As described in the previous article, a comprehensive evaluation of sleep disordered breathing is contingent on examining respiratory patterns within the context of sleep/wake physiology. Yet, historically, respiratory event scoring has largely been based on respiratory tracings and oximetry alone, with minimal reference to the electroencephalogram (EEG) and other routine polysomnographic (PSG) parameters. This limits the scorer’s ability to identify the significance and etiology of the events in question. The objective of this article is to expand upon existing scoring rules by adding a practice-based perspective that includes a more in-depth analysis of the PSG data.

**Obstructive Apnea**

Following historical and current definitions, obstructive apnea is defined as cessation in airflow for at least 10 seconds with evidence of continued inspiratory effort. Of all existing scoring definitions, this one is usually the least problematic because the classic obstructive apnea pattern is quite obvious even in the absence of corroborating data (Figure 1). A possible exception may be the occurrence of central apnea with concomitant movement artifact that could be misinterpreted as “effort.” For example, a patient with narcotic-induced central apnea might have frequent body movements that distort the effort tracings, creating the illusion of obstructive events.

Patterns that resemble obstructive apnea (or hypopnea) also can be seen during wakefulness in patients who are restless and have difficulty falling asleep. This especially poses a problem when interpreting limited-channel home studies that do not record the neurophysiological aspects of sleep.

**Central Apnea**

The traditional definition for central apnea is based on absence of effort during an apneic event, suggesting cessation or alteration of central respiratory drive. The problem with this definition is that in some instances, events that appear ostensibly central are actually caused by upper airway obstruction. Interpreting such events strictly as central may lead to incorrect diagnosis and treatment assignment. There are several possible reasons why obstructive events might potentially appear as central:

- Effort may be present, but undetected by external sensors.
- The cessation of effort may represent a normal compensatory pause that follows a preceding arousal.
- The cessation of effort may represent a physiological variant of obstructive apnea.
- The respiratory belts may be loose, or the signal may be over-attenuated.

Determining the etiology of an event arguably holds greater relevance to the patient’s diagnosis than simply tabulating numbers and measuring event duration. It might be suggested that the term “central” is somewhat vague, and that a more meaningful way to describe an event is whether or not it is associated with a closed airway. Examining respiratory tracings alone is insufficient for this purpose, but a more global evaluation of the data usually provides the answer.

When upper airway closure occurs, the patient must perform an active maneuver to reopen it. This typically results in some form of activation, seen in the EEG, the electrooculogram (EOG), and/or the chin electromyogram (EMG). An obstructive event also is typically accompanied by a characteristic breakthrough snort. By examining these effects on a multi-channel polysomnogram, and correlating them with direct behavioral observation, one can more accurately determine the etiology of the event (Figures 2 and 3). This not only facilitates more meaningful data interpretation, but is also imperative during positive airway pressure (PAP) titrations. For example, when coinciding arousals and snores accompany residual “central” apneas on CPAP, one might suspect mask leak or an insufficient pressure level.

Once upper airway obstruction has been ruled out, it becomes imperative to seek an explanation for the events in question. Does the patient have a history of heart disease or stroke? Is the patient taking narcotic medications? Are the apneas prevalent throughout the study, or are they limited to transitional sleep? Are the apneas temporarily induced by CPAP treatment? Do the apneas resolve during rapid eye movement (REM) sleep? (While obstructive apneas typically worsen during REM sleep, central apneas often tend to improve or resolve during REM sleep.)

Some forms of central apnea may be benign, such as transitional, or post-arousal, central pauses that resolve with reestablished sleep. Other forms of central apnea may be related to an underlying medical condition. Central apneas and hypopneas appearing in a Cheyne-Stokes pattern are often seen in patients with congestive heart failure. Central apneas also may stem from cerebrovascular or neurological causes.

The appearance of central apnea on CPAP recently has received much attention, although this phenomenon has been observed since the early days of CPAP. As mentioned, one possible cause may be unresolved upper airway obstruction, especially when the events are accompanied by arousals or snores. Another possibility is that central apneas are temporarily induced by CPAP, especially when higher pressures are used. The terms “complex sleep apnea” and “CPAP emergent” central apnea have been proposed, but these terms are non-specific and should not be applied arbitrarily to all instances of
FIGURE 1. OBSTRUCTIVE APNEA (2-MINUTE WINDOW). THIS IS AN EXAMPLE OF CLASSIC OBSTRUCTIVE APNEA WITH ABSENCE OF AIRFLOW, CONTINUED INSPIRATORY EFFORT, AND AROUSAL OCCURRING AT THE END OF EACH EVENT.

central apnea on CPAP. If the level of CPAP is appropriate for the patient, and the patient does not have coexisting pathology affecting central respiratory drive, these residual events often disappear after acclimation to therapy.

AROUSALS WITH CENTRAL APNEA

In contrast to obstructive apneas, true central apneas are quiet and typically are not accompanied by arousal. However, exceptions do exist. For example, patients with Cheyne-Stokes respiration sometimes exhibit arousals; but the arousal tends to occur at the peak of the hyperventilatory phase, not at the end of the apneic event. This suggests that the airway is open and the patient is able to reinitiate breathing without having to perform a reopening maneuver. A possible cause for a subsequent arousal may be a venturi effect, with increased upper airway resistance occurring at the peak of the hyperventilatory phase; or the arousals may be related to unstable transitional sleep, resolving when sleep becomes more established.

MIXED APNEA

The term “mixed apnea” describes an apneic event that shows an absence of inspiratory effort during the initial portion of the event, followed by resumption of effort during the latter portion of the event. In most cases the initial absence of effort simply represents a normal compensatory pause following the preceding arousal. In some instances, it also is possible that a mixed apnea represents a true overlap of central and obstructive pathology. To bring greater relevance to the study, it is suggested that the latter be described as central apnea with an obstructive component. Clues to overlapping central pathology might include excessive duration of the central pause, the occurrence of purely central apnea during other parts of the study, and a plausible explanation for central etiology based on the patient’s clinical history.

HYPOPNEA

Of all the scoring guidelines presented to the sleep field, hypopnea has always been the most elusive. By definition, a hypopnea is a temporary reduction in airflow. The question has always been: How much of a reduction, and how does one measure it? Short of using a pneumotachograph, which is impractical for clinical sleep studies, the airflow signals recorded by a PSG are non-quantitative and susceptible to artifact. In addition, when sleep disordered breathing occurs, the signals are continually distorted by recovery breaths and/or movement artifacts occurring at the end of each event. Thus, the concept of measuring percentage drops in airflow from baseline is more hypothetical than practical. And even if precise measurements were possible, a reduction in airflow alone does not reveal the significance or etiology of the event, unless correlated with other PSG parameters.

Consequently, hypopneas historically have been scored based on their association with either a drop in O2 saturation or arousal. The AASM scoring manual currently offers two hypopnea definitions: one based on a 30-percent airflow signal amplitude drop accompanied by at least 4-percent desaturation; the other based on a 50-percent airflow signal amplitude drop accompanied by either a 3-percent desaturation or an arousal. From a practical perspective, the second definition is more realistic, because it brings the EEG into the equation, and because milder forms of obstructive hypopnea are more likely to be associated with arousal than O2 desaturation. However, Medicare currently does not recognize the second hypopnea definition.

The AASM scoring manual also states that “classification of a hypopnea as central, obstructive or mixed should not be performed without a quantitative assessment of ventilatory effort.” However, if only respiratory signals are examined, even quantitative measures can produce misleading results. As when differentiating central and obstructive apneas, a more practical way to classify hypopneas is by examining them within the context of other PSG channels, and by correlating the data with direct behavioral observation. When upper airway resistance is present, arousals, or other forms of activation occurring at the end of each event, accompanied by snoring or breakthrough snores, are far more reliable determinants of hypopnea etiology than attempts to measure ventilatory effort. In contrast to obstructive hypopneas, central hypopneas are quiet and typically occur without disturbances in the EEG, EOG or EMG channels (Figures 4 and 5).

SUMMARY

It is not the intent of this article to suggest that all respiratory disturbances can be precisely evaluated based on the presence or absence of arousals and snores, but clearly an expanded PSG analysis is more useful than relying on respiratory data alone. Moreover, a meaningful interpretation of sleep disordered breathing cannot be based solely on a set of rules. While rules are necessary as a basic framework, they must be tempered by clinical judgment and a thorough examination of all available data revealed by multichannel polysomnography.

REFERENCES


