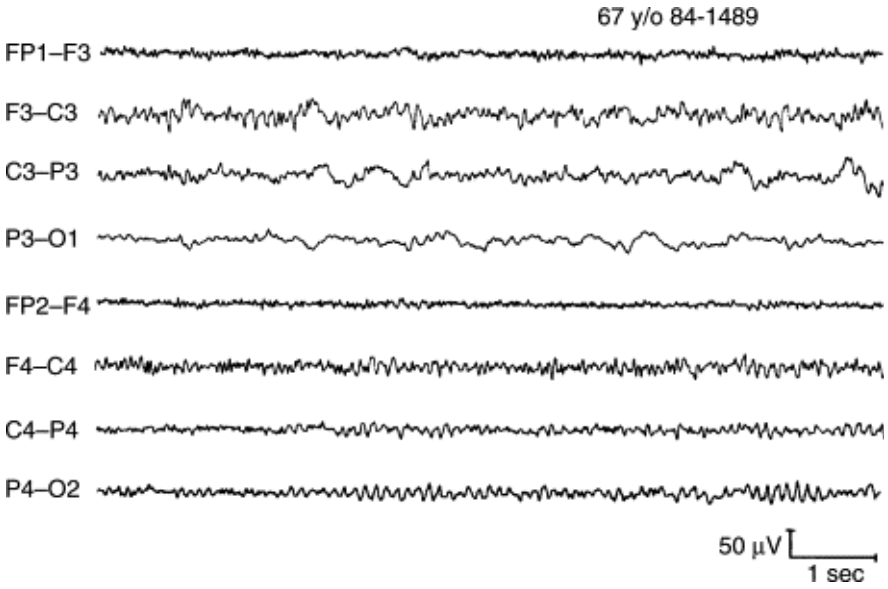

FIGURE 12-16 Focal left hemispheric slowing in a 67-year-old (y/o) patient with a large left hemispheric infarction. Left hemispheric alpha rhythm is also attenuated.
(Courtesy of Dr. Timothy Pedley.)

Focal attenuation of background rhythms means that frequencies in one region of the brain have significantly lower amplitude than elsewhere. In experimental models, focal attenuation of background is produced when the gray matter is lesioned and the underlying white matter remains intact.[95] Consequently, focal attenuation is often interpreted as indicating focal cortical dysfunction. In practice, attenuation of background frequencies is usually seen in combination with focal slowing (see Fig. 12-16). Neuroradiologic investigations usually reveal large lesions involving both cortex and white matter.[97,][98] Any fluid collection between the cortex and the recording electrode attenuates the recorded EEG activity. Thus, subdural fluid collections and subgaleal hematomas may result in a focal attenuation of background, although the cortex may not be damaged.

The detection of *epileptiform discharges* is important because these potentials have close association with epilepsy. Pedley[99] suggested that an epileptiform discharge should meet several criteria:

1. It must be paroxysmal, which means that it must clearly stand out from the background.
2. An epileptiform discharge must be spiky, which means that the transition from ascending to descending phase is abrupt and the duration of the discharge is short (by convention, 200 msec).
3. It must have a clear field—that is, it should not be confined to one electrode.
4. It should have negative polarity, because epileptiform discharges with positive polarity are uncommon.*
5. Finally, a slow wave often follows an epileptiform discharge.

Several varieties of epileptiform discharges have been described and associated with epilepsy syndromes.[99,][100] A basic distinction is made between generalized and focal epileptiform discharges. Generalized epileptiform discharges indicate that the patient's seizure is likely to start simultaneously throughout the brain. An example is the generalized 3-Hz spike-and-wave discharge (see Fig. 30-2) that is characteristic of petit mal absence seizures. Focal epileptiform discharges indicate that the patient's seizure is likely to start in a restricted area of the brain, although it may subsequently spread. An example is the anterior temporal sharp wave that is characteristic of complex partial seizures of temporal lobe origin (Fig. 12-17). This is an important distinction because the treatment and prognosis in these two epilepsy syndromes are very different.[100] Approximately 90% of adults with epileptiform discharges will have a history of seizures,[101,][102] and incidental epileptiform discharges are very uncommon in normal adults.[103] The association of epileptiform discharges with seizures in the pediatric age group is not as strong and varies with patient age and type of epileptiform discharge.[104]

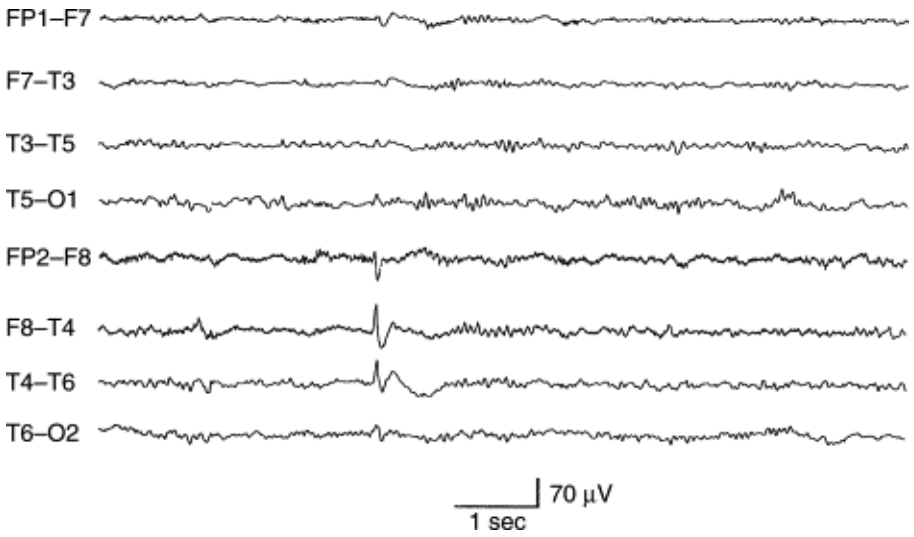

FIGURE 12-17 Right temporal interictal epileptiform discharge in a 32-year-old patient with complex partial seizures. Calibration: vertical bar = 70 µV, horizontal bar = 1 sec.
(Reproduced with permission from Emerson RE, Walczak TS, Pedley TA. EEG and evoked potentials. In LP Rowland [ed], Merritt's Textbook of Neurology. Philadelphia: Lippincott Williams & Wilkins, 2000:64.)

The polysomnographer must be able to recognize *electrographic seizures* (see Fig. 30-10). These may occur in patients with epilepsy or in patients with sleep apnea during severe hypoxia. The EEG patterns associated with seizures are extremely variable. In general, an electrographic seizure has abrupt onset, has sustained and rhythmic evolution of frequencies, spreads to contiguous areas of the brain, and terminates abruptly, often followed by irregular postictal slowing. Typically, faster frequencies are seen at seizure onset and these gradually decrease in frequency as the seizure continues. Seizures associated with hypoxia usually have a generalized onset. A good deal of experience is necessary to recognize the various EEG patterns that can occur during a seizure. In practice, any sustained and evolving rhythm with an abrupt onset raises concern about electrographic seizures. However, the polysomnographer must recall that drowsiness and arousal responses may begin abruptly and have rhythmic, sustained characteristics as well, especially in children.

Periodic lateralizing epileptiform discharges (PLED) are another important pattern to recognize. In this pattern, epileptiform discharges are recorded continuously over a given region (Fig. 12-18). The epileptiform discharges occur at regular intervals, usually every 1–2 seconds, and are thus labeled periodic.[105,][106] Background activity is usually significantly attenuated on the side with the discharges, and excessive slow frequencies are often seen bilaterally.[105,][106] This pattern is usually associated with an acute focal cerebral insult. In a review of 586 cases reported in the literature,[107] 35% were related to an acute cerebral infarction, 26% to other sorts of mass lesions, and the remainder to infection, anoxia, or other causes. Clinically, PLED is associated with obtundation, seizures, and focal neurologic deficits. Seventy to 90% of patients with PLED have seizures during the acute stage of their illness.[105–107] Twenty-five to 40% of patients with this pattern die in the hospital or shortly after discharge. Mortality may be especially high in patients with acute stroke and PLED.[105,][106,][108] PLED is almost always a transient phenomenon. The discharges become less frequent and lower in amplitude over the 2 weeks after the acute insult and are gradually replaced by focal delta slowing.[109]

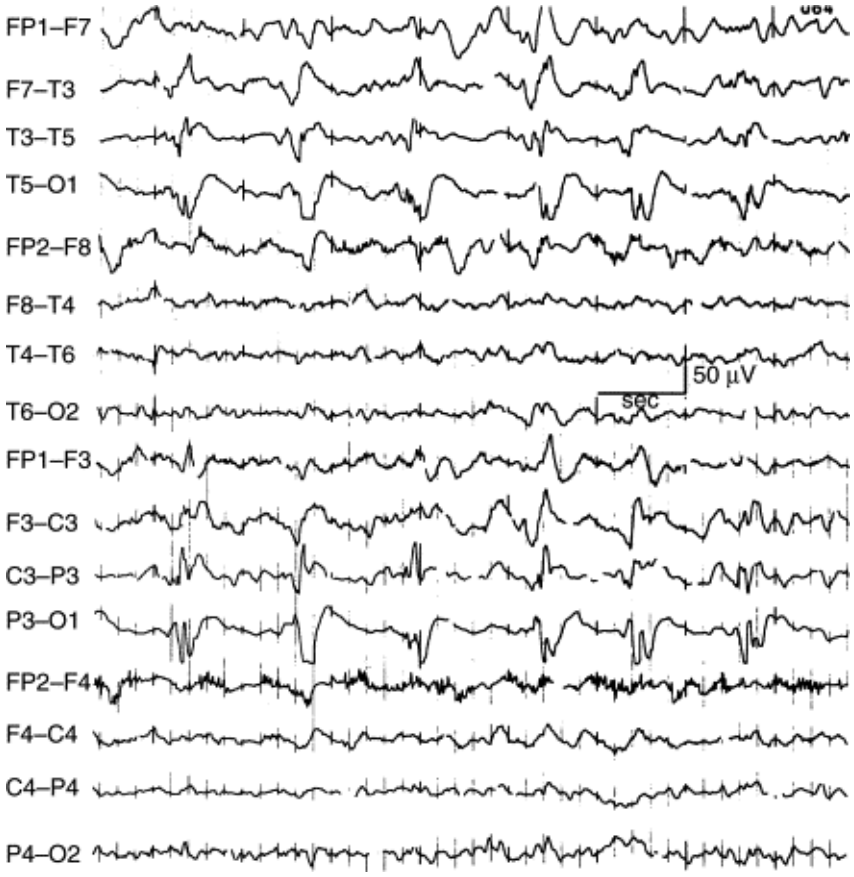

FIGURE 12-18 Periodic lateralized epileptiform discharges at a rate of 0.8/sec arising from the left parietal and posterior temporal regions (P3, T5) in a 7 year-old woman with a history of confusion and falling episodes.

Polysomnography typically does not utilize a full complement of scalp electrodes because the major clinical issue is scoring of sleep stages rather than detection of electrocerebral abnormalities. Distinguishing EEG abnormalities such as persistent focal slowing may be difficult if only a few electrodes are devoted to EEG. Nonetheless, the polysomnographer should be thoroughly familiar with common EEG abnormalities. Suspicious activity should prompt re-montaging and further examination. If this is unrevealing and suspicions remain high, routine EEG with a full complement of electrodes should be performed.

Artifacts

The polygraph is designed to record the relatively small voltages generated by the human brain, muscles, eyes, and heart. Unfortunately, the remainder of the human body and the surrounding environment are not electrically silent. These generate abundant electrical activity that may obscure the biological signals of interest. This extraneous electrical activity is called *artifact* (see also Chapter 11).[2–5] Making the distinction between the signal of interest and artifact is a central task for the polysomnographer, and the task is most difficult when interpreting an EEG. Because high sensitivities are required to record the relatively low voltages generated by the brain, extraneous voltage sources are especially likely to contaminate the EEG recording.

Four sources of artifact exist: (1) irrelevant physiologic signals, (2) environmental signals, (3) aberrant signals due to faulty or improperly applied electrodes, and (4) aberrant signals produced by the polygraph. More than one of these sources can contribute to a particular artifact. The following discussion summarizes frequently encountered artifacts and is by no means exhaustive.

Irrelevant Physiologic Signals

Irrelevant signals may contaminate recording of biological signals of interest, especially EEGs. *Myogenic potentials* originating from scalp muscles may obscure EEG recording (see Figs. 11-9 and 11-11). Myogenic activity may be difficult to distinguish from electrocerebral activity in the beta frequency range, especially at slow paper speeds. It may obscure lower amplitude electrocerebral activity.

Head movement also causes artifacts, frequencies of which are usually in the delta range (see Fig. 11-13). These artifacts are due to changes in electrode impedance, together with spurious static and capacitative potentials. Head movement artifacts are induced by slight movement of the electrodes on the scalp and the swaying of wires. The head movements associated with respiration often elicit movement artifact on the EEG, especially when the patient is lying on the recording electrodes. Correlating the spurious delta waves on the EEG with a respiratory monitor establishes their

artifactual source. This may be important because these spurious potentials should not be used to score slow-wave sleep.

Sweating may result in very slow frequencies and changes in baseline, especially when direct current amplifiers are used in the polygraph. The salt content of sweat changes the ionic composition of the conducting gel, resulting in this particular artifact. Potentials that arise from the sweat glands also play a role. Sweating may be asymmetric, and the resulting EEG asymmetry may mislead the interpreter.

Pulse artifact occurs when an electrode is placed on one of the scalp arteries. The electrode movement caused by the pulsations produces a delta wave. The regular relationship of the delta wave to the electrocardiogram (ECG) indicates the extracerebral origin of this activity.

The electrical fields generated by *ECG* and eye movements are commonly recorded from scalp electrodes (see Fig. 11-10) and may be confused with electrocerebral activity. Again, referring to the channels that are recording ECG and eye movements demonstrates whether suspicious activity recorded at the scalp is caused by these extracerebral sources.

Environmental Signals

The hospital environment contains many sources of electrical signals that may mimic electrocerebral or other physiologic activity. The *circulation of moistened air* through a respirator tube may induce bursts of alpha or theta frequencies at scalp electrodes. The electrostatic charges on drops entering an intravenous cannula—*intravenous drop artifacts*—may cause periodic spike-like artifacts. *Intravenous infusion pumps* can cause bursts of spiky transients followed by slower components. These artifacts are thought to be due to electromagnetic (rather than electrostatic) sources. *Telephones and pager systems* are among the other potential sources of environmental artifacts. The interpreter relies on the technician to correlate unusual recorded potentials with specific events in the environment, thereby establishing the artifactual nature of the potentials.

Sixty-hertz electromagnetic radiation due to alternating current in power lines is ubiquitous in the hospital environment and may contaminate the recording (in Europe, mains frequency is 50 Hz). The resulting 60-Hz artifact may be impossible to distinguish from myogenic activity at the slow paper speeds commonly used for PSG. The presence of this artifact in EMG leads may persuade the interpreter that tonic EMG activity is at a high level when it is actually low. The 60-Hz artifact is verified when 60 cycles are counted in 1 second of recording. Usually, paper speed must be increased to at least 60 mm/sec to distinguish adjacent potentials of this frequency and count them accurately. After the presence of 60 Hz is verified, the technician should proceed systematically to determine the source of the artifact. First, the technician must ensure that both of the involved electrodes are in fact attached to the patient, plugged into the jackbox, and connected to the relevant amplifier. The integrity of the patient ground and the reference electrode must be similarly ensured. Next, the technician should check the impedances of the involved electrodes. Impedances in any electrode pair should not exceed 10 kohm, and the impedances of the two electrodes should be roughly equal. Only then can the technician conclude that the electrode-scalp interface is probably not the source of the artifact. At this point, the technician should search for 60-Hz sources in the environment. A “dummy patient,” consisting of two leads shorted with a 10-kohm resister, may be carried around the room until the 60-Hz artifact reaches maximal amplitude and the source is identified. Finally, the technician should remember that faults with instrument ground may result in 60-Hz activity as well.

Aberrant Signals from Faulty Electrodes

Improperly applied electrodes or electrode faults may result in other sorts of artifact.

Electrode “pops” are the most common electrode artifact. These are abrupt vertical transients (Fig. 12-19), usually of positive polarity, that are confined to one electrode. They are superimposed on but do not modify ongoing recording. Pops are due to abrupt changes in impedance and usually indicate either that the electrode is not securely attached or that electrolyte gel is insufficient. When confronted with a popping electrode, the technician should reset the electrode and apply more gel. If popping persists, the electrode needs to be changed. Occasionally the electrode impedances change more gradually, mimicking slow activity. Again, the observation that the slow activity is confined to one electrode indicates that the electrode, rather than the body, is the source of the potential.

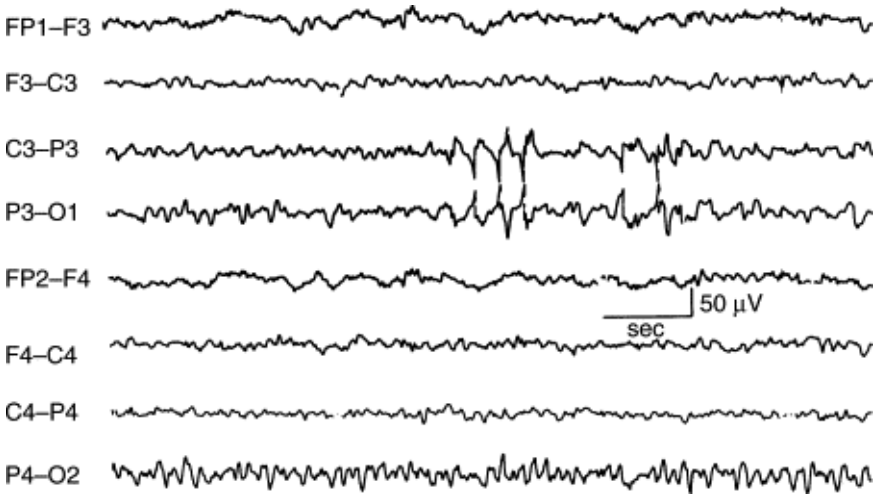

FIGURE 12-19 Electrode pops at P3 electrode.

Other electrode faults may result in artifact even if the electrode-scalp interface is intact. An interruption in the plating of the electrode may result in battery potentials, which can appear as bizarre, high-amplitude discharges confined to the faulty electrode. A similar artifact may occur when electrode gel connects the disk electrode and the wire lead, which are usually made of different metals.

Aberrant Signals from the Polygraph

Finally, the polygraph can be a source of artifact. Random fluctuation of charges in any complicated recording instrument results in some spurious output. In contemporary digital equipment, this *instrument noise* is infrequent and has low amplitude. It should not contaminate recording at standard sensitivities but may occasionally appear when sensitivities greater than 2 μV/mm are required.

Corrosion or loosening of contacts in switches or wires may cause abrupt changes in voltage or sudden loss of signal. The nonphysiologic nature of such potentials is usually readily apparent, but finding the source in the instrument may be difficult, especially if the artifact is intermittent. Again these issues are rare in digital polygraphs.

A meticulous, alert, and experienced technician is the first and best defense against artifact. The critical importance of properly applied and gelled electrodes cannot be overemphasized for PSG, because adjusting or changing electrodes usually means waking the patient. The technician should be on the lookout for bizarre potentials and seek to determine whether these are physiologic or artifactual. Observation of the patient and environment, correlations with the recorded activity, and careful documentation are critical. The technician must then decide whether the artifact significantly interferes with recording of the signal of interest. Deciding whether to change an electrode and possibly wake the patient, or allow a partially interpretable recording to continue, requires seasoned judgment. Technicians should be aware of the major issues involved in interpreting a PSG so they can make these on-the-spot decisions wisely.

* Positive rolandic sharp waves are occasionally recorded in premature infants with intraventricular hemorrhage or periventricular leukomalacia. Otherwise, positive sharp waves are very uncommon in older patients.

CONCLUSION

The interpretation of a PSG can be considered a pattern-recognition task. The EEG is the most complicated and variable recording the polysomnographer interprets. Several issues continuously preoccupy the polysomnographer when interpreting the EEG. One question is whether the recorded signal is a true cerebral potential or whether it represents artifact. Another is whether the signal is present throughout the scalp or whether it is confined to a single region of the scalp. The use of multiple channels for EEG recording allows the polysomnographer to answer these questions with greater certainty. Unfortunately, even in today's digital environment, EEG recorded during routine PSG is often limited to a few channels. Limited montages and slow paper speeds often do not allow confident interpretation of unusual activity. This is especially unfortunate because EEG abnormalities important to the patient's care are more likely to occur during the longer PSG recordings than during routine EEG. The cost of a few additional EEG channels is more than repaid by the greater certainty in interpretation and the greater likelihood that important abnormalities will be found. Sometimes a confident decision regarding the nature of suspicious potentials cannot be made, even when several EEG channels are available. It is important not to overinterpret suspicious events. The polysomnographer should not be afraid to admit uncertainty in a situation in which data are insufficient. Referral for routine sleep EEG is usually appropriate in these circumstances. The full complement of scalp EEG channels often provides the necessary information. Similarly, information from additional EMG and EOG channels often clarify ambiguities. Equivocal changes are often interpreted more confidently when more data are available.

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