eMedicine Specialties > Neurology > Sleep-Related Diseases

Sleep Stage Scoring

Ron A Shatzmiller, MD, MSc, Fellow in Clinical Neurophysiology, Department of Neurology, Keck School of Medicine of the University of Southern California, LAC+USC Medial Center

Andres A Gonzalez, MD, Assistant Professor of Clinical Neurology, Medical Director, Division of Intraoperative Neurophysiology and Sleep, Keck School of Medicine of the University of Southern California; David Y Ko, MD, Associate Professor of Clinical Neurology, Associate Director, USC Adult Epilepsy Program, Keck School of Medicine of the University of Southern California; Michelle R Zeidler, MD, Assistant Professor, Department of Pulmonary and Critical Care, University of California, Los Angeles, David Geffen School of Medicine; Attending Physician, Division of Pulmonary, Critical Care and Sleep Medicine, Greater Los Angeles, West Los Angeles Veterans Affairs Medical Center

Updated: Jun 17, 2010

Introduction/ Historical Perspective

This article is based on the updated *American Academy of Sleep Medicine Manual for Scoring Sleep and Associated Events*.^[1]This manual replaces the original Rechtshaffen and Kales sleep scoring manual of 1968, commonly known as the "R and K" rules.^[2]

Prior to the R and K rules, electroencephalographic (EEG) recording revealed distinct brain rhythms uniquely seen during sleep but that were not universally defined and categorized. Loomis and colleagues^[3]noted fragmentation and fallout of alpha rhythm with sleep onset, and subsequent onset of sleep spindles, K complexes and high amplitude slow waves. Sleep was divided into 5 stages (A-E), with later stages possessing more slow-frequency and high-amplitude waves.^[3]The discovery of rapid eye movement (REM) sleep by Kleitman and Dement in 1957^[4]led to a classification of sleep stages that included REM sleep.

In 1968, Rechtschaffen and Kales convened a panel of experts to agree on a standardized manual for the scoring of sleep stages. Sleep stages were divided into wakefulness, stage 1-4 (non-REM), or REM. At least 1 EEG lead was recommended (C3 or C4 referenced to the opposite ear or mastoid) as well as 2 electro-oculogram (EOG) leads and a submental electromyography lead. The R and K rules recommended dividing the polysomnographic record of sleep into 30 second epochs, commencing at the start of the study. Historically, the 30-second interval was used because at a paper speed of 10 mm/s, ideal for viewing alpha and spindles, 1 page equates to 30 seconds. A stage was assigned to each epoch and if 2 or more stages coexist during a single epoch, the stage comprising the greatest portion of the epoch was used.

In 2004, the American Academy of Sleep Medicine (AASM) commissioned a steering committee to assemble a new sleep scoring manual that would address sleep staging as well as

the scoring of arousals, respiratory, cardiac, and movement events. Eight separate task forces were assembled to address the various issues. The establishment of rules was guided by the following principles: the rules should be compatible with published evidence, they should be based on biologic principles, they should be applicable to both normal and abnormal sleep, and they should be easy to use by clinicians, technologists, and scientists.

The main changes in sleep scoring in the 2007 AASM manual include the following:

- □ The addition of a frontal EEG derivation in addition to the central and occipital leads commonly used: The R and K classification required that the central lead always be used if only 1 derivation was possible.
- Changing the sleep stages to stage W (wake), stages N1-N3 (non-REM), and stage R (REM) from the previously described R and K stages of wakefulness, 1-4, and REM: The change in abbreviations is to avoid confusion between the 2 classification systems. Stage 3 and 4 in the old R and K rules were abbreviated to stage N3 in the new rules, as no physiologic or clinical basis exists for a difference between stages 3 and 4.

Polysomnographic Leads and Minimal Technical Requirements for Sleep Scoring

Sleep stage scoring is a rule-based neurophysiology test requiring an understanding of the basic mechanisms underlying the generation of cephalic electric potentials coupled with eye movements and muscle signal. Signals of interest are generated from the brain (ie, cortex and deeper structures) and the facial muscles (ie, signals picked up by periorbital and submental electromyographic [EMG] leads).

Interference with the signals of interest is encountered through many mechanisms, including physiological attenuation of the cerebral electric potentials by scalp muscle and bone, intrusion into the signal by slow cyclic respiration, movement, ECG signal, or external electric fields, and impaired contacts between the recording electrodes and the skin surface. Discriminating true signal from artifact can be one of the most challenging aspects of scoring the stages of sleep.

Cortical signals

The EEG signal is of primary importance in interpreting polysomnographic studies. It records electric potentials generated by the interaction between the cortex and the deeper brain structures, especially the thalamus. Two centrocephalic, 2 occipital, and 2 frontal cortical channels are recorded. Measurement of EEG signals is possible because of the relative difference in potential between the 2 recording electrodes; 1 electrode is considered negative with respect to the other. Negative discharges, by convention, are represented by an upwardly deflecting wave. The left channels are ascribed odd numbers and the right channels are ascribed even numbers.

The polysomnograph references the left and right centrocephalic electrodes (C3, C4), the left and right occipital electrodes (O1, O2), and the left and right frontal leads (F3, F4) to electrodes on the opposite right and left mastoid processes (M2, M1). These electrodes are chosen to capture slow waves (best represented frontally), spindles (best seen in central derivation), and the posterior dominant or alpha rhythm (best seen occipitally). The general rule is to read only from the left cortical channel. However, when the left channel develops artifact or the validity of the signal is unclear, comparison is made with the right cortical channel. Cortical signals are defined slightly differently according to the reference used. The following convention is used here:

 \Box Delta is the slowest activity at <4 cycles per second (cps).

- \Box Theta is 4-8 cps.
- \Box Alpha is 8-12 cps.
- \Box Beta is >12 cps.

Minimal EEG technical requirements include the following:

- □ Three EEG derivations (frontal, central, occipital regions) for sampling
- □ Right and left mastoid referential leads

Muscle signals

The EMG signals are muscle twitch potentials that may offer additional assistance in defining a sleep stage. Their use is based on the finding that, during sleep, muscle activity decreases. During REM sleep, muscle activity is at its nadir. However, in many cases, appreciating the decreasing tone is difficult. The relative silence during REM sleep may not be of help in distinguishing REM sleep from the preceding or subsequent sleep stages.

Compounding the problem of interpreting EMG channels is intrusion of artifact into the signal. Some examples include cyclic chewing movements, irregular teeth grinding, or steady high-amplitude noise generated by increased pressure on the electrode (eg, as caused by lying on the chin). Additionally, muscle artifact may spill over into the cortical leads. ECG signal is a specific type of cardiac artifact that can appear in all or several channels and can be recognized by the telltale QRS complexes.

Minimal EMG technical requirements include3 chin EMG electrodes; 2 are used throughout the study with the additional lead used as a backup.

Eye movements

The EOG signals measure changes in the electric potential of the positive anterior aspect of the eye relative to the negative posterior aspect. Horizontal axis electrodes are placed near the outer canthi and vertical axis electrodes below and above the eye to measure transient changes in potential during the actual eye movement. During any eye movement, the cornea (positive) moves toward 1 electrode, while the fundus (negative) moves away from the same electrode. When the eye is not moving, the change in relative position is 0, and the eye leads do not record a signal.



Electro-oculogram. During any eye movement, the cornea (positive) moves toward one electrode, while the fundus (negative) moves away from the same electrode. When the eye is not moving, the change in relative position is zero, and the eye leads do not record a signal. Conjugate eye movements thus cause out-of-phase EOG deflections.

Slow rolling eye movements are recorded as long gentle waves, while rapid jerking movements are represented by sharply contoured fast waves. Blinking of the eyes produces rapid vertical movements. Eye movements during drowsiness and stage N1 sleep may be jerky, irregular, or gently rolling. In deeper stages of sleep, macro eye movements cease altogether. During REM sleep, eye movements again become active and jerky. The intensity of the bursts of activity is used to describe the density of REM sleep.

Minimal EOG technical requirements include the following:

- □ One lead placed 1 cm below the left outer canthus
- □ One lead placed 1 cm above the right outer canthus

Sleep Stages

Initially, the clinician should scroll through the entire record to evaluate the quality of the recording and the usefulness of specific channels. The major stages should be identified, and any variability associated with the patient should be noted.

Stage W (equivalent to R and K manual stage Wake)

The first several epochs of the record will usually be stage W, although occasionally a patient is so sleepy and is asleep when the recording is started. During normal wakefulness with closed eyes, the posterior dominant rhythm (PDR) is detected over the occipital leads. It is a sinusoidal rhythm with a frequency of 8.5-13 Hz, roughly the range of alpha frequency, and thus is also called the alpha rhythm.



Stage W is scored when there is alpha rhythm in greater than 50% of the epoch.

Stage W. Alpha activity is present for greater than 50% of the epoch.

10-20% of normal patients will not have a detectable alpha rhythm on EEG. In such cases, Stage W may still be scored if 1 of the 3 following markers of alertness are detected:

- □ Eye blinks with the eyes open or closed
- □ Reading eye movements, consisting of a slow phase followed by a rapid movement in the opposite direction
- □ The presence of irregular conjugate eye movements with normal or high chin muscle tone suggesting that the subject is awake and looking around

Additional features of stage W include the following:

- □ The EEG shows beta activity while eyes are open and alpha activity when the eyes close.
- □ The EMG reflects the high-amplitude muscle contractions and movement artifacts.
- □ The EOG shows eye blinking and rapid movement.

Stage N1 (equivalent to R and K manual stage I)

Sleep onset is defined as the first stage of the polysomnogram scored other than stage W. Stage N1 is scored when alpha rhythm is attenuated and replaced by low amplitude, mixed frequency for more than 50% of the epoch.



Stage N1. Alpha rhythm is attenuated and replaced by low-amplitude, mixed-frequency for more than 50% of the epoch.

Some patients may have no discernable background rhythm, even when their eyes are closed while awake. In such patients, distinguishing the onset of sleep is more difficult. For such cases, stage N1 may be scored based on the presence of the following:

- □ Vertex sharp waves, which are prominent sharply contoured negative waves lasting <0.5 seconds and maximal over the central region
- □ Slow, rolling eye movements, detectable on EOG
- □ Activity in the range of 4-7 Hz with slowing of the background frequencies of the EEG by 1 Hz or more when compared with stage W

There is often a decrease in muscle tone as detected by chin EMG when compared with stage W. Of note, slow rolling eye movements may begin even in wakefulness; thus, stage N1 may be scored earlier in patients without a discernable alpha rhythm.

Stage N2 (equivalent to R and K manual stage II)

Stage N2 is characterized by the presence of sleep spindles and K complexes on a background of low-amplitude, mixed frequency activity.



Stage N2. Note K complexes unassociated with arousal and trains of sleep spindles.

- □ Sleep spindles are bursts of waves, named according to their shape. They are seen maximally over the central leads, have a frequency of 12-16 Hz and last 0.5 seconds or longer (see image above).
- □ K complexes have an initial negative wave, followed by a positive wave. They are clearly different than the background and last 0.5 seconds or longer (see image above). They are usually maximally seen over the frontal derivations.
- □ K complexes are also seen with arousals.

The rules governing the beginning, continuation, and end of stage 2 sleep are as follows:

Begin scoring stage N2 sleep if either of the following are present in the first half of the epoch or the second half of the preceding epoch.

- □ [Здесь снова вероятно, по ошибке вставлен предыдущий рисунок Stage N2. Note K complexes unassociated with arousal and trains of sleep spindles.]
- One or more K complexes unassociated with arousal
- □ One or more trains of sleep spindles

Continue scoring subsequent epochs of mixed frequency, low-amplitude activity without K complexes or sleep spindles as stage N2 if they are preceded by one of the following:

- □ K complexes unassociated with arousals
- □ Sleep spindles

End scoring stage N2 sleep in the case of any of the following:

- A transition to stage WAn arousal, that is, a burst of activity lasting 3 seconds or longer. This may involve the EEG, EOG, and EMG channels, and minimally involves a burst of alpha or theta PDRA major body movement followed by slow eye movements and lowamplitude mixed EEG without nonarousal-associated K complexes or sleep spindles
- □ A transition to stage N3
- □ A transition to stage R

Stage N3 (equivalent to R and K manual stage III and stage IV)



Stage N3 is scored when there is slow wave activity in greater than 20% of the epoch.

Stage N3. There is slow wave activity in greater than 20% of the epoch.

Slow waves are defined as waves with a frequency of 0.5-2 Hz and amplitude of 75 microvolts peak to peak when measured over the frontal region.

Although not required for scoring, sleep spindles may persist in stage N3 sleep. Eye movements are unusual in this stage. While variable, chin EMG tone is often low, sometimes as low as in stage R.

Stage R (equivalent to R and K manual stage REM)

Stage REM is scored when the following occur:

- □ The EOG leads demonstrate REMs, which are irregular, conjugate, sharply peaked eye movements with an initial phase lasting less than 500 msec.
- □ The EEG shows relatively low-voltage and mixed-frequency activities and may resemble the EEG of stage N1 or the slow alpha activity of stage W
- □ Chin EMG tone is at the lowest of any stage in the polysomnogram



Stage R. This demonstrates rapid eye movements, relatively low EMG tone, and low-voltage, mixed-frequency EEG activity.

Polysomnographic features that are supportive but not required in stage R include sawtooth waves and phasic muscle twitches. Sawtooth waves are 2-6 Hz serrated bursts of activity maximal centrally that precede REMs. Phasic muscle activity are bursts of EMG activity lasting less than 0.25 msec and may be detected on chin EMG, anterior tibialis, or EOG-EEG leads.



Stage R. This demonstrates sawtooth waves.

The rules governing the scoring of epochs surrounding stage R have received much attention. In essence, epochs following stage R are scored as stage R unless (1) there is a clear change to another sleep stage, (2) an arousal or major body movement followed by eye movements takes place, or (3) K complexes or sleep spindles, hallmarks of stage N2 sleep, occur in the absence of REMs. If REMs are present with low chin EMG, an epoch of REM is scored, even if sleep spindles and K complexes are present. There are rules governing the special case of the transition from N2 sleep to REM sleep without clear REMs. If low chin tone is present, but no sleep spindles or K complexes are present in an epoch, stage R is scored. If low chin tone is present but K complexes and sleep spindles are present, an epoch of stage N2 sleep is scored.

Special Considerations

Arousals

Arousals are paroxysms of activity lasting 3 seconds or longer with at least 10 seconds of sleep preceding the change. The minimum arousal is simply a paroxysmal burst in the EEG channel, usually to alpha or theta activity. Arousal from stage N1 is common and usually represented by a burst of activity on the EEG, EOG, and EMG. For an arousal to be scored during REM, there also must be an increase in the submental EEG for at least 1 second. If the burst results in alpha activity for greater than 50% of the record, then the epoch is scored as wake.

Major body movements

At times, movement and muscle artifact obscures the EEG for more than half the epoch such that the stage cannot be determined. In such cases, the following are true:

□ Epochs with alpha rhythm present for any part of the epoch are scored as stage W.

- □ Epochs with a major body movement, but no discernable alpha rhythm, that precede or follow an epoch of stage W are scored as stage W. (This rule reflects the logic that a body movement usually reflects wakefulness or a lighter stage of sleep.)
- \Box Otherwise, the epoch is scored as the same stage as the epoch that follows it.

Normative Sleep Stage Data

Normative values have been constructed based on sleep staging results to help quantify the composition and quality of sleep. Normative data change with age and vary from center to center. In addition, most were collected using the R and K rules.

The AASM manual has recommended recording the values listed below.

For the purposes of illustration, normative data for different age groups is presented. (See Table below.)

- \Box Total time sleep (TST)
 - Total minutes of sleep Stage N1-N3 + stage R
- □ Time in bed (TIB) or total recording time
 - Monitoring period Lights out to lights on
- $\Box \quad \text{Sleep latency (SL)}$
 - Total number of minutes from lights out to first period of sleep
- $\Box \quad Stage R \ latency$

 \circ $\;$ Time from sleep onset to first epoch of REM sleep $\;$

- □ Wake after sleep onset (WASO)
 - o Minutes of wake after initial sleep onset and before the final awakening
- \Box Sleep efficiency (%)
 - (TST*100)/TIB
- □ Total time in each stage (Separate values for stage N1, N2, N3, and R)
- □ Percent of TST in each stage
 - Time in each stage/TST*100

Table. Normative Sleep Stage Data Across Age Groups.*

Age (y)	20-29	30-39	40-49	50-59	>60
TST (min)	374.9	375.8	370.2	366.6	348.8
Sleep Efficiency (%)	94.4	94.4	90.2	90.4	85.8
Sleep Latency (min)	6.3	10.0	8.4	6.1	8.2
Number of Awakenings	6.3	4.7	8.4	9.7	12.3
Stage R (%TIB)	22.2	23.1	20.4	20.9	16.4
Stage N1 (%TIB)	3.0	2.5	4.3	4.7	4.0

Stage N2 (%TIB)	50.5	52.8	54.6	56.7	57.6			
Stage N3 (%TIB)	18.8	16.1	10.9	8.1	7.7			
Adapted from Hirshkowitz M. Normal human sleep: an overview. <i>Med Clin North Am</i> . May 2004;88(3):551-65. ^[5]								

These values are useful in the diagnosis and management of sleep disorders. For example, increased sleep latency may be increased in primary insomnia and decreased in obstructive sleep apnea, whereas stage R latency may be decreased in narcolepsy.

Sleep architecture changes with advancing age. The percentage of stage R is greatest in neonates, with 50% of sleep time spent in this stage. This proportion decreases gradually to 20-25% at adolescence, with only a slight decrease after age 65.

Greater sleep fragmentation occurs with aging, such that wakefulness is intermixed with sleep. The percentage of stage N3 sleep decreases with age while percentage of time spent in N1 and N2 and WASO increases. Percent of TST spent in N3 may decrease because of a decrease in EEG amplitude with age, making it harder to meet criteria for N3. Some elderly individuals will have almost no N3 sleep.

Multimedia [list]

Media file 1: Electro-oculogram. During any eye movement, the cornea (positive) moves toward one electrode, while the fundus (negative) moves away from the same electrode. When the eye is not moving, the change in relative position is zero, and the eye leads do not record a signal. Conjugate eye movements thus cause out-of-phase EOG deflections.

Media file 2: Stage W. Alpha activity is present for greater than 50% of the epoch.

Media file 3: Stage N1. Alpha rhythm is attenuated and replaced by low-amplitude, mixed-frequency for more than 50% of the epoch.

Media file 4: Stage N2. Note K complexes unassociated with arousal and trains of sleep spindles.

Media file 5: Stage N3. There is slow wave activity in greater than 20% of the epoch.

Media file 6: Stage R. This demonstrates rapid eye movements, relatively low EMG tone, and low-voltage, mixed-frequency EEG activity.

Media file 7: Stage R. This demonstrates sawtooth waves.

11

References

- 1. C Iber, S Ancoli-Israel, A Chesson, SF Quan. *The AASM Manual for the Scoring of Sleep and Associated Events*. Westchester, IL: American Academy of Sleep Medicine,; 2007.
- Rechtschaffen A, Kales A, eds. A Manual of Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Subjects. US Department of Health, Education, and Welfare Public Health Service - NIH/NIND. 1968.
- 3. Loomis AL, Harvey EN, Hobart GA. Cerebral states during sleep, as studied by human brain potentials. *1937*. 1937;1:24-38.
- DEMENT W, KLEITMAN N. Cyclic variations in EEG during sleep and their relation to eye movements, body motility, and dreaming. *Electroencephalogr Clin Neurophysiol*. Nov 1957;9(4):673-90. [Medline].
- 5. Hirshkowitz M. Normal human sleep: an overview. *Med Clin North Am.* May 2004;88(3):551-65, vii. [Medline].
- 6. Niedermeyer E, Lopes Da Silva F. Electroencephalography: Basic Principles, Clinical applications, and Related Fields. *3rd ed.* Baltimore, Md: Williams and Wilkins;1993.
- Silber MH, Ancoli-Israel S, Bonnet MH, Chokroverty S, Grigg-Damberger MM, Hirshkowitz M, et al. The visual scoring of sleep in adults. *J Clin Sleep Med*. Mar 15 2007;3(2):121-31. [Medline].

Keywords

polysomnography, electroencephalography, EEG, cortical electroencephalography, sleep stages, wake stage, drowsiness, rapid eye movement, REM, sleep scoring, sleep-stage scoring, cephalic electric potentials

Contributor Information and Disclosures

Author

Ron A Shatzmiller, MD, MSc, Fellow in Clinical Neurophysiology, Department of Neurology, Keck School of Medicine of the University of Southern California, LAC+USC Medial Center

Ron A Shatzmiller, MD, MSc is a member of the following medical societies: American Academy of Neurology

Disclosure: Nothing to disclose.

Coauthor(s)

Andres A Gonzalez, MD, Assistant Professor of Clinical Neurology, Medical Director, Division of Intraoperative Neurophysiology and Sleep, Keck School of Medicine of the University of Southern California

Andres A Gonzalez, MD is a member of the following medical societies: American Academy of Neurology, American Clinical Neurophysiology Society, and American Medical Association

Disclosure: Nothing to disclose.

David Y Ko, MD, Associate Professor of Clinical Neurology, Associate Director, USC Adult Epilepsy Program, Keck School of Medicine of the University of Southern California

David Y Ko, MD is a member of the following medical societies: American Academy of Neurology, American Clinical Neurophysiology Society, American Epilepsy Society, and American Headache Society Disclosure: Pfizer Honoraria Speaking and teaching; UCB Honoraria Consulting; Lundbeck Consulting fee Consulting; Westward Consulting fee Consulting

Michelle R Zeidler, MD, Assistant Professor, Department of Pulmonary and Critical Care, University of California, Los Angeles, David Geffen School of Medicine; Attending Physician, Division of Pulmonary, Critical Care and Sleep Medicine, Greater Los Angeles, West Los Angeles Veterans Affairs Medical Center

Michelle R Zeidler, MD is a member of the following medical societies: American Academy of Sleep Medicine, American College of Chest Physicians, and American Thoracic Society Disclosure: Nothing to disclose.

Medical Editor

Carmel Armon, MD, MSc, MHS, Professor of Neurology, Tufts University School of Medicine; Chief, Division of Neurology, Baystate Medical Center

Carmel Armon, MD, MSc, MHS is a member of the following medical societies: American Academy of Neurology, American Academy of Sleep Medicine, American Association of Neuromuscular and Electrodiagnostic Medicine, American Clinical Neurophysiology Society, American College of Physicians, American Epilepsy Society, American Medical Association, American Neurological Association, American Stroke Association, Massachusetts Medical Society, Movement Disorders Society, and Sigma Xi

Disclosure: Avanir Pharmaceuticals Consulting fee Consulting

Pharmacy Editor

Francisco Talavera, PharmD, PhD, Senior Pharmacy Editor, eMedicine Disclosure: eMedicine Salary Employment

Chief Editor

Selim R Benbadis, MD, Professor, Director of Comprehensive Epilepsy Program, Departments of Neurology and Neurosurgery, University of South Florida School of Medicine, Tampa General Hospital

Selim R Benbadis, MD is a member of the following medical societies: American Academy of Neurology, American Academy of Sleep Medicine, American Clinical Neurophysiology Society, American Epilepsy Society, and American Medical Association

Disclosure: UCB Pharma Honoraria Speaking, consulting; Lundbeck Honoraria Speaking, consulting; Cyberonics Honoraria Speaking, consulting; Glaxo Smith Kline Honoraria Speaking, consulting; Ortho McNeil Honoraria Speaking, consulting; Pfizer Honoraria Speaking, consulting; Sleepmed/DigiTrace Speaking, consulting

Acknowledgments

The authors wish to thank Dr. Silverio Santiago for his insightful comments.

Further Reading

© 1994- by Medscape. All Rights Reserved (http://www.medscape.com/public/copyright)
