On the Potential Clinical Relevance of the Length of Arousals From Sleep in Patients With Obstructive Sleep Apnea

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Study Objectives: To assess, in individuals referred for evaluation of obstructive sleep apnea, the potential clinical significance of brief versus longer arousals from sleep.

Methods: Full-night polysomnographic tracings from 100 patients referred for evaluation of obstructive sleep apnea were analyzed to delineate the duration of each arousal event. These data were then correlated to the patient’s subjective perception of sleepiness as estimated by the Epworth Sleepiness Scale (ESS).

Results: A significant relationship (p < .0001, r² = .167) was noted between the frequency of the longer arousals (≥15 seconds) and the ESS. This relationship was significant, but distinctly weaker (p = .004, r² = .073), with the shorter arousals (3-15 seconds); moreover, the association with the brief arousals failed to remain significant (p = .679) after controlling for the effect of the longer arousals.

Conclusions: Individuals with obstructive sleep apnea experience frequent respiratory event associated cortical arousals, many of which are greater than 15 seconds in duration. These longer arousals, which, in this study, constituted 18.4% of all arousals and accounted for 37.5% of the total arousal time, correlate more closely with the ESS than does the frequency or time attributable to the more numerous brief arousals. This suggests that these more-prolonged arousal events may have a greater impact on the restorative aspect of sleep, or on the perception thereof.

Keywords: Arousals from sleep, obstructive sleep apnea, sleepiness

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METHODS

Patients

Patients referred for evaluation of OSA who completed a full-night PSG evaluation without intervention (without continuous positive airway pressure or supplemental oxygen) were eligible for this study. Each had been interviewed and examined by a sleep medicine physician and found to meet criteria for the indications for PSG. Patients were excluded if the study failed to have a minimum of 4 hours of sleep, if periodic limb movement (PLM)-associated arousals were identified at a rate that exceeded 4 per hour, or if alpha intrusion made delineation of arousals impracticable. This report details the findings from 100 consecutive individuals referred to our center who met these criteria. This study protocol was reviewed and approved by the Research Ethics Review Board of University Community Hospital.

Polysomnography

A standard PSG in our lab uses the following protocol: after the patient is acclimated to the facility, they are fitted with EEG (C3/A2, C4/A1, O2/A1, O1/A2), electrooculographic (ROC/A1, LOC/A2), and chin electromyographic (EMG) electrodes for sleep staging, according to the criteria outlined by Rechtschaffen and Kales.2 Electrodes are placed on both legs, as described in American Sleep Disorders Association (ASDA) Atlas Task Force Report,6 to monitor myoclonic activity. Uncalibrated inductive
plethysmography bands are used to monitor chest and abdominal movement, and impedance devices are placed to monitor intercostal muscle activity. A nasal pressure transducer (Pro-Tech™, Mukilteo, WA) is used to monitor airflow. Pulse oximetry (Nonin™, Minneapolis, MN) is assessed at the finger to evaluate oxygen saturation. Electrocardiographic leads are placed to monitor cardiac rhythm (modified lead 2), and concurrent audio, video, and body-position monitoring supplement the other measured parameters.

**PSG Scoring Parameters**

**Respiratory-Event Staging and Scoring**

Apnea was defined as a reduction of the measured parameter of airflow to 10% of baseline or less, with a duration of at least 10 seconds. Hypopnea was defined as any reduction of the measured parameter of airflow with a duration of at least 10 seconds, accompanied by a 4% or greater decrease in measured oxygen saturation, or a contiguous arousal. The Respiratory Disturbance Index (RDI) was the total number of apneas and hypopneas per hour of sleep. The Desaturation Index (DSI) was the number of desaturation events (≥ 4%) per hour of sleep.

**Limb-Movement Staging**

Limb movements and PLMs were scored as defined in the ASDA Atlas Task Force Report. For this study, patients were excluded if PLM-associated cortical arousals (PLM arousals) exceeded 4 per hour.

**Sleep Staging**

Sleep was staged as defined by Rechtschaffen and Kales. Each study was initially scored by Rechtschaffen and Kales criteria, with the scorer ignoring all other events (respiratory events, limb movements). Scoring of Stages 3 and 4 are combined into Stage Delta. After the sleep-stage scoring was initially completed, the study was then scored to delineate the occurrence and duration of each arousal. This was accomplished with the scorer ignoring epoch markers (arousals were marked that may have traversed 2 or more epochs).

**Arousals**

We have traditionally used the criteria proposed by the ASDA. However, in addition to this standard definition from the ASDA, Thomas has suggested that a cortical arousal associated with a respiratory event might include Delta bursts and K-complex arousals (especially if accompanied by an increase in EMG amplitude). We elected to include these parameters in our definition of an arousal.

Therefore, a cortical arousal in this study was defined as follows: (1) an apparent abrupt shift in EEG frequency, which may include Delta bursts or K-complex arousals (if associated with an increase in EMG amplitude) or frequency shifts, which include theta, alpha, and/or frequencies greater than 16 Hz but not spindle; (2) arousal could be scored in rapid eye movement (REM) sleep only if accompanied by an increase in EMG amplitude, but an increase in EMG amplitude alone was not sufficient to score an arousal; (3) an arousal was scored if the change in cortical EEG frequency was ≥ 3 seconds and < 60 seconds—if the period of cortical EEG arousal exceeded 59.9 seconds, it was scored as a “wake event”; (4) any scored arousal was required to have been preceded by at least 5 continuous seconds of any stage of sleep. Though a respiratory event had to be at least 10 seconds in length, they were often noted to have exceeded 30 seconds. As the length of an associated arousal exceeds 15 seconds it may, especially in individuals with an RDI > 30, overlap the beginning of the next respiratory event; in attempting to count every associated arousal event, it was necessary to decrease the required intervening sleep interval from 10 seconds to 5. We reasoned that, if an intervening arousal during sleep can be reliably scored at 3 seconds, a period of intervening sleep could be equally reliably scored if it lasted at least 5 seconds.

Thus, we had 3 categories of cortical EEG arousal: brief arousal—defined as a cortical EEG arousal ≥ 3 seconds and < 15 seconds; long arousal—defined as a cortical EEG arousal ≥ 15 seconds and < 60 seconds; and wake event—defined as a cortical EEG arousal ≥ 60 seconds.

**Calculation of the Number and Duration of Arousal Events**

The beginning and end of each arousal was marked by hand with a light pen on the computer screen. These arousals were then grouped using a custom software report package designed for this purpose by Medcare Systems, Inc (Buffalo, New York). No person at Medcare had access to any of the patient data. After the report package had been developed and the accuracy of the results confirmed by review of template studies (none of which are included in this report), the personnel at Medcare then neither participated in the scoring nor reviewed any of the data for any patient in this study.

Arousals were defined as Brief Arousals, Long Arousals, and Wake Events—both overall and within each stage of sleep. We chose to use, for comparative analysis of events within each stage of sleep, only those values with at least 10 minutes of a given stage of sleep. This was done to avoid the potential for a few events to produce a high index (number of events per hour) in the setting of too brief a time span for assessment. As an example, if we have only 2 minutes of Stage Delta to assess for a given individual, a single arousal during this time will produce an arousal index of 30. With very brief periods of time for analysis, the potential for a few events to obscure the data is high. We therefore felt that some minimum amount of time was needed to allow comparative analyses, and we somewhat arbitrarily used a minimum of 10 minutes at any stage for inclusion in the statistical analyses. This time restriction is applied only to an analysis of the data between stages. When analyzing the data for the total sleep-period time, all arousal events for every individual were included.

We have previously reported comparative scoring of arousals using this paradigm for these same 2 authors. For brief and long arousal indexes, the interscorer reliability, using a Pearson correlation analysis, were 0.93 and 0.96 respectively (p < .0001). For brief and long arousal durations, the correlations were 0.77 and 0.88 respectively (p < .0001). These data confirm a strong correlation for the ability of different scorers to reliably determine the frequency and duration of both brief and long arousals as defined by this protocol.

**Statistical Analysis**

Data are presented as the mean (± SD). These data were ana-
lyzed using SPSS version 12.0 (SPSS Corp, Chicago, IL). Scalar data (arousals, respiratory events) were analyzed using a Pearson correlation, paired-sample t test, or an independent-sample t test. Ordinal data (ESS) were analyzed using a Mann Whitney U test. An initial simple linear-regression analysis was used to identify potential relationships between the ESS and the various arousal parameters in each stage of sleep. A multiple linear regression was used to evaluate the contribution of each sleep variable to models predicting the ESS. A 2-tailed p value < .05 was considered statistically significant.

RESULTS

Patient Demographics

As noted in Table 1, these individuals had moderate sleep apnea (RDI = 30). There was no statistically significant difference between men and women with regard to age, RDI, DSI, PLMs, PLM arousals, or ESS, but the women were more obese than the men.

Sleep Parameters

As noted in Table 2, average sleep-period time was 5.9 hours (354.4 minutes ± 57.3). There was no significant difference in the total sleep-period time, wake time after sleep onset, or in the percentage of any stage of sleep between the men and women in this study.

<table>
<thead>
<tr>
<th>Arousal Index</th>
<th>n</th>
<th>All</th>
<th>Men</th>
<th>Women</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brief Arousals</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Sleep</td>
<td>100</td>
<td>18.9 ± 11.6</td>
<td>53</td>
<td>19.2 ± 12.8</td>
<td>47</td>
</tr>
<tr>
<td>Stage 1</td>
<td>93</td>
<td>43.2 ± 28.8</td>
<td>48</td>
<td>39.4 ± 21.8</td>
<td>45</td>
</tr>
<tr>
<td>Stage 2</td>
<td>100</td>
<td>19.9 ± 13.6</td>
<td>53</td>
<td>20.0 ± 14.7</td>
<td>47</td>
</tr>
<tr>
<td>Stage REM</td>
<td>98</td>
<td>10.7 ± 8.1</td>
<td>51</td>
<td>12.1 ± 9.6</td>
<td>47</td>
</tr>
<tr>
<td>Stage Delta</td>
<td>60</td>
<td>2.0 ± 2.9</td>
<td>26</td>
<td>2.6 ± 3.6</td>
<td>34</td>
</tr>
<tr>
<td>Long Arousals</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All Sleep</td>
<td>100</td>
<td>4.3 ± 4.6</td>
<td>53</td>
<td>5.0 ± 5.7</td>
<td>47</td>
</tr>
<tr>
<td>Stage 1</td>
<td>93</td>
<td>12.2 ± 10.2</td>
<td>48</td>
<td>13.0 ± 11.1</td>
<td>45</td>
</tr>
<tr>
<td>Stage 2</td>
<td>100</td>
<td>3.8 ± 4.1</td>
<td>53</td>
<td>4.5 ± 5.1</td>
<td>47</td>
</tr>
<tr>
<td>Stage REM</td>
<td>98</td>
<td>3.2 ± 5.3</td>
<td>51</td>
<td>3.5 ± 5.1</td>
<td>47</td>
</tr>
<tr>
<td>Stage Delta</td>
<td>60</td>
<td>0.5 ± 0.9</td>
<td>26</td>
<td>0.6 ± 1.2</td>
<td>34</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD. Arousal Indexes are presented as arousals per hour. Sex comparison by independent samples t test. REM refers to rapid eye movement.
DISCUSSION

For the individuals in our study, the number of brief arousals exceeded the number of long arousals by 4:1, yet the ESS score was better predicted with the long arousals. From these data, it would seem that the subjective estimation of daytime sleepiness in patients with obstructive sleep apnea may have more to do with the length of the arousals than with the number of disruptions that occur. If this is true, we may be counting the wrong parameter when it comes to patients with sleep apnea. It may be more pertinent to count not just the number of arousals, but also the length of those arousals, delineating specifically the frequency and effect of these longer arousals (those exceeding 15 seconds in length) on sleep continuity.

There are, however, a number of questions that remain to be answered as pertains to the findings of this study. First, how long is long? Our initial investigation concentrated on those arousals that equaled or exceeded 15 seconds in length because we noted that there was the potential for these arousals to be included in wake after sleep onset (WASO) time and might not be counted as separate arousal events. Therefore, we segregated our arousals into those shorter than 15 seconds in length and those 15 seconds in length or longer. Interestingly, there seems to be a substantive difference in the average arousal length for these 2 subsets, with but limited overlap. The brief arousals averaged 8.0 seconds ± 1.7, whereas the long arousals averaged 22.8 seconds ± 3.6 (p < .0001). Thus, the longer arousals were usually clearly distinct from their shorter counterparts. Nonetheless, our study could not delineate at what point an arousal was likely to begin to produce daytime impairment. If the length of an arousal is important, how long is long? Perhaps future studies might separate arousals into more-finite time periods in an attempt to identify the relative role of arousals of varying length as they correlate with an individual’s symptoms.

A second concern is with the applicability of these data to a more-general population of patients with sleep apnea. Ours is a biased sample. At our center, individuals with more-severe OSA are likely to have a split-night protocol. Therefore, this study tended to select for those individuals with less-severe levels of OSA or those who were unwilling to use continuous positive airway pressure (2 of the 100 individuals refused continuous positive airway pressure, 98 of the 100 failed to meet criteria for a split-night protocol).

The role of the longer arousals in REM sleep seems somewhat limited (r = 0.052). Thus, the longer arousals were usually clearly distinct from their shorter counterparts. Nonetheless, our study could not delineate at what point an arousal was likely to begin to produce daytime impairment. If the length of an arousal is important, how long is long? Perhaps future studies might separate arousals into more-finite time periods in an attempt to identify the relative role of arousals of varying length as they correlate with an individual’s symptoms.

Table 4a—Prediction of the Epworth Sleepiness Scale Score With Simple Linear Regression by Brief and Long Arousals for the Entire Sleep Period

<table>
<thead>
<tr>
<th>Coefficients</th>
<th>n</th>
<th>β(SE)</th>
<th>95% CI</th>
<th>Adj r²</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brief Arousals</td>
<td>100</td>
<td>0.288 (0.045)</td>
<td>0.044-0.233</td>
<td>0.073</td>
<td>.004</td>
</tr>
<tr>
<td>Long Arousals</td>
<td>100</td>
<td>0.419 (0.108)</td>
<td>0.280-0.710</td>
<td>0.167</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

SE refers to the standard error of β; 95% CI = 95% confidence interval; Adj r² = adjusted r²

The effect of the longer arousals in a multiple regression, the association with the brief arousals was no longer significant. (The brief arousals do not improve the ability of the model to predict the outcome.) This relationship was the same for both men and women.

Using this same analysis format and looking at the ability of arousals within each individual stage of sleep to predict the ESS, a significant relationship was identified for brief and long arousals in Stages 1, 2, and REM sleep (Table 5a).

Once again, however, in a multiple-regression analysis including only those arousal indexes that were significant in the simple regressions as independent variables, the significance of the brief arousals in both Stage 2 and REM sleep was lost (Table 5b), and this was unaffected by sex.

Therefore, it is the longer arousals that best account for the relationship to the ESS. These analyses pertain to the longer arousals overall and to those longer arousals in Stages 1 and 2 sleep. The role of the longer arousals in REM sleep seems somewhat more complex. A multiple linear-regression analysis identified a difference between the men and women in this study for the longer arousals in REM sleep.

For the men, the adjusted r² for the longer arousals in Stages 1 and 2 sleep was 0.275 (p < .0001), whereas the REM-associated long arousals had an r² of 0.258 (p < .0001). As shown in Table 6, in a multiple regression model predicting ESS, both remained statistically significant. Thus, the ESS in men is influenced by long arousals overall and to those longer arousals in Stages 1 and 2 sleep. For the group as a whole, the r² for Stages 1 and 2 sleep was 0.275 (p < .0001), whereas the REM-associated long arousals had an r² of 0.258 (p < .0001). As shown in Table 6, in a multiple regression model predicting ESS, both remained statistically significant. Thus, the ESS in men is influenced by long arousals overall and to those longer arousals in Stages 1 and 2 sleep.

For the group as a whole, the r² for Stages 1 and 2 sleep is 0.256, suggesting that the longer arousals of varying length as they correlate with an individual’s symptoms. For the group as a whole, the r² for Stages 1 and 2 sleep is 0.256, suggesting that the longer arousals of varying length as they correlate with an individual’s symptoms.

The linear relationship of the ESS score to the total sleep time (r² = .006) was not significant (p = .524) but was, as might be expected, a negative relationship (the more sleep, the lower the ESS score). This was essentially the same for both men (r² = -0.004) and women (r² = -0.022).

Table 4b—Multiple Regression with Long and Brief Arousals

<table>
<thead>
<tr>
<th>Coefficients</th>
<th>β (SE)</th>
<th>95% CI</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long Arousals All Sleep</td>
<td>0.392 (0.139)</td>
<td>0.187-0.739</td>
<td>.001</td>
</tr>
<tr>
<td>Brief Arousals All Sleep</td>
<td>0.044 (0.055)</td>
<td>-0.088-0.129</td>
<td>.712</td>
</tr>
</tbody>
</table>

Table 5a—Prediction of the Epworth Sleepiness Scale by Brief and Long Arousals According to Sleep Stage

<table>
<thead>
<tr>
<th>n</th>
<th>β (SE)</th>
<th>95% CI</th>
<th>Adj r²</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brief Arousals</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>93</td>
<td>0.068 (0.028)</td>
<td>-0.037-0.073</td>
<td>-0.006</td>
</tr>
<tr>
<td>Stage 2</td>
<td>100</td>
<td>0.238 (0.039)</td>
<td>0.017-0.172</td>
<td>0.047</td>
</tr>
<tr>
<td>Stage REM</td>
<td>98</td>
<td>0.359 (0.063)</td>
<td>0.112-0.360</td>
<td>0.120</td>
</tr>
<tr>
<td>Stage Delta</td>
<td>60</td>
<td>-0.139 (0.230)</td>
<td>-0.706-0.215</td>
<td>0.002</td>
</tr>
<tr>
<td>Long Arousals</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>93</td>
<td>0.528 (0.047)</td>
<td>0.187-0.376</td>
<td>0.271</td>
</tr>
<tr>
<td>Stage 2</td>
<td>100</td>
<td>0.352 (0.125)</td>
<td>0.217-0.713</td>
<td>0.115</td>
</tr>
<tr>
<td>Stage REM</td>
<td>98</td>
<td>0.249 (0.100)</td>
<td>0.053-0.451</td>
<td>0.052</td>
</tr>
<tr>
<td>Stage Delta</td>
<td>60</td>
<td>-0.067 (0.733)</td>
<td>-1.84-1.094</td>
<td>-0.013</td>
</tr>
</tbody>
</table>

SE refers to the standard error of β; 95% CI = 95% confidence interval; Adj r² = adjusted r²; REM refers to rapid eye movement.
split-night study). More men than women present to our center with severe OSA, and, therefore, more men undergo a split-night evaluation. Thus, women tend to be overrepresented in this cohort. Nonetheless, the number of men and women in this study, the similar severity of their sleep apnea, and the equally strong correlation noted in both groups is such that the statistical validity of these data is likely secure.

We might also note that, for the same reasons, our selection bias might alter the severity of the symptoms with which this cohort might present. If the severity of an individual’s OSA correlates with the severity of the daytime symptoms reflected in the ESS, the fact that this study tended to select for individuals with less-severe OSA might also select for individuals with less-elevated ESS scores.

Thus, though the question as to whether the association between long arousals and the ESS might be equally applicable to other patient populations is unknown, we feel that the fact that our cohort had a mean RDI of 30 and a mean ESS of 10.7, which might be considered rather typical of patients presenting with signs or symptoms of OSA, makes it likely that these data will be replicable when assessed across a wider contingent of patients.

Another criticism of our data might be reflected in our limited EEG montage. We used a standard montage (C3/A2, C4/A1, O1/A2, O2/A1) for analysis of cortical arousal events. Some have suggested that extending the montage to include frontal leads might improve the sensitivity of the assessment. Though had we used a more extensive montage, our results might perhaps have been different, the montage used to define a cortical arousal in our study is the standard recommended by the American Academy of Sleep Medicine.

Additionally, we did not measure the effect, or presence, of subcortical arousal events in this study. Subcortical events have been shown to have the potential to cause daytime impairment. Whether subcortical events might have contributed to the subjective assessment of neurocognitive impairment in our subjects is unknown.

Another question that arises is why do the longer arousals, which constitute only 18.4% of all arousals, correlate with the ESS, whereas (after controlling for the effect of the long arousals) the more-prevalent brief arousals fail to do so? Our data do not permit a definitive answer, but we would posit that the effect may be correlated with the amount of uninterrupted sleep obtained. The mean RDI in our study was 30. Any respiratory event associated with an arousal will result in some impingement on the time available for sleep. A brief arousal, which averaged 8.0 seconds in our study, would tend to impact the continuity of sleep far less than the longer arousals which averaged 22.8 seconds.

Though Steptanski initially noted that the number of arousals did correlate with sleepiness in a small group of individuals with OSA, he failed to note whether longer arousals (which he defined as 6-29 seconds) correlated any better or worse than shorter arousals (which he defined as ≤5 seconds). In a reevaluation of that study and those that followed, Stepanski discussed the potential effect of arousals on sleep continuity, as perhaps being more relevant than the simple number of arousals that occur, suggesting that the impairment in subsequent daytime function may be more closely related to the effect of arousals on the length of sleep continuity rather than on the frequency of any disturbance thereof.

The notion that the restorative effects of sleep, or the perception thereof, may be related to sleep continuity has, however, been a matter of debate, with some suggesting that an alternative explanation might lie in the nonrestorative effects of Stage 1 sleep.

Our study did not permit an analysis of the role of a change in the amount of Stage 1 sleep. We would note, however, that the occurrence of an arousal will increase the likelihood of the succeeding epochs being scored as Stage 1 sleep. Thus, not only will the occurrence of an arousal influence the subsequent sleep staging, but the staging paradigm will influence which stage will have been scored when the next arousal occurs. Therefore, the two are not completely independent.

Nonetheless, as has been noted by Punjabi et al, our data would seem to confirm that the most significant association between events that disrupt sleep and the symptoms of daytime sleepiness occurs with nondelta, non-REM sleep (Stages 1 and 2).

As noted, a relationship does exist, in men, between the ESS and the longer arousals that occur during REM sleep. This association was not seen in the women. The amount of REM sleep did not differ between the men and women in this cohort, nor did the RDI or Epworth scores; thus, the reason for this discrepancy is unclear.

Others have noted a potential sex difference in some of the manifestations of OSA. Dancey et al noted that men have a higher RDI than women for any given BMI. They defined respiratory events using a thermistor and did not use contiguous cortical arousals in the definition of a hypopnea; thus, it might be difficult to compare their data with ours, though a similar phenomenon is noted in our cohort. Leech et al have noted that men have longer respiratory events than women. Again, in their study, they used thermistor technology to define a respiratory event and used desaturation to define a hypopnea, with no mention of associated arousals. We measured arousal length but did not measure respiratory-event length. Rota et al have recently noted that women have longer WASO times than men. They used full-night polysomnography, but they defined sleep onset as the first epoch of stage 2 sleep. This definition is likely to effect WASO in that it...
ignores Wake time, which might otherwise be included in WASO using a more-standard definition of sleep onset (3 consecutive epochs of stage 1 or the first epoch of any other stage of sleep). Using this more-standard paradigm, we determined WASO for our cohort (men = 58.6 ± 43.9, women = 64.8 ± 42.0, p = .473). WASO determined using our study paradigm (eliminating any WASO during which an arousal was counted) resulted in similar data (men = 44.8 ± 41.0 minutes, women = 54.0 ± 42.0 minutes, p = .721). Thus, as has been noted by Resta et al, the women had a slightly larger amount of WASO than did the men, but the difference in our cohort did not reach statistical significance.

Shepertycky et al have also commented upon sex differences in OSA, with their study identifying differences in the clinical presentation of OSA between men and women, noting that women more commonly express symptoms of insomnia or depression than do men.

Thus, though others have identified potential sex differences in various parameters related to the OSA, to the best of our knowledge, no one has ever reported on a specific sex difference as pertains to the relationship of these symptoms and the length of cortical arousals associated with respiratory events.

Our study demonstrates that the most significant relationship between the subjective symptoms expressed in the ESS is with the long arousals occurring in Stages 1 and 2 sleep. This was consistent for both men and women. Whether a sex difference will be identified for other parameters (as suggested for the events in REM sleep) will require further study, but we would stress that, for both men and women, by far the most significant association with the ESS was with the longer arousals in Stages 1 and 2 sleep.

Our data would tend to support the sleep-continuity theory, with the finding that the effect of sleep fragmentation on subsequent perceived daytime sleepiness was associated more closely with the duration of the interruption than with the frequency thereof.

We would be remiss not to note that the ESS is perhaps a rather crude subjective assessment of sleepiness, and, though our data would suggest a rather remarkable association between this subjective scale and the longer arousals that disrupt sleep in individuals with OSA, further work is required to determine whether a relationship will be identified with other measures of daytime performance or objectively tested levels of sleepiness.

In summary, though there may be many unanswered questions generated by the findings of this study, the significant relationship between the long arousals and the subjective estimate of sleepiness appears fairly strong and should, perhaps, influence how we look at our PSG data in the future. We would propose that simply counting the number of arousal events that occur may be inadequate and that some assessment of the length of the arousals may need to be included in our analyses.

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REFERENCES


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