What Every Clinician Should Know About Polysomnography

Susheel P Patil MD PhD

Polysomnography studies are an essential tool for the sleep physician and aid in the diagnosis and treatment of sleep disorders. Polysomnography refers to the recording, analysis, and interpretation of multiple physiologic signals collected simultaneously. Rapid advancements in technology have transformed the field from a time when analog studies were collected on paper to computer-assisted collection of digitally transformed studies. Sleep clinicians, whether physicians, respiratory therapists, or sleep technologists, must therefore have an understanding of a broad array of principles underlying the collection of the various signals. In addition, an understanding of basic technical rules in the evaluation of polysomnography studies is necessary for both the scoring and interpretation of such studies. The American Academy of Sleep Medicine published a new manual for the scoring of sleep and associated events in 2007. These changes included modifications to the visual scoring of sleep, the scoring of sleep-disordered breathing events, and movement disorders during sleep. A few early studies have evaluated the effects of the changes in scoring guidelines to the previous Rechtschaffen and Kales (R&K) rules for sleep and the American Academy of Sleep

Medicine rules for respiratory events. Some controversy regarding the scoring of respiratory events continues to exist and requires further studies to be performed. *Key words: polysomnography; AASM scoring manual; filters; sampling rate; amplifiers; aliasing; movement disorders; sleep staging; obstructive sleep apnea; arousals*. [Respir Care 2010;55(9):1179–1193. © 2010 Daedalus Enterprises]

Introduction

Polysomnography studies are an essential tool for the sleep physician and aid in the diagnosis and treatment of sleep disorders.[1] The term polysomnography[2,3] refers to the recording, analysis, and interpretation of multiple physiologic signals collected simultaneously. Typical physiologic signals that are collected as part of the polysomnography study include, but are not limited to, electroencephalogram (EEG), electromyogram (EMG), electro-oculogram (EOG), electrocardiogram (ECG), and respiratory signals. Rapid advancements in technology have transformed the field from a time when analog studies were collected on paper to computer-assisted collection of digitally transformed studies. Sleep clinicians, whether physicians, respiratory therapists, or sleep technologists, must therefore have an understanding of a broad array of principles underlying the collection of the various signals. In addition, an understanding of basic technical rules in the evaluation of polysomnography studies is necessary for both the scoring and interpretation of such studies.

This paper describes basic electrical concepts, the operation of differential amplifiers, the use of filters and filtering, aspects unique to digital polysomnography, common artifacts encountered during recordings, and recent changes in technical rules for the scoring of polysomnography studies that were established by the American Academy of Sleep Medicine (AASM) in 2007.[4]

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Electrical Concepts

The physiologic signals collected during a polysomnography study are representations of bio-electrical potentials (ie, electrical activity) from different sources within the human body.[5-7] It is worthwhile to take a moment to discuss basic electrical concepts that govern the movement of current and the properties that affect it. An understanding of these concepts provides a foundation for understanding the function of amplifiers.

Current, Voltage, Resistance

Electrical current (*I*, measured in amperes) refers to the movement of charged particles through conductive material.[5] Particles of similar charge will distribute uniformly within a conductive material, but will not result in the net movement of the charged particles (ie, a current, through the material). An electrical current is produced when a net movement of charge occurs across the conductive material. Electrical current can be direct (*DC*) or alternating (*AC*). DC refers to the net movement of charged particles within a circuit in one direction. AC refers to an oscillatory current that changes polarity and direction periodically. The number of oscillations or cycles per second is referred to as the *frequency* (*f*, measured in *Hz*).

The net movement of charge is generally induced by an object, such as a battery, that can generate an *electromotive force*. The electromotive force is quantified as the potential difference in charge between 2 terminals within an electromotive-force device, and is represented as *voltage* (*V*, measured in *volts*).

All materials have a property called *resistance* (*R*, measured in *ohms*), which restricts the flow of current through the material. Resistance is inversely related to conductance. The mathematical relationship between current, voltage, and resistance are provided by Ohm's law:

$$V = I \times R$$

Capacitance, Inductance, and Impedance

Ohm's law, as stated above, can be directly applied to DC circuits. In AC circuits, however, the concept of resistance is more complicated and requires an understanding of 2 additional electrical concepts: *capacitance* and *inductance*. A capacitor is an electrical device within a

circuit that stores energy between 2 plates and resists changes in voltage. Capacitors are useful in the design of low-frequency and high-frequency filters. Capacitance (*C*, measured in *farads*) is a measure of the charge stored for a given voltage. The application of an AC current to a capacitor results in the development of resistance to the current, which is referred to as *capacitive reactance* (X_C).

Inductors are another electrical component that are used in circuits, and are formed by coils of conductive material (eg, copper wire) around a core of ferromagnetic material (eg, iron). Energy is stored within the inductor as an electromagnetic field, which opposes a change in current. Inductors are also useful in the design of electrical filters. Inductance (*L*, measured in *henries*) is the amount of voltage generated per change in current rate of one ampere per second. The resistance to an alternating current by an inductor is referred to as *inductive reactance* (X_L).

Resistance within an AC circuit, referred to as *impedance* (*Z*, measured in ohms), is calculated from the resistance of the circuit, the capacitive reactance, and the inductive reactance. Once determined, impedance can replace resistance in Ohm's law for the analyses of AC circuits and would take the following form:

$$V = I \times Z$$

Knowledge of impedance has important applications in polysomnography. Specifically, low impedance will result in a high-quality physiologic signal through improvements in the signal-to-noise ratio. Conversely, high impedance will result in a low-quality signal through reductions in the signal-to-noise ratio. Impedances under 5 KΩ are ideal to optimize the collection of AC signals.

Differential Amplifiers

Amplifiers are used in polysomnography recordings for differential discrimination and amplification.[8] *Differential discrimination* refers to the ability of the amplifier to reveal differences in potentials between 2 inputs and reject potentials that are common to the 2 inputs. *Amplification* refers to the ability of the amplifier to increase the size of the potential differences and drive the analog-to-digital converters.

The purpose of differential discrimination is to minimize electrical interference from other electrical sources within the body, the environment within which the recording is collected, and other equipment in the local environment. This is performed by relating 2 inputs into the amplifier to each other and results in a single output voltage that is then displayed (Fig. 1).

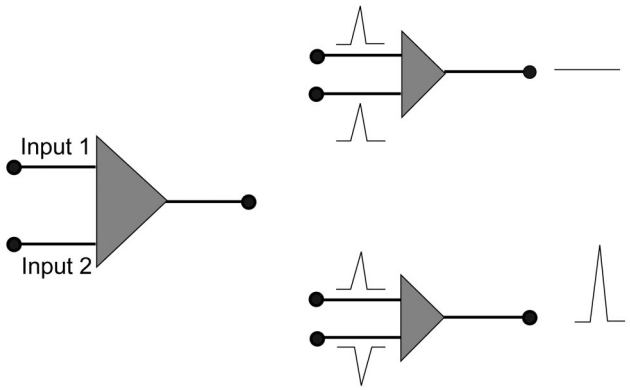

Fig. 1. Basic operation of differential amplifiers. Two inputs come in to an amplifier, and there is one signal output after signal conditioning. If the 2 inputs are of the same polarity and amplitude (upper right), the output signal is zero (common mode rejection of the potentials). If the 2 inputs are of different polarities but the same amplitude (lower right), the output signal is twice the amplitude of the input signals (amplification of different potentials). (From Reference 1, with permission).

Polarity

The *polarity* (ie, direction of displayed deflection) of the output signal is dependent on the polarity of the 2 inputs into the amplifier, and on the input terminal to which it is applied (input 1 vs input 2).[8] In other words, when 2 electrical signals are sent to an amplifier, greater negativity at input 1 with respect to input 2 results in an upward deflection. In contrast, greater negativity at input 2 relative to input 1 results in a downward deflection. By convention, potentials with *negative* polarity are presented as an *upward deflection*, while potentials with a *positive* polarity are presented as a *downward deflection*. If the 2 inputs into the amplifier are of similar polarity and amplitude, the output signal from the amplifier will zero (rejection of the potentials, see Fig. 1). If the 2 inputs into the amplifier are of different polarities but the same amplitude, the output signal from the amplifier will be twice the amplitude of the input signals (amplification of different potentials, see Fig. 1). The examples above represent the most simplistic representation of the amplifier output. With experience it becomes more evident that a particular signal output can be the result of an infinite number of input signals with differences in polarity, amplitude, and timing.

Common Mode Rejection

Biopotentials are not equally distributed over the source of interest (eg, brain); however, electrical interference typically affects all areas over the source of interest. The purpose of differential amplification is to eliminate the electrical interference at the inputs in order to detect the biopotential of interest that is localized to the area of in-

terest. The ability to discriminate between like potentials and differing potentials is also referred to as *common mode rejection*. This ability is quantified as the *common mode rejection ratio* and is the ratio of the common mode input voltage and the output voltage. The higher the ratio, the greater the ability of the amplifier to reject the common voltages at the 2 inputs. Most high-quality polysomnography systems have a common mode rejection ratio of 10,000:1. Although the ability of the amplifier to reject common voltages is greater in devices with high ratios, this does not always guarantee the elimination of common voltages, and could make distinguishing biopotentials from electrical interference difficult. *Failure of the amplifier to discriminate is typically due either to a loss of an effective ground connection or to unequal impedances between the 2 inputs*.[8] A loose connection from a ground electrode or an input electrode can result in electrical interference that is difficult to distinguish from the true biopotentials at the input electrode. Unequal impedance between electrodes typically occurs when there is a loss of contact. As a result of unequal impedance, biopotentials of equal amplitude at the scalp will appear with different amplitudes at the amplifier input. A common result of unequal impedance is the appearance of a 60-Hz artifact. The absence of a ground or an ineffective ground results in the inputs "floating" without reference to the potential level at which the amplifier references input 1 to input 2.[7]

Amplifier Gain, Sensitivity, and Type

Differential amplifiers amplify the difference in voltage between the input signals in order to drive an analog-to-digital converter. This amplification factor is also called *gain*. For example, if an amplifier is set to provide an output voltage of 1 V for a 1 mV input, the gain would be 1,000. Gain is sometimes expressed in *decibels* [dB = 20 × log(gain); in our example this would be 60 dB]. Amplifier *sensitivity* refers to the ratio of the input voltage to the vertical size of the waveform produced, and is often expressed in μV/mm. As sensitivity increases, the vertical size of the waveform decreases. As sensitivity decreases, the vertical size of the waveform increases.

Amplifiers that are used in polysomnography equipment include both AC and DC amplifiers. An AC amplifier can amplify only AC signals, whereas a DC amplifier can amplify both AC and DC signals.[5] AC amplifiers are typically used to record high-frequency signals such as EEG and EMG. AC amplifiers have controls to set the sensitivity, polarity, and filters, in order to condition the signal. Low filters and high filters are available to isolate the bandwidth and biopotentials of interest (see Filters, below). A notch filter is often provided to assist in the elimination of 60-Hz artifact if needed. DC amplifiers generally have a setting to alter the reference baseline of the signal recorded and have adjustments for sensitivity and filtering as well. Because DC amplifiers are used for recording slow-frequency signals (eg, respiration, pressure, oximetry, respiratory effort), a low filter is not included, although a high-frequency filter is present.

Patient Ground, Electrical Ground, and Electrical Safety

It is important to distinguish between the patient ground and the electrical ground when considering the issue of electrical safety to the patient and the technologist in the sleep laboratory.[6,8] The purpose of the patient ground is only to reduce the presence of electrical interference and improve common mode rejection. It does nothing to protect the patient or the technologist. A common location for the patient ground is the head, typically at the frontopolar (Fpz) point, located at 10% of the nasion-inion distance, above the nasion in the midline, based on the 10–20 system. The frontopolar point is best suited to be the patient ground because it generally lacks true bipotential sources from the brain and can aid in the detection of unequal impedance because of the lead's proximity to the eyes.[7]

Electrical injury can occur only if an individual is connected to an electrical apparatus. Injuries can include burns, seizures, arrhythmias, and permanent damage to nervous tissue. Dry, intact skin has high impedance, which can protect the patient from current flow. Wet skin, wounds, and pacemaker wires have low impedance and can make the patient susceptible to electrical injury from current flow. A 0.5–1 mA current at 60 Hz is near the threshold of pain perception. A current of 100–300 mA (macroshock) at 60 Hz is enough to induce fibrillation.[6] For current to flow requires a source of current and a complete circuit.[6,8] A circuit is designed so that the patient does not form a complete circuit through which excessive current can flow and cause injury. The power unit to the polysomnography equipment represents one source. If there is a short circuit within the electrical power source, the fault current flows to the chassis. If the patient or technologist is touching a conductor and the ground is disabled, excessive current will be received by the individual and electrical injury could occur. To prevent electrical injury the PSG equipment has an electrical ground at the source of power. One of the prongs represents a low-resistance circuit, which allows the earth to act as a sink in case of a short circuit.

Filters and Filtering

Filters are used to exclude high and low frequencies so that biopotentials in the range of interest are more clearly recorded without distortion. *Low-frequency filters* (also known as high-pass filters) are used to attenuate the amplitude of slow-frequency waveforms. *High-frequency fil-*

Table 1. Recommended Sampling Rates and Filter Settings*

Signal	Sampling Rate (Hz)		Low-Frequency Filter (Hz)	High-Frequency Filter (Hz)
	Desirable	Minimal		
Electroencephalogram (EEG)	500	200	0.3	35
Electro-oculogram (EOG)	500	200	0.3	35
Electro-myogram (EMG)	500	200	10.0	100
Electrocardiogram (ECG)	500	200	0.3	70
Air flow	100	25	0.1	15
Oximetry	25	10		
Nasal pressure	100	25	0.01 or direct current (DC)	15 (100 for snoring)
Esophageal pressure	100	25		
Body position	1	1		
Snoring sounds	500	200	10.0	100
Rib cage and abdominal movements	100	25		

* Maximum electrode impedance, 5 kohms; minimal digital resolution of amplifiers, 12 bits per sample.
(Adapted from Reference 1, with permission. Additional specifications available in Reference 4.)

ters (also called low-pass filters) are used to attenuate the amplitude of high-frequency waveforms. *Notch filters* are used to eliminate frequencies due to electrical interference, typically centered at 60 Hz. The most important concept to bear in mind is that filters are not absolute in their elimination of particular frequencies. Filters provide a continuum of gradual filtering, both below and above the specified frequency cutoff. Newer digital systems have a more narrowly applied filter than older analog systems. Table 1 lists commonly recommended filter settings from the recent AASM Manual for the Scoring of Sleep and Associated Events.[4]

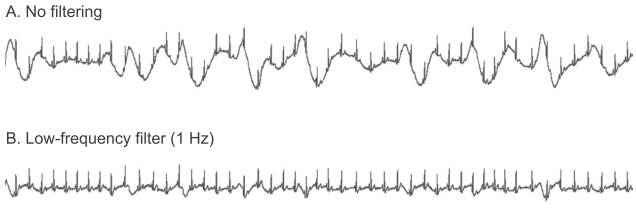

Fig. 2. Low-frequency filtering. A: A slow oscillatory frequency in the electrocardiogram signal, which may be consistent with respiratory artifact or sweat artifact. B: A 1 Hz low-frequency filter is applied and most of the signal's slow-frequency component is eliminated.

Low-Frequency Filters

Low-frequency filters are designed to attenuate slow-wave-frequency waveforms not of physiologic interest, such as galvanic skin responses, DC electrode imbalance, and respiratory artifact (Fig. 2).[2,8,9] The setting of a low-frequency filter specifies the *cutoff frequency* at which the amplitude of that frequency is reduced by a set percentage, and is typically 20–30%, depending on the system used. Regardless of the cutoff frequency that is selected, that particular frequency will be attenuated by 20–30%. Attenuation of frequencies below the cutoff frequency become more severe as the frequency progressively gets slower, and is also known as *roll-off*.

Another important concept in filtering is the *fall time constant* (typically referred to as the time constant). The time constant is the time required for a square wave voltage applied to an amplifier to decay to 63% of its peak amplitude, and describes the effects of a low-frequency filter on the square wave pulse (Fig. 3).[8] In electrical terms, the time constant is equal to the resistance times the capacitance ($TC = R \times C$). The low-frequency filter setting and the time constant are inversely related. The higher the low-frequency filter, the shorter the time constant. The lower the low-frequency filter, the longer the time constant. By setting a time constant for an amplifier at a longer time, slower frequency waveforms are amplified without significant filtering. In older analog systems, knowledge of the time constant was important because certain amplifiers did not have a low-frequency filter and only had the ability to set the time constant.

High-Frequency Filters

High-frequency filters are designed to attenuate fast-frequency waveforms (Fig. 4).[8] The setting of a high-frequency filters is similar to low filters, whereby a *cutoff frequency* is specified above which the amplitude of that frequency is reduced. In contrast to low-frequency filters,

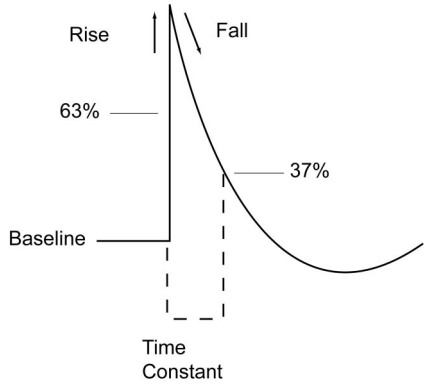

Fig. 3. Time constant. The fall time constant refers to the time required for the current from a square wave voltage to decay to 65% of its peak amplitude. Time constant = resistance × capacitance). The low-frequency-filter setting α is 1/time constant. The shorter the time constant the higher the low-frequency-filter setting. The longer the time constant the lower the low-frequency-filter setting.

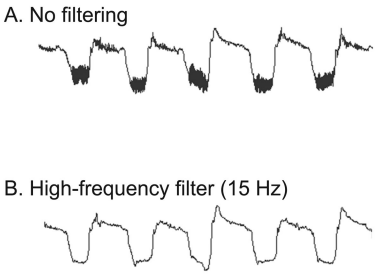

Fig. 4. High-frequency filtering. A: A high-frequency signal that is particularly prominent during inspiration represents snoring artifact. B: A 15 Hz high-frequency filter is applied and the signal's high-frequency component is eliminated.

many digital systems have a steeper roll-off for the high-frequency filters. This allows analog filtering prior to digitization and reduces demand on the sampling rate of the analog-to-digital converter.

Notch Filters

Notch filters are designed to eliminate electrical interference generated by other devices using alternating current (Fig. 5).[5,8] In the United States this is typically at 60 Hz, whereas in other countries the frequency of AC current may be 50 Hz. Notch filters severely attenuate (ie, high roll-off) frequencies centered at 60 Hz, but may not completely filter them out. Notch filters should not be used routinely, because the presence of a 60-Hz artifact can represent a useful warning sign to the astute technologist. The appearance of a 60-Hz artifact should warn the technologist of the possibility of unequal impedance between electrodes (eg, loss of electrode contact or sweating) or

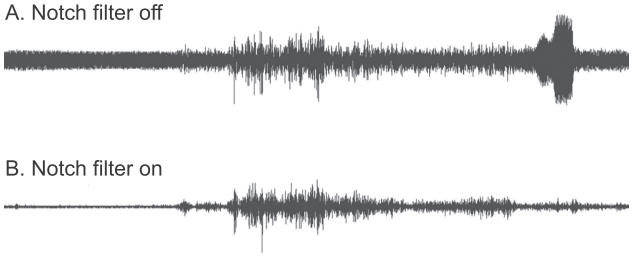

Fig. 5. Notch filtering for 60-Hz artifact. A: A 60-Hz artifact in the submental electromyogram is particularly notable in the first third of the signal. Ideally the technologist should try to replace the sensor to see if the artifact resolves. B: The notch filter appears to significantly attenuate the 60-Hz artifact.

incorrect input selection. The technologist would be better served to correct the etiology of the problem rather than to use the notch filter. The notch filter should be used only after this troubleshooting has occurred, since application of the filter can obscure relevant physiologic electrical phenomenon such as spike activity.

Aspects Unique to Digital Polysomnography

Rapid advancements in technology have led to computer-assisted collection of polysomnography recordings.[8,10] Digital polysomnography refers to the collection, digitization, and display of data.[10] The advantages of digital polysomnography include flexibility in time-scale display of recorded data, auto-correction of amplifier gains, self-diagnostic tests of amplifier functions, software-controlled in-line impedance testing, easy storage of data, the ability to move between different time points within a recording, and the application of various digital filters while keeping original digitized signal, to name a few.[2,6] Disadvantages of digital polysomnography include a lower display resolution than paper, signal distortion due to aliasing, the need for a more attentive technologist, and computer-related problems. There are several features unique to digitization of data, compared with the analog collection and display of the data, that the technologist and sleep physician should be cognizant of. In particular the conversion of an analog signal to a digital signal is dependent on several key components: the sampling rate, the bits of resolution available, and the input voltage range.

Sampling Rate and Aliasing

The *sampling rate* is the frequency at which an analog signal is converted to a digital signal.[8] Table 1 lists commonly recommended sampling rates by signal type.[4] For example, a sampling rate of 100 Hz implies that the analog

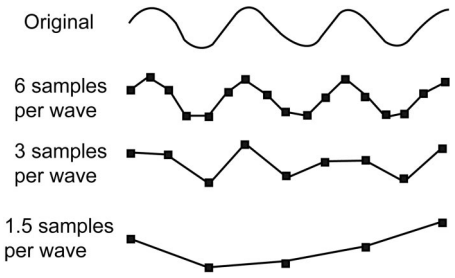

Fig. 6. Aliasing. A signal frequency is misrepresented as a slower-frequency waveform. This is typically due to under-sampling. Progressively lower-frequency sampling results in a loss of fidelity of the original waveform.

signal has been sampled every 0.01 seconds, resulting in the digitized signal having 100 samples per second of the analog signal. To adequately represent an analog signal in a digitized form, it must be appropriately sampled. According to *Shannon's Sampling Theorem* (also known as *Nyquist's Theorem*), a signal can be digitized and restored to accurately represent the frequency of the analog signal if sampled at twice the highest frequency (known as the *Nyquist* rate) contained within the signal. For a digital signal to reproduce the quality of an analog signal would require a sampling rate at least 6 times faster than the fastest frequency to be visualized. If a signal is sampled below the Nyquist rate, aliasing can occur.

Aliasing is the misrepresentation of a signal as a slower-frequency waveform. For example if a 100 Hz signal is sampled at 100 Hz, only 1 data point per second at the same amplitude would be represented after the analog-to-digital conversion process (Fig. 6). Each point would be connected by a line and would be interpreted to represent a 0 Hz signal. As the sampling rate is increased above 100 Hz, a digital waveform slower than 100 Hz is created (still a false representation of the frequency of the original signal) until a sampling rate of at least 200 Hz is achieved. Aliasing can be avoided by either: (1) increasing the sampling rate, or (2) filtering the analog signal prior to digitization to remove activity with a faster frequency than half of the analog-to-digital converter sampling rate.[10] Aliasing is difficult to recognize when it occurs, so appropriate sampling and filtering are important safeguards in the digital collection of polysomnography data.

Bit Resolution and Input Voltage

The vertical resolution of a signal is dependent on the number of binary bits used to represent the digital values.[8] The number of *bits* used to represent a sampled analog signal is known as the resolution of the analog-to-digital converter. The amplitude of a signal is assigned to discrete, non-overlapping amplitude levels that are defined by the number of bits available in the digital recording equipment. For example, if a system has 12 bits, this would represent 2^{12} or 4,096 levels ($\pm$ 2048 levels) for the voltage range of the signal to be represented. Since most amplifiers allow an input voltage range of $\pm$ 5 v, this would mean that each level could discriminate between a data point of no less than 2.4 mV (10 V/4,096 levels). In other words, changes in voltages smaller than 2.4 mV would not be reflected as a change in amplitude. Changes in voltage greater than 2.4 mV would be reflected as a change in amplitude and would be reflected as a smoother signal that represented a higher-fidelity reproduction of the analog signal. For polysomnographic recording systems a bit resolution of 10–12 bits is desirable. Fewer bits would result in large voltage changes being undetected and small voltage changes being overrepresented.

Monitor Aliasing

It is important to also be aware of the potential for *monitor aliasing*. Monitor aliasing refers to aliasing of a signal that occurs due to limitations of the monitor display. A digital representation of a signal over a 30-second epoch rarely has the fidelity of the same signal over the same epoch seen with paper recordings, because of the limitations of pixel resolutions on computer screens. A minimum computer monitor resolution of 1600 $\times$ 1200 pixels is recommended. A 30-second epoch represented over the horizontal span of 1600 pixels would result in an effective sampling rate of 53 Hz. As a result, the displayed signal may appear distorted. As the time scale is decreased to small epochs, the resolution of the signal will improve. Nevertheless, the display of a digital system rarely rivals that seen in the traditional paper recording.

Artifacts

Despite careful attention to the preparation of the patient and the recording equipment, the presence of high-quality differential amplifiers with high common mode rejection ratios, and the use of filters, extraneous electrical activity can appear in a recording and are called *artifacts*.[6,11] The recognition and correction of artifacts is central to the duties of the recording technologist and the supervising sleep physician. A complete discussion of all artifacts that one can encounter is beyond the scope of this paper, but Table 2 summarizes common artifacts, their sources, and approaches to troubleshooting.

Troubleshooting of any artifact begins with the patient and the recording. The technologist should follow the signal path from the patient to the recording device.[2] Very often an artifact can be localized to a set of leads. In this situation the technologist should identify the leads in which the artifact is seen in order to identify the common lead and localize the source of the artifact.

Table 2. Common Artifacts, Causes, and Strategies to Address

Artifact	Appearance	Cause	Strategies to Address
Muscle artifact	High-frequency artifact (> 25 Hz) typically seen in EEG (electroencephalogram) and EOG electro-oculogram) channels; difficult to distinguish from activity in beta frequency range	Increased muscle tension	Will generally resolve with sleep onset, due to muscle relaxation
Movement artifact	Sudden, brief bursts of high-amplitude, high-frequency; can also appear as slow-frequency movements when isolated to the head	Spurious static potentials caused by body movement; slower movements may be due to slight movement of electrodes on scalp or swaying of wires (eg, head movement)	No intervention; position wires away from contact with the head or body
Sweat artifact	Slow-frequency waveforms seen in EEG and EOG channels can appear asymmetric	Changes in salt content of sweat can alter the ionic composition of the conducting gel	Cool the room for the patient. Consider repositioning electrode.
Pulse artifact	Small-amplitude, low-frequency waveform that corresponds to the pulse rate	Pulsations of nearby artery resulting in electrode movement	Reposition electrode
ECG artifact	Appearance of QRS-like waveform corresponding with the ECG signal	Detection of electrical current from the myocardium	Reposition the ECG electrodes higher up on the chest or to the upper back, close to the shoulders. Reposition the reference leads (ie, M1 or M2). Use a jumper cable between M1 and M2 to increase common signal within the head electrodes, to increase likelihood of common mode rejection.
Respiratory artifact	Slow-frequency waveform seen in high-frequency channels that corresponds to the frequency of respiration	Most often due to an electrode wire in contact with the chest and abdomen	Reposition affected electrode wires.
60 Hz artifact	Moderate frequency, highly regular	Poor electrode contact; defective lead wires; inadequate grounding; nearby electrical equipment	Reapply or replace electrode. Reapply or replace the ground. Turn off unnecessary electrical equipment. Increase shielding.
Electrode popping	Abrupt vertical transients, typically of positive polarity confined to single electrode	Due to abrupt changes in impedance	Reapply electrode more firmly. Reapply conducting gel to improve conductance. Replace lead.
Reversed polarity	Change in polarity of waveform	Accidental inverting of waveform; movement of sensor (eg, migration of an abdominal effort gauge more superiorly)	Adjust sensor if possible. Review biocalibrations to determine if inverting of signal was accidental.

(Adapted from Reference 1, with permission.)

When artifacts are more generalized, this may reflect a poor patient ground electrode or loose equipment connection. Artifacts due to amplifier problems are more unusual.

Updates in Polysomnography Scoring

The scoring of sleep studies has evolved to include various criteria that assess sleep, arousals, respiratory events, and movement-related events in multiple publications. In 2004 the AASM established a task force to develop a scoring manual that integrated all criteria for the scoring of sleep studies, with the purpose of providing standards for the collection of sleep studies, performing an evidence-based review and updating scoring guidelines as deemed necessary, and improving the reliability of sleep study scoring between technologists and laboratories. A steering committee supervised the work of 8 task forces assigned to consider major areas, including visual scoring, digital scoring, arousals, movement, respiration, cardiac rhythm, pediatric issues, and geriatric issues.[12-19] Industry input was obtained regarding software and hardware technical requirements. The AASM Manual was then approved by the Board of Directors and published in 2007.[4]

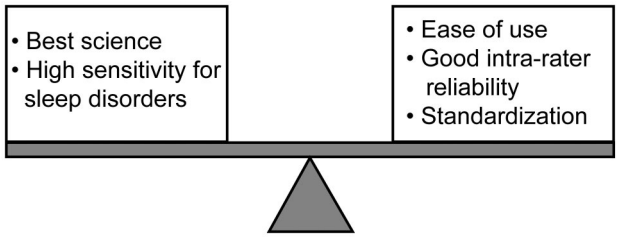

Fig. 7. Balancing concerns in the development of the American Academy of Sleep Medicine Manual for the Scoring of Sleep and Associated Events.

The final AASM Manual has endured some controversy.[20-22] For example, the Italian Association of Sleep Medicine noted that of the 29 rules established for the visual scoring of sleep, only 3 had the sufficient evidence base to be considered as a "standard recommendation," with the vast majority of rules established by "consensus."[21] Concerns were raised regarding the continued static assessment of sleep dynamics with fixed epochs, limited comparability of sleep assessment under the new rules and previously scored studies using the Rechtschaffen and Kales (R&K) rules,[23] the use of arousals to define sleep-stage transitions, and competing definitions for respiratory events, to name a few.[21,22] Task force members had to consider the balance between standardization of collection and scoring, reliability, and ease of use against the best science and parameters that were highly sensitive for the detection of sleep disorders (Fig. 7). A process was put in place to periodically review the evidence base behind the established rules and to amend the manual when deemed necessary. What follows is a brief review of major changes to previous guidelines that clinicians should be aware of. For more details, particularly related to changes in pediatric scoring, readers are referred to other publications.[4,12-20]

Visual Rules for Sleep

Previously under the R&K rules established in 1968 for the scoring of sleep,[23] only one central electroencephalogram (EEG) derivation (eg, C4-M1) was required for the assessment of EEG. Since then the addition of other EEG derivations, including occipital (O2-M1) and frontal derivations (F4-M1), have been recognized to provide improved identification of sleep stages and wakefulness.[24,25]

Changes in the Scoring of Sleep Under the AASM Manual

Several important changes regarding the scoring of sleep were implemented within the AASM Manual.[13] First, as shown in Table 3, the nomenclature for sleep stages was changed. Most importantly, slow-wave sleep (previously stage 3 and 4 sleep) was consolidated into stage N3 and required the use of frontal EEG leads to maximize detection of slow-wave activity, with peak amplitudes $> 75\ \mu V$. The visual scoring task force chose to consolidate stages 3 and 4 sleep into one stage because of a lack of evidence that the distinction was of biological significance.[26] Second, movement time scoring was eliminated because of consensus that such movements typically result in a transition to wakefulness. Third, the onset of sleep was more liberally defined. The AASM Manual defined sleep onset as the occurrence of any stage of sleep (stage N1-N3 or stage R), whereas previously 3 consecutive epochs of stage N1 or one epoch of stage N2, N3, or R was necessary. Fourth, the 3-min rule under the R&K guidelines was eliminated because of the lack of an evidence base. Under this rule, stage N2 reverted to stage N1 after 3 min if a K-complex or spindle, respectively, were not observed. Under the new rules, a sleep stage is maintained until intervening events occur to score an awakening or another sleep stage. Fifth, the presence of an arousal results in a transition to stage N1 with the epoch scored as the predominant stage of sleep that occurs within the epoch. Finally, the scoring of arousals in REM sleep requires only a transient increase in submental EMG $>$ 1 second duration in association with an abrupt shift of EEG frequency. Previously, a submental EMG increase $>$ 3 seconds duration with an abrupt shift in EEG frequency was required.

Table 3. Sleep Staging Nomenclature

Rechtschaffen and Kales (R&K) Rules	American Academy of Sleep Medicine Manual
Waking	Stage W (wakefulness)
Stage 1	Stage N1 (NREM 1)
Stage 2	Stage N2 (NREM 2)
Stage 3	Stage N3 (NREM 3)
Stage 4	
Stage REM	Stage R (REM)

NREM = non-rapid-eye-movement sleep

Comparison of the AASM Manual and R&K Sleep Staging Rules

Recently, an attempt was made to compare the sleep scoring parameters between the AASM Manual and the R&K rules.[27] Those authors utilized a database of sleep studies from 72 subjects used in the SIESTA project, an automated algorithm for sleep study analyses.[28] The subject sample consisted of 56 healthy subjects and 16 subjects with medical issues (generalized anxiety disorder, periodic limb movement disorder, and Parkinson disease),

with an age range of 21–86 years (mean age 58 ± 19 y), and roughly even distribution by sex (38 females). Each subject had 2 consecutive sleep study nights, to eliminate the first-night effect. The second sleep study night was used for analyses. Under each of the scoring rule guidelines, each study was scored by 2 independent scorers; however, the authors presented analyses comparing the first scorings by the AASM Manual or R&K rules. They identified several statistically significant differences in several sleep scoring parameters between the 2 guidelines, but of unclear clinical importance. Specifically, compared to the R&K rules, the AASM Manual rules identified increased wake after sleep onset (WASO) (+4.1 min), increased stage N1 (+10.6 min, +2.8%), decreased stage N2 (−20.5 min, −4.9%), and increased stage N3 (+9.1 min, +2.4%). Total sleep time was preserved, with a difference of only −2.5 min between the 2 scoring systems. The effects of age (< 60 y vs ≥ 60 y) were also examined. Only stage R time was significantly shorter, in the younger subjects, with the AASM rules (−5.8 min, −1.3%). Sleep scoring parameters were similar by sex.

The authors provided potential explanations for some of the differences identified. In the case of wake after sleep onset, the increase appeared to be due to the elimination of movement time and the change in the definition of sleep onset (ie, an epoch of stage N1-N2 or stage R defines sleep onset). Increases in stage N1 were attributed to the new AASM Manual's rule that sleep transitions to stage N1 after an arousal. Increases in stage N3 were attributed to the addition of frontal derivations of the EEG, which appear to be more sensitive to the detection of high amplitude slow-wave activity.[24] Finally, reductions in rapid-eye-movement (REM) time in young individuals were due to the increase scoring of non-REM stages. They concluded that the differences identified, although generally small, may have important implications for current normative data for sleep scoring across the lifespan. Since previous normative data were obtained using the R&K rules, it may be necessary to develop a new body of normative data using the new AASM rules.

Scoring Reliability of Sleep Staging Under the AASM Manual

A stated purpose of the AASM Manual was to improve scoring reliability between scorers and centers. The SIESTA group also compared inter-rater reliability of sleep-related parameters between the AASM Manual and R&K rules in another publication, using the same study sample.[29] In general, the AASM rules demonstrated marginally better intraclass correlations between scorers than did the R&K rules, with several notable exceptions. The intraclass correlation was markedly higher between scorers for stage N1 (0.74 vs 0.40) and stage N3 (0.73 vs 0.62) with the AASM rules. In contrast, the correlation between scorers was rather low for stage N2 (0.40 and 0.34) for both the AASM and R&K rules. The deviation in scoring was primarily attributed to decisions related to stages N1 versus N2, N1 versus wake, and N3 versus N2, and accounted for 77.7% and 80.9% of the scoring discrepancies with the AASM rules and R&K rules, respectively. The authors also compared the Cohen's kappa (a measure of agreement that accounts for agreement due to chance) of the 72 studies, and reported a slightly higher Cohen's kappa with the AASM rules (median 0.78, interquartile range 0.70–0.83), compared to the R&K rules (median 0.74, interquartile range 0.66–0.80), and that difference was statistically significant. Sleep-stage-specific analyses demonstrated that stage N1, stage R, and wakefulness demonstrated a statistically greater agreement with the AASM Manual rules, compared to the R&K rules. A greater proportion of AASM Manual scored studies were also found to have perfect agreement, compared to the R&K rules (42.3% vs 22.5%), when using the criteria of Landis and Koch (kappa > 0.8 is perfect). Finally, greater age correlated with reduced agreement. The results of these analyses suggested that the AASM Manual and the R&K rules demonstrate similar agreement, if not better for certain sleep parameters (eg, stage N1) under the AASM Manual rules. However, further investigation is needed to assess methods to improve agreement between scorers for other parameters (eg, stage N2).

Scoring of Arousals

The AASM Manual retained the previously established rules for scoring of arousals from sleep established by the American Sleep Disorders Association (ASDA) in 1992,[30] with one important modification. In the original ASDA rules, an arousal during stage R could be scored if there was: (1) an abrupt shift of EEG frequency (alpha, theta, and/or beta frequencies that are at least 3 seconds, with at least 10 seconds of stable sleep preceding), and (2) a concurrent increase in submental EMG of at least 3 seconds. Under the new AASM Manual rules, a burst in submental EMG of only 1 second in association with an abrupt shift in EEG frequency is necessary to score an arousal during stage R.

In the evidence-based review accompanying the AASM Manual, the task force on arousals reviewed the literature regarding the reliability of ASDA arousal scoring. The task force reported that reliability of arousal scoring could be high (intraclass correlation of 0.84) and could be improved with the addition of an increase in EMG (from 0.84 to 0.92) in some studies[31] but not others.[32] Factors found to affect the reliability of scoring of arousals included the expertise of the scorers, the stage of sleep during which arousals are scored (scoring of arousals during stage N3 is

most reliable), and contextual scoring (ie, scoring of arousals in the presence of a visible respiratory channel). For example, one study demonstrated that removal of the respiratory signal resulted in a decrease in agreement of arousals between scorers from 91% down to 59%.[33]

Movement Rules

For the first time, rules for the assessment of common sleep-related movement disorders were brought together under the AASM Manual. The Manual provides scoring rules for periodic limb movements during sleep, REM behavior disorder (RBD), alternating leg muscle activation (ALMA), hypnogogic foot tremor, excessive fragmentary myoclonus, and bruxism.

For the assessment of periodic limb movements during sleep, the AASM Manual adopted the guidelines established by the Periodic Limb Movements Task Force of the International Restless Legs Syndrome Study Group.[34] Several changes to the scoring of leg movements have been made, when compared to the guidelines established by the ASDA in 1993.[35] First, the maximum duration of a leg-movement event can be as long as 10 seconds (previously as long as 5 seconds). Second, specific criteria for the onset and offset of a leg movement have been provided. A leg-movement event occurs when the amplitude of the EMG rises by 8 μV above the resting EMG. A leg movement ends when the EMG amplitude is less than 2 μV above the resting EMG for at least 0.5 seconds. Third, the guidelines for scoring of arousals associated with leg movements have changed. Previously, an arousal after a leg movement was considered movement-related if it occurred within 3 seconds after the leg movement. The current guidelines now define a leg movement arousal when < 0.5 seconds is present between the end of one event and the onset of the other event, regardless of whether the leg movement or the arousal occurred first.

REM behavior disorder is a parasomnia characterized by the lack of REM-related atonia, which results in range of movement behaviors during REM sleep including hand motions, gestures, punching, kicks, and dream-related behaviors. A requirement for the diagnosis of REM behavior disorder is the documentation of behaviors enacted during REM sleep or demonstration of excessive amounts of sustained or intermittent elevations in submental, upper, or lower limb EMG twitching. The AASM Manual attempts to codify the identification of sustained and intermittent elevations in muscle tone during REM sleep. Sustained muscle activity (tonic EMG) is defined as an epoch of REM sleep with at least 50% of the duration of the epoch having a chin EMG amplitude greater than the minimum amplitude than in non-REM sleep. Excessive transient muscle activity (phasic EMG) is defined by the presence of bursts of transient muscle activity in 50% of sub-epochs of 3-second duration (each 30-second epoch is divided into ten 3-second epochs). The presence of either of these observations in the context of an appropriate clinical history suggests the diagnosis of REM behavior disorder.

Scoring guidelines for other sleep-related movement disorders are summarized in Table 4.

Scoring of Respiratory Events

The AASM Manual provides additional specificity in both the technical requirements and the scoring rules for the detection of respiratory events during sleep. A summary of the scoring rules for respiratory events is shown in Table 5. A new technical requirement is the use of a thermistor to define an apnea, and a nasal cannula for the detection of hypopneas. Two new rules have been defined to assess the presence of hypoventilation and Cheyne-Stokes breathing during sleep. Hypoventilation is present when a $\geq$ 10 mm Hg increase in P_{aCO_2} is observed during sleep, compared to wakefulness. The necessary duration to score periods of hypoventilation was not defined, due to insufficient evidence. Cheyne-Stokes breathing is considered to be present if there are at least 3 consecutive cycles of crescendo-decrescendo changes in breathing and either a central apnea-hypopnea index (AHI) of 5 events/h or crescendo-decrescendo change in breathing amplitude of at least 10 consecutive minutes.

The area that has created perhaps the most controversy is the AASM Manual's definition of hypopneas. The Manual provides both a recommended and an alternative definition for hypopneas. The recommended definition of a hypopnea requires an event of at least 10 seconds duration in association with a $\geq$ 30% drop in the baseline amplitude and a $\geq$ 4% desaturation from the baseline saturation. The alternative definition defines a hypopnea as an event of at least 10 seconds duration in association with a $\geq$ 50% drop in the baseline amplitude and either a $\geq$ 3% desaturation from the baseline saturation or an arousal.

The recommended definition conforms to the definition of hypopneas advocated by the Centers for Medicare and Medicaid Services (CMS) and is based on the definition of hypopneas utilized by the Sleep Heart Health Study.[36] Although a clear body of evidence has been established regarding the role of oxyhemoglobin desaturations in the contribution to OSA morbidity, the contribution of arousals to OSA comorbidity has not been clearly established.[37,38] Nevertheless, it is important to recognize that the definition adopted by both CMS and the AASM Manual are based primarily on consensus rather than on a clearly established evidence base. Furthermore, the current definitions are primarily based on data using thermistor technology, instead of the more sensitive and reliable nasal pressure technology, suggesting that the current recommendations may already be outdated and that a new evidence base is required for validation of the current

Table 4. Movement Disorder Rules from the American Academy of Sleep Medicine Manual

Movement Disorder	American Academy of Sleep Medicine Manual Definitions
Leg movement	1. The minimum duration of a leg movement (LM) is 0.5 s.
	2. The maximum duration of a leg movement is 10 s.
	3. The minimum amplitude of a LM is an 8 μV increase in EMG (electromyogram) voltage above the resting EMG.
	4. The timing of the onset of a LM is defined as the point at which there is an 8 μV increase in EMG voltage above the resting EMG.
	5. The timing of the ending of a LM event is defined as the start of a period lasting at least 0.5 s during which the EMG does not exceed 2 μV above the resting EMG.
PLM (periodic leg movements) series	1. The minimum number of consecutive LM events needed to define a PLM series is 4 LMs.
	2. The minimum period length between LMs (defined as the time between the onsets of consecutive LMs) to include them as part of a PLM series is 5 s.
	3. The maximum period length between LMs (defined as the time between the onsets of consecutive LMs) to include them as part of a PLM series is 90 s.
	4. LMs on 2 different legs separated by $<$ 5 s between movement onsets are counted as a single movement.
Alternating leg muscle activation (ALMA)	1. The minimum number of discrete and alternating bursts of leg muscle activity needed to score an ALMA series is 4 ALMAs.
	2. The minimum frequency of the alternating EMG bursts is 0.5 Hz.
	3. The maximum frequency of the alternating EMG bursts is 3.0 Hz.
Hypnagogic foot tremor (HFT)	1. The usual maximum EMG burst duration seen in fragmentary myoclonus is 150 msec.
	2. At least 20 min of NREM (non-rapid-eye-movement) sleep with EFM must be recorded.
	3. At least 5 EMG potentials per minute must be recorded.
Bruxism	1. Bruxism may consist of brief (phasic) or sustained (tonic) elevations of chin EMG activity that are at least twice the amplitude of background EMG.
	2. Brief elevations of chin EMG activity are scored as bruxism if they are 0.25–2 s in duration and if at least 3 such elevations occur in a regular sequence.
	3. Sustained elevations of chin EMG activity are scored as bruxism if the duration is more than 2 s.
	4. A period of at least 3 s of stable background chin EMG activity must occur before a new episode of bruxism can be scored.
	5. Bruxism can be scored reliably by audio in combination with polysomnography by a minimum of 2 audible teeth-grinding episodes per night of polysomnography, in the absence of epilepsy.
REM behavior disorder (RBD)	1. Sustained muscle activity (tonic EMG): An epoch of REM sleep with at least 50% of the duration of the epoch having a chin EMG amplitude greater than the minimum amplitude than in NREM sleep, or
	2. Excessive transient muscle activity (phasic EMG): the presence of bursts of transient muscle activity during REM sleep in 50% of sub-epochs of 3-s duration (each 30-s epoch is divided into 10 sequential 3-s epochs).
	3. The polysomnographic characteristics of REM behavior disorder are characterized by either or both of the following features:
	a. Sustained muscle activity in REM sleep on the chin EMG
	b. Excessive transient muscle activity during REM on the chin or limb EMG.
Rhythmic movement disorder	1. The minimum frequency for scoring rhythmic movements is 0.5 Hz.
	2. The maximum frequency for scoring rhythmic movements is 2.0 Hz.
	3. The minimum number of individual movements required to make a cluster of rhythmic movements is 4 movements.
	4. The minimum amplitude of an individual rhythmic burst is 2 times the background EMG activity.

definitions. It is of interest to note that the alternative definition was in fact proposed as the recommended definition, but the Board of Directors of the AASM chose to conform with the CMS definition and adopted it as the recommended definition for hypopneas (see the discussion that follows this article).[20]

Ruehland et al[39] made the first attempt to evaluate the impact of the AASM Manual's two hypopnea definitions on the AHI, and compared it to the so-called "Chicago criteria,"[40] which are most comparable to the AASM's alternative definition, with a few important distinctions. First, the Chicago criteria specify an amplitude reduction of $<$ 50% for at least 10 seconds in association with a 3% desaturation from the baseline saturation or an arousal, whereas the alternative definition specifies an amplitude reduction of $>$ 50% in association with the desaturation or

Table 5. Respiratory Rules from the American Academy of Sleep Medicine Manual

Respiratory Events	Rule
Event duration	1. The event duration for an apnea or hypopnea is measured from the nadir preceding the first breath that is clearly reduced to the beginning of the first breath that approximates the baseline breathing amplitude.
	2. When baseline breathing amplitude cannot be easily determined (and when underlying breathing variability is large), events can also be terminated when either there is a clear and sustained increase in breathing amplitude or, in the case where a desaturation has occurred, there is event-associated resaturation of at least 2%.
Event type	1. Obstructive apnea: associated with continued or increased inspiratory effort throughout the entire period of absent air flow.
	2. Central apnea: associated with absent inspiratory effort throughout the entire period of absent air flow.
	3. Mixed apnea: associated with absent inspiratory effort in the initial portion of the event, followed by the resumption of inspiratory effort in the second portion of the event.
Apnea	1. There is a drop in the peak thermal sensor excursion by ≥ 90% of the baseline.
	2. The duration of the event is at least 10 s.
	3. At least 90% of the event's duration meets the amplitude-reduction criteria for apnea.
Hypopnea	Recommended Rule
	1. The nasal pressure signal excursion drops by ≥ 30% of baseline.
	2. The duration of this drop occurs for a period ≥ 10 s.
	3. There is a ≥ 4% desaturation from pre-event baseline.
	4. At least 90% of the event's duration must meet the amplitude reduction of criteria for hypopnea.
	Alternative Rule
	1. The nasal pressure signal excursion drops by ≥ 50% of baseline.
	2. The duration of this drop occurs for a period ≥ 10 s.
	3. There is at least ≥ 3% desaturation from pre-event baseline or there is an associated arousal.
	4. At least 90% of the event's duration must meet the amplitude reduction of criteria for hypopnea.
Respiratory effort-related arousal (RERA)	If there is a sequence of breaths lasting ≥ 10 s characterized by increasing respiratory effort or flattening of the nasal-pressure waveform leading to an arousal from sleep when the sequence of breaths does not meet the criteria for an apnea or hypopnea.
Hypoventilation	A ≥ 10 mm Hg increase in P_{aCO_2} is observed during sleep, compared to the awake supine value.
Cheyne-Stokes breathing	Cheyne-Stokes breathing is present if there are at least 3 consecutive cycles of crescendo-decrescendo changes in breathing and either:
	1. A central apnea-hypopnea index of 5 events/h of sleep, or
	2. Crescendo-decrescendo change in breathing amplitude of at least 10 consecutive minutes.

arousal. Second, the Chicago criteria allow for a hypopnea to be scored if there is an amplitude reduction of > 50% without an arousal or desaturation, which the alternative definition no longer allows. Thus, the Chicago criteria can be considered the most "liberal" for scoring hypopneas, when compared to the alternative definition or the recommended definition.

The authors scored 328 consecutive in-lab sleep studies using the 3 criteria and determined the effects of each approach on the AHI. The sample was 65% male, obese (mean body mass index 32.7 kg/m^2), and middle age (mean age 51 years). The authors found dramatic differences in the reported AHI among the definitions. The median (inter-quartile range) AHI was 25.1 (11.1–48.5) events/h with the Chicago criteria, 8.3 events/h with the recommended criteria, and 14.9 events/h with the alternative criteria. The median contribution of hypopneas to the AHI was 82.3% with the Chicago criteria, 38.9% with the recommended criteria, and 64.7% with the alternative criteria. The recommended criteria resulted in a median reduction in AHI of 3.5 events/h, but the inter-quartile range was from 0–20.1 events/h. The most significant impact of the scoring rules was on identification of OSA cases (an AHI ≥ 5 events/h). Under the Chicago criteria, 92% of patients studied would have had OSA. In contrast, only 59% had OSA based on the recommended criteria, and 76% had OSA based on the alternative criteria. The authors concluded that there was substantial variability in AHI results, based on the definition of hypopnea in the AASM Manual, and that consideration should be given to the adoption of a single standardized hypopnea definition. They also identified that arousals rather than amplitude criteria were the major source of variability between the alternative and recommended criteria.[39]

Although evidence exists to support the role of intermittent hypoxia in the myriad consequences of OSA, there is less evidence demonstrating the role of arousals in OSA outcomes. In part, previous difficulties in the reliability of

scoring arousals, particularly if arousals are not scored in the context of respiratory signals, have contributed to this issue. Furthermore, the concurrence of arousals with oxyhemoglobin desaturations with respiratory events can make it difficult to discern the independent contributions of both physiologic events to OSA outcomes.

Guilleminault et al recently challenged whether the recommended criteria for hypopnea were appropriate in identifying OSA in lean patients.[41] Lean individuals are less likely to experience large desaturations than obese patients, so arousals may be more important in explaining symptoms of daytime function. In a group of 35 patients, age 36.1 ± 10.3 years, body mass index 24.4 ± 1.1 kg/m^2, and Epworth Sleepiness Scale score 10.9 ± 2.3, the authors scored sleep studies before and 3 months after therapy (CPAP, oral appliance, or upper-airway surgery), using the Chicago criteria, the recommended criteria, and the alternative criteria. There were significant discrepancies in the AHI: Chicago 26.9 ± 7.3 events/h; recommended 6.4 ± 3.1 events/h; alternative 20.6 ± 8.2 events/h. Fourteen of the 35 patients would have had an AHI < 5 events/h under the recommended criteria, whereas none of the patients had an AHI < 5 events/h under the alternative or Chicago criteria. The authors reported that the change in Epworth Sleepiness Scale score best correlated with the change in AHI, using the arousal-based definition of AHI (ie, Chicago or alternative criteria, r = 0.6 for both) rather than a non-arousal-based definition of AHI (ie, recommended criteria, r = 0.4). The authors concluded that the recommended criteria would have failed to detect OSA in these thin individuals and would have categorized severe cases of OSA as mild in a significant proportion of patients. Thus, the authors concluded that the recommended criteria should not be applied in lean patients. If CMS rules are to be changed, the sleep community will need to perform further studies demonstrating the role of arousals in the clinical sequelae of OSA. Of note, the AASM recently convened a task force to review the respiratory scoring guidelines in part to re-evaluate the 2 definitions.

Summary

The conduct of polysomnography studies requires an understanding of basic electrical concepts, differential amplifiers, filtering, and analog-to-digital conversion. Furthermore, recent changes to the approach of scoring sleep studies have been adopted that may affect interpretation of sleep study results, particularly with respect to defining OSA. An understanding of these concepts will assist sleep physicians, respiratory therapists, and sleep technologists in the interpretation of polysomnography studies and place the results in the context of the patient history.

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Discussion

Minkley:* I don't mean to give you insomnia over hypopneas, but I want to ask you about central hypopneas. In the definition for what needs to be reported in the manual it refers to central events apneas and hypopneas, yet there's no definition for central hypopnea. In most labs it isn't standard practice to use esophageal pressure, which would be a clear, easy way to score central hypopneas, This has an impact when we look at Medicare guidelines for the diagnoses of central sleep apnea, which requires 50% of the AHI to be "central events," which currently really means 50% central apneas because central hypopneas aren't scored. This impacts eligibility for bi-level and adaptive support ventilation. Could you comment on that?

Patil: Yes, that's a difficult one because it also has potential implications for treatment and whether you really should be using CPAP or perhaps some sort of bi-level device. The accepted standard for assessing the presence of central hypopneas should be a signal that measures effort, as defined in the recent AASM manual. Esophageal pressure would be the ideal, but that's hard to do in every patient. At one point our laboratory used to do esophageal pressure measurements in every patient on a CPAP titration study. However, there are other existing tools that I think can discern the presence of an obstructive hypopnea versus a central hypopnea, in the setting of an inductive plethysmography belt for effort assessment. That signal is a high-fidelity flow or nasal pressure signalthat is not filtered. From a high-fidelity flow signal we can determine the presence of flow limitation, an airflow pattern associated with airflow obstruction.

In my experience, filtering of the airflow signal in many labs is performed in a way that makes it impossible to determine whether flow limitation is present. Snoring artifact in an airflow signal helps to identify flow limitation, so why would we want to filter it out? If we are able to identify flow limitation well, we can start to

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differentiate between obstructive hypopneas and central hypopneas and understand the patient's pathophysiology and treat the patient more appropriately. So I think there are approaches available, and educating physicians and technologists is vital.

Minkley: Yes, and sometimes defining it in context with the total flow and effort pattern can help. Cheyne-Stokes respiration is a perfect example. If the effort channels and the flow correlate (have the same pattern), then you have a reasonable expectation that it's a central hypopnea.

Patil: Absolutely.

Gay: With respect to hypopneas, 30% versus 50% are relative values, and there's the fine art of creative scoring that can get you to where you want to be with a diagnosis that's not to be overlooked. With respect to another aspect of the new scoring, it seems these new guidelines with the stroke of a pen—or are at least by proposing these new rules—they have eliminated UARS [upper-airway resistance syndrome] as a concept from the whole disease management side. These new rules may or may not make it permissible to treat under the present Medicare guidelines. Also, the robust literature that we have on UARS now seems to be in question. That entity is suddenly disappearing from all of this discussion. Do you want to comment on that?

Patil: In my view, for UARS, I tend to be more of a lumper than a splitter, so I tend to think of UARS as just another manifestation of OSA (predominantly hypopneas with arousals), because I believe the arousals are telling us something. I believe you're right that we are missing a group of patients whom we see in the clinic and can tell they're impaired by their fatigue or sleepiness. Their sleep studies show that their respiration and sleep continuity are abnormal, but the current guidelines are not recognizing that, and the entities, such as Medicare, that pay for these studies are saying, "We're not going to look at your events you score with arousals in your respiratory disturbance index; we're only going to look at events with 4% desaturations in your AHI."

I believe what we need to do as a field is to rise to the challenge and push back by producing research studies that assess the impact of hypopneas with arousals, UARS, or however you want to call it, on patient outcomes. If we can show in well designed studies that these events are important and result in poor patient outcomes, such as fatigue, tiredness, or other morbidity, we can change the scoring manual and convince payers to include such events in the definition of OSA.

Kuna: I think it's going to be very difficult to standardize the scoring of polysomnograms, because we're using qualitative signals. These are not pulmonary function tests, and one of the problems I have as a clinician is when somebody comes in with a sleep study result from another laboratory. I'm not looking at a report based on metric measurements such as in a pulmonary function test, but a report based on pattern recognition, like a chest radiograph. I think it would be very helpful to the field to standardize the data we're collecting and standardize how it's reported across all recording systems so that the raw files can be shared.

That is certainly the case in radiology. If a patient comes to my pulmonary clinic and says, "I had an MRI at such-and-such hospital," that hospital can send me the file and I can bring it up on my system regardless of the MRI manufacturer, and that has made an enormous difference in the ability to care for patients with pulmonary disorders. We need that capability for polysomnography.

Patil: You're absolutely right. I can't tell you the number of times I've asked a lab to send me a sleep study and they've asked me what sleep system I'm using. I'm using Somnologica (Embla Recording Systems, Broomfield, Colorado) and they're using Rembrandt (Embla Recording Systems, Broomfield, Colorado). Certainly there is an attempt to standardize, with the EDF [European data format] files, but there are limitations from that standpoint as well. I agree with you; that's something we need to develop as a field. It would reduce costs too.

Quan: As an author of the AASM Scoring Manual, I'd like to provide a little background. Some people might wonder why there's an alternative definition and why it's recommended to use the other definition. The committee recommended to the AASM board of directors to use the alternative definition as the primary definition. However, the board thought that the field would have to go back to Medicare and say, "You've gotta change your definition," and we felt that was probably too big of a political battle to fight at that time, because Medicare was just finally recognizing and paying for sleep studies. It was not necessarily the best scientific decision based on the data we had, but it was one that considered what was best for patient care, so that the sleep studies would be covered.

Patil: That is unfortunate, as it will make it that much more difficult to convince Medicare to change their definition now.

Quan: With respect to UARS, when we were analyzing the published scientific data, only one study[1] did an in-depth investigation of UARS. We felt that the prevalence of the disorder was not that high, so we need more studies. The manual was designed to be updated: that was the intent. So

what we have to do is to accumulate sufficient data to update the manual.

For many lab tests the definition of abnormal is based on running a large number of tests on "normal" people and defining abnormal as the top and bottom 5% of the population. I would submit that this is not what should be done for sleep apnea. What we should do is look at the outcomes associated with sleep apnea and see what metric or definition correlates best with an abnormal outcome, whether the outcome is glucose tolerance, cardiovascular disease, hypertension, neurocognitive function, or some other test. That's how we should define what's normal and abnormal, and what we should treat. The definition of sleep apnea shouldn't just be derived from just a bell-shaped curve and from just looking at statistics.

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Patil: Absolutely, and Dr Kapur touched on that in his presentation when he illustrated that different oxyhemoglobin desaturation levels are associated with cardiovascular and glycemic control outcomes.[1] It seems like our field has some marching orders when it comes to goals for revisions to the next edition of the manual.

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