Use of Chest Wall EMG to Classify Hypopneas as Obstructive or Central

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Study Objectives: To compare classification of hypopneas as obstructive or central based on an effort signal derived from surface chest wall electromyography (CW-EMG-EF) coupled with airflow amplitude versus classification using The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications (AASM Scoring Manual) criteria; and to characterize hypopneas classified as obstructive versus central using a resistance surrogate.

Methods: CW-EMG was recorded in the eighth intercostal space at the right midaxillary line. Five hypopneas were randomly selected from 65 consecutive adult clinical positive airway pressure titration studies meeting study criteria. A blinded scorer classified the hypopneas based on two groups of signals: Group 1: positive airway pressure flow (PAP flow), chest and abdominal effort, and snoring; or Group 2: smoothed PAP flow (for blinding amplitude but not flattening visible) and effort (CW-EMG-EF). A resistance surrogate (CW-EMG-EF / PAP flow) normalized to a pre-event breath was compared between obstructive and central hypopneas classified by AASM Scoring Manual criteria.

Results: The percentage agreement (Group 1 versus Group 2) was 92% and the kappa was 0.75 (95% confidence interval 0.65 to 0.85). The resistance surrogate was significantly higher in obstructive hypopneas versus central hypopneas during the first and second half of hypopneas. The resistance surrogate (mean ± standard deviation) for the second half of hypopnea was obstructive: 7.59 ± 7.24 versus central: 1.27 ± 0.56, \( P < .001 \). The resistance surrogate increased from the first to second half of hypopnea only for obstructive hypopneas.

Conclusions: CW-EMG provides a useful complementary signal for hypopnea classification and a resistance surrogate based on CW-EMG is much higher in hypopneas classified as obstructive by AASM Scoring Manual criteria.

Keywords: EMG, hypopnea, respiratory effort, diaphragmatic EMG

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BRIEF SUMMARY

Current Knowledge/Study Rationale: The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications (AASM Scoring Manual) classification of hypopneas as central or obstructive does not utilize chest and abdominal effort signals as they often do not reflect the degree of inspiratory effort. When suitably processed, the surface electromyography (EMG) activity of the chest wall provides a measure of inspiratory effort (CW-EMG-EF) that could potentially be useful for hypopnea classification. This study compared hypopnea classification using AASM Scoring Manual criteria versus the combination of airflow amplitude and CW-EMG-EF.

Study Impact: The study suggests that an inspiratory effort signal derived from the surface chest wall electromyography is useful for classification of hypopneas as obstructive versus central.

INTRODUCTION

The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications (AASM Scoring Manual) provides the option of scoring hypopneas as obstructive or central.1,2 Obstructive hypopneas are those with evidence of increased airflow flattening, chest/abdominal effort belt paradox, or snoring during the event. If none of these are present the hypopnea is classified as central.

To our knowledge a systematic analysis of the characteristics of hypopneas classified as obstructive or central by AASM Scoring Manual criteria has not been performed. Of note, the AASM Scoring Manual classification of hypopneas as central or obstructive does not utilize chest and abdominal effort signals as they often do not reflect the magnitude of inspiratory effort. If accurate measurements of airflow and driving pressure are available, classification of hypopneas based on changes in upper airway resistance is possible. In central hypopneas there is a reduction in airflow roughly in proportion to effort, whereas upper airway resistance undergoes minimal change. In obstructive hypopneas, there is an increase in airway resistance often associated with airflow limitation, chest-abdominal paradox, or snoring. Measurement of esophageal pressure to reflect the level of effort3–6 has been used to classify hypopneas, but this type of monitoring is not practical for routine clinical practice. In addition, a study of hypopnea classification using esophageal pressure deflections found that 33% of hypopneas could not be classified due to artifact in the esophageal signal.8 Measurement of the surface chest wall electromyography (CW-EMG) has also been used to reflect inspiratory effort and could provide a potentially useful signal for hypopnea classification.9 When rectified and integrated the inspiratory deflections in the surface CW-EMG (diaphragmatic EMG) signal are highly correlated with esophageal pressure deflections during...
both apneas and hypopneas.\(^7\) In a previous study we demonstrated that the surface CW-EMG signal can be used to help classify apneas as central, mixed, or obstructive\(^8\) but did not analyze hypopneas.

We hypothesized that the rectified and integrated CW-EMG signal coupled with a measurement of flow amplitude would allow classification of hypopneas. Although measurements of deflections in nasal pressure during diagnostic studies provide an estimate of airflow (after a square root transformation), displacements of the cannula during the night or periods of oral breathing make measurement of inspiratory flow during hypopneas in routine clinical studies problematic.\(^9\) However, the flow signal from the positive airway pressure (PAP) device during PAP titration sleep studies provides an accurate estimate of flow as well as the ability to detect inspiratory flattening.\(^7\) Given these considerations, we performed a retrospective analysis of PAP titrations to (1) compare the ability of a measurement of effort from CW-EMG to classify hypopneas versus use of AASM Scoring Manual criteria and (2) to analyze relative changes in airflow and effort in hypopneas classified as obstructive or central by AASM Scoring Manual criteria.

**METHODS**

A CW-EMG signal is routinely recorded in all patients at the UF Health Sleep Center. Consecutive adult sleep studies with PAP titration (including split studies) that were recorded over a 6-month period were retrospectively analyzed. The retrospective analysis was approved by the institutional review board of the University of Florida. The first 65 studies meeting the following criteria were analyzed:

1. Age older than 18 years
2. At least 10 hypopneas on PAP. Hypopneas were scored when there was a \(\geq 30\%\) drop in PAP flow for \(\geq 10\) seconds and the event was associated with a \(\geq 4\%\) arterial oxygen desaturation (acceptable hypopnea definition)\(^7\)
3. Snoring, PAP flow, and chest and abdominal respiratory inductance plethysmography effort belt signals were of adequate technical quality
4. The CW-EMG tracing was of adequate technical quality for more than \(80\%\) of the study. A technically adequate tracing was defined as the ability to see inspiratory bursts during reductions in airflow and absence of a large amount of 60 Hz or electrocardiogram (ECG) artifact obscuring the signal of interest.

A total of 463 consecutive adult sleep studies (PAP titration, and split night) were reviewed to select the required number for analysis. Of these studies 147 had 10 or more hypopneas as well as adequate snoring, PAP flow, and chest/abdominal signals. However, only 65 of these studies had adequate CW-EMG signals. Of the 82 excluded studies, 41 had faulty EMG electrodes (very large 60 Hz artifact), 17 displayed no EMG bursts during hypopneas, 18 displayed minimal EMG bursts obscured by 60 Hz artifact, and 6 had artifact due to frequent premature ventricular contractions or a pacemaker.

Standard polysomnographic techniques were utilized with recording of frontal, central, and occipital electroencephalogram and right and left eye movement derivations (E1-M2, E2-M2), chin derivations, and an ECG channel as recommended by the AASM Scoring Manual.\(^1\) Airflow and leak signals from a laboratory PAP device (Omni-lab, Philips Respirronics, Murrysville, Pennsylvania, United States) were recorded. Un-calibrated chest and abdominal respiratory inductance plethysmography signals were used to detect respiratory effort. A snoring signal (piezo snore sensor, S.L.P. Inc, Elgin, Illinois, United States), pulse oximetry, and right and left anterior tibial EMG were also recorded. The Nihon Khoden Digital polysomnography amplifier (Nihon Khoden PSG-1100, Nihon Khoden, Irvine, California, United States) was used during the studies. All signals (including the CW-EMG) were acquired with a 200 Hz sampling rate and viewed using Polysmith software version 10.0 (Nihon Khoden, Irvine, California, United States).

**Chest Wall Electromyography**

The CW-EMG signal was recorded using the PSG-1100 amplifier as a bipolar AC signal with two adhesive electrodes placed 2 cm apart in the eighth intercostal space at the right midaxillary line. The low- and high-filter settings were 10 and 100 Hz as recommended by the AASM Scoring Manual.\(^1\) An electrode impedance of less than 10K ohm was considered acceptable. Contractions of the intercostal muscles, diaphragm, and possibly other nearby muscles are believed to contribute to the signal.\(^10\)

The selected studies were retrospectively exported to a European data format (EDF) file (containing all the acquired data including the CW-EMG signal) for analysis. A custom software program was used to analyze the CW-EMG and ECG signals in the EDF file and remove ECG artifact from the EMG signal. The ECG artifact was removed by applying an adaptive filter. A filtered version of the recorded ECG signal was subtracted from the EMG signal such that most of the ECG interference was removed, while keeping the loss of information in the EMG as minimal as possible.\(^11,12\) The adaptive filter that was used to minimize the ECG artifact was a symmetric finite impulse response filter with no delay and therefore perfect time alignment with the original EMG signal was possible. The filtered EMG signal was added to the original EDF file time synchronized to the original signals. An amplitude signal reflecting inspiratory effort (CW-EMG-EF) was derived by computing the root mean square power of the ECG-filtered CW-EMG signal using overlapping symmetric time averaging windows of 0.5 seconds, resulting in no time delay. Addition of the two new channels (ECG-filtered CW-EMG signal and CW-EMG-EF) in a time synchronized manner to the original EDF file was performed by reading, analyzing, and writing the entire recording as one operation.

The processed EDF file was then imported into LabChart version 7.3.8 (ADInstruments, Dunedin, New Zealand) for display and further analysis. The sensitivity of both respiratory inductance plethysmography and CW-EMG signals was adjusted for optimal event classification.

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Event Selection
In each study, 5 hypopneas were randomly chosen. The random number function (between specified limits) in Excel 2016 (Microsoft, Redmond, Washington, United States) was used to select an epoch number. The next hypopnea with technically adequate tracings was selected (if not previously selected) until the required number of events were identified. If the randomly selected epoch was after the last respiratory event, another random epoch number was generated. In individuals using a nasal mask, events were excluded if mouth leak was suspected based on a truncation in the expiratory portion of the PAP flow, oral venting was noted on video, or leak increased by more than 10 liters per minute during or just following an event.

Hypopnea Classification
Screen shots of the tracings displayed in LabChart showing either Group 1 signals (snoring, PAP flow, chest and abdominal respiratory inductance plethysmography signals) or Group 2 signals (smoothed PAP flow and the CW-EMG-EF signals) for each hypopnea were made (Figure 1). For Group 2 signals, the smoothed PAP flow (using LabChart smoothing function) was used to allow visualization of the amplitude of airflow, but removed any flattening (if present). Flattening was removed to preserve blinding as airflow flattening is an essential component of classification by AASM Scoring Manual criteria (Group 1 signals). The amplitude of all signals was adjusted to allow optimal determination of hypopnea characteristics. The screen shots were de-identified and copied into Power Point 2016 (Microsoft, Redmond, Washington, United States) for viewing, with each slide showing a single hypopnea using either Group 1 or Group 2 signals. Each file contained either Group 1 or Group 2 slides with hypopneas in random order to prevent comparison of a given hypopnea using the two methods. A single blinded observer classified hypopneas as central or obstructive using files with either Group 1 or Group 2 signals. AASM Scoring Manual criteria for hypopnea classification (obstructive versus central) were used for Group 1 signals. For Group 2 signals, an obstructive hypopnea was defined as exhibiting a decrease in airflow relative to inspiratory effort (CW-EMG-EF) over at least the last half of the hypopnea. To assist in Group 2 classification, plots of inspiratory deflections in the PAP flow and CW-EMG-EF signals as well as the CW-EMG-EF/PAP flow ratio (method described in the following section) for each breath of the displayed hypopnea were included on the same slide as the Group 2 tracings (Figure 1). This figure illustrates a hypopnea scored as obstructive by both Group 1 and Group 2 signals. The Group 1 signals show inspiratory flow flattening and snoring. Group 2 signals show an increase in effort relative to flow in the last part of the event (compared to pre-event baseline). An example of a hypopnea classified as central by both Group 1 and Group 2 signals is illustrated in Figure 2. For simplicity both Group 1 and Group 2 signals are combined. However, the blinded score saw only either Group 1 or Group 2 signals on a given slide.
Flow and Effort Characteristics

The flow and effort characteristics of each selected hypopnea were assessed by measuring the inspiratory deflections in the PAP flow and CW-EMG-EF signals using LabChart. The measurements were normalized using a pre-event breath. For example, if the inspiratory flow on a pre-event breath was 0.4 liters per second and the next breath had an inspiratory flow of 0.2 liters per second, the values were transformed to 1 and 0.5, respectively.

We defined a resistance ratio (RR) as a resistance surrogate for each breath during hypopnea as the ratio of the normalized deflection in the CW-EMG-EF signal divided by normalized deflections in the PAP flow signal. Therefore, the RR of the baseline breath was 1 (1/1). The PAP flow signal without smoothing was used for this analysis. The rationale of the RR definition is that resistance is defined as driving pressure divided by flow and CW-EMG-EF deflections are taken as a surrogate for esophageal pressure deflections.\(^7\) Plots of the normalized inspiratory flow deflections, CW-EMG-EF deflections, and the RR for each breath were plotted using Excel and included on slides showing Group 2 signals.

Analyzing Agreement (Group 1 Versus Group 2) and AASM Scoring Manual Hypopnea Characteristics

The agreement between methods of hypopnea classification (Group 1 versus Group 2 signals) was determined by both kappa analysis and intraclass correlation. Statistical software was used (MedCalc version 17.9.2, MedCalc Software Bvba Ostend, Belgium) for this analysis. The percentage agreement was computed as the number of pairs with agreement \(X\) \(\times 100/\text{total number of pairs.}\)

We also analyzed the flow and effort characteristics of hypopneas that were classified as obstructive or central by AASM Scoring Manual criteria. The RR for the first (RR1) and second half (RR2) of each hypopnea was determined. If there were an odd number of breaths, the middle breath was used in the average for both the first (RR1) and second halves (RR2) of the hypopneas. The RR1 and RR2 ratios for obstructive and central hypopneas were calculated and compared.
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Hypopnea Classification by Chest Wall EMG

Scoring Manual criteria, 82% of the hypopneas were obstructive based on CW-EMG-EF. In

Twelve hypopneas were classified as obstructive by AASM Scoring Manual criteria. The RR2/RR1 ratio (mean ± standard deviation) was 2.57 ± 2.58 for obstructive hypopneas. The RR2/RR1 ratio (mean ± standard deviation) was 0.71 ± 0.45 for NREM hypopneas. The RR2/RR1 ratio was significantly higher for obstructive (2.57 ± 2.58) versus central (1.21 ± 0.45) hypopneas (P < .001). Thus, obstructive hypopneas showed a significant increase in effort relative to flow in the second half of the event compared to the first half, whereas central hypopneas did not exhibit this pattern. A graph of each of the RR values (RR1, RR2, RR2/RR1) for obstructive and central hypopneas is displayed in Figure 4. The mixed analysis of variance showed a significant interaction between the repeated factor (first versus second half) and the group factor (obstructive versus central). Analysis of simple effects using the Student-Newman-Keuls test showed obstructive RR1 and RR2 to differ from the corresponding central RRs (P < .001). In addition, the average obstructive hypopnea RR2 was significantly higher than RR1, but the average central hypopnea RR1 and RR2 did not differ significantly. The RR2/RR1 ratio (mean ± standard deviation) was also significantly higher for obstructive (2.57 ± 2.58) versus central (1.21 ± 0.45) hypopneas (P < .001). Thus, obstructive hypopneas showed a significant increase in effort relative to flow in the second half of the event compared to the first half, whereas central hypopneas did not exhibit this pattern. A graph of each of the RR values (RR1, RR2, RR2/RR1) for each hypopnea is displayed in Figure 5. This clearly shows that obstructive hypopneas had a much higher RR than central hypopneas, although there was overlap in the values. When analyzing RR2, 85% of obstructive hypopneas had a RR2 greater than 2 and 86% of central hypopneas had a RR2 less than 2. An RR2 between 1 and 2 was a region of overlap between central and obstructive hypopneas. The RR2/RR1 ratio was much higher for obstructive than central hypopneas classified by AASM Scoring Manual criteria. The RR2/RR1 ratio

Table 1—Subject demographics in studies with 10 or more hypopneas.

<table>
<thead>
<tr>
<th></th>
<th>Analyzed (n = 65)</th>
<th>Excluded (n = 82)*</th>
<th>Analyzed Versus Excluded***</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>56.6 ± 13.5</td>
<td>57.3 ± 14.1</td>
<td>NS</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>44/21</td>
<td>48/34</td>
<td>NS</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>36.4 ± 7.2</td>
<td>38.9 ± 8.1</td>
<td>P = .06</td>
</tr>
<tr>
<td>AHI (events/h)**</td>
<td>39.3 ± 28.6</td>
<td>39.5 ± 32.4</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation except for sex. * = excluded due to inadequate CW-EMG signal. ** = AHI based on diagnostic study or diagnostic portion of a split study. *** = age and BMI analysis by independent sample t test, sex by chi-square test, and AHI by Mann-Whitney U test. AHI = apnea-hypopnea index, BMI = body mass index, CW-EMG = chest wall electromyography, NS = not significant.

RESULTS

The demographic information for the studies with 10 or more hypopneas including the analyzed studies and the studies excluded due to inadequate CW-EMG signal is shown in Table 1. The patients were middle aged or older with an increased body mass index. The apnea-hypopnea index from the preceding diagnostic study (or diagnostic portion of the split sleep study) was in the moderate to severe range. The mean ± standard deviation number of hypopneas on PAP was 31.9 ± 28.1 during non-rapid eye movement (NREM) sleep and was 0.75 (95% CI 0.71 to 0.80). Of the 325 hypopneas analyzed, 266 were during non-rapid eye movement (NREM) sleep and 59 (18.2%) were central based on CW-EMG-EF. In most of these hypopneas there was evidence of flattening in the PAP flow signal, but only a minor increase in effort relative to flow often clearly visible only at the end of an event (Figure 3). Twelve hypopneas were classified as central by AASM Scoring Manual criteria but obstructive by CW-EMG-EF criteria. In most cases airflow flattening was absent or equivocal but associated with a definite increase in effort relative to flow in the second part of the event (Figure 4).

AASM Scoring Manual Hypopnea Characteristics

The resistance ratios (RR1 and RR2 for the AASM Scoring Manual obstructive hypopneas and central hypopneas) are displayed in Table 3. The mixed analysis of variance showed a significant interaction between the repeated factor (first versus second half) and the group factor (obstructive versus central). Analysis of simple effects using the Student-Newman-Keuls test showed obstructive RR1 and RR2 to differ from the corresponding central RRs (P < .001). In addition, the average obstructive hypopnea RR2 was significantly higher than RR1, but the average central hypopnea RR1 and RR2 did not differ significantly. The RR2/RR1 ratio (mean ± standard deviation) was also significantly higher for obstructive (2.57 ± 2.58) versus central (1.21 ± 0.45) hypopneas (P < .001). Thus, obstructive hypopneas showed a significant increase in effort relative to flow in the second half of the event compared to the first half, whereas central hypopneas did not exhibit this pattern. A graph of each of the RR values (RR1, RR2, RR2/RR1) for each hypopnea is displayed in Figure 5. This clearly shows that obstructive hypopneas had a much higher RR than central hypopneas, although there was overlap in the values. When analyzing RR2, 85% of obstructive hypopneas had a RR2 greater than 2 and 86% of central hypopneas had a RR2 less than 2. An RR2 between 1 and 2 was a region of overlap between central and obstructive hypopneas. The RR2/RR1 ratio was much higher for obstructive than central hypopneas classified by AASM Scoring Manual criteria. The RR2/RR1 ratio

Table 2—Agreement table for classification by Group 1 versus Group 2 signals.

<table>
<thead>
<tr>
<th></th>
<th>AASM Scoring Manual (Group 1 Signals)</th>
<th>Obstructive Hypopnea</th>
<th>Central Hypopnea</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>CW-EMG-EF (Group 2 Signals)</td>
<td></td>
<td>254</td>
<td>12</td>
<td>266</td>
</tr>
<tr>
<td>Central Hypopnea</td>
<td></td>
<td>12</td>
<td>47</td>
<td>59</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>266 (81.8%)</td>
<td>59 (18.2%)</td>
<td>325</td>
</tr>
</tbody>
</table>

Figure 3—Hypopnea classified differently by Group 1 and Group 2 signals.

An example of a hypopnea classified as obstructive by Group 1 criteria but central by Group 2 criteria. While effort rises abruptly after the baseline breathing was restored, effort increased relative to flow only at the very end of the event (dark circle). Plots of the deflections in PAP flow and CW-EMG-EF for each breath along with the resistance ratio (CW-EMG-EF / PAP flow) were included with Group 2 signals and are also illustrated on the right. Chest/Abdomen = respiratory inductance plethysmography effort belt signals, CW-EMG = chest wall electromyography, CW-EMG-EF = effort signal derived from the chest wall electromyography, CW-EMG ECG filter = the CW-EMG after the ECG filter has been applied, ECG = electrocardiogram, PAP = positive airway pressure, PAP flow = the flow signal from the PAP device, Smoothed PAP flow = the PAP flow smoothed to eliminate flattening.

Figure 4—Hypopnea classified differently by Group 1 and Group 2 signals.

An example of a hypopnea classified as central by Group 1 signals and obstructive by Group 2 signals. The PAP flow did not exhibit an unequivocal increase in flattening during the event. However, there was a clear increase in effort relative to flow during the hypopnea. Plots of the deflections in PAP flow and CW-EMG-EF for each breath along with the resistance ratio (CW-EMG-EF / PAP flow) were included with Group 2 signals and are also illustrated on the right. Chest/Abdomen = respiratory inductance plethysmography effort belt signals, CW-EMG = chest wall electromyography, CW-EMG-EF = effort signal derived from the chest wall electromyography, CW-EMG ECG filter = the CW-EMG after the ECG filter has been applied, ECG = electrocardiogram, PAP = positive airway pressure, PAP flow = the flow signal from the PAP device, Smoothed PAP flow = the PAP flow smoothed to eliminate flattening.
was greater than 1 for 91% of the obstructive hypopneas and less than 2 for 95% of the central hypopneas.

**DISCUSSION**

The main finding of this study is that there is a high degree of agreement when hypopneas are classified as obstructive or central by AASM Scoring Manual criteria versus a classification based on inspiratory effort derived from a noninvasive CW-EMG signal coupled with airflow amplitude. Disagreement occurred when there is an increase in effort coupled with equivocal flattening in the flow signal or when inspiratory flattening was associated with a more subtle increase in effort. Analysis of flow and effort characteristics of hypopneas classified as obstructive or central by AASM Scoring Manual criteria showed that obstructive hypopneas did have a much higher surrogate for resistance (RR) and that unlike central hypopneas, the ratio of effort to flow increased significantly in the second half of the events. Thus, AASM Scoring Manual classification of central hypopneas does identify events with no or relatively small increase in a surrogate of resistance compared to pre-event breaths during the event, whereas in obstructive hypopneas a decrease in flow is associated with a larger increase in a surrogate of resistance. The resistance surrogate increased in the second compared to the first half of obstructive hypopneas, but not central hypopneas.

As discussed by other investigators, classification of hypopneas even with invasive measurements is not without ambiguity. As pointed out by Mooney and coworkers, evaluation of inspiratory effort alone is not sufficient for classification of hypopnea. In their analysis, a relative increase in effort (compared with flow) or maintenance of effort at a time of reduced airflow reflects obstruction of the upper airway. Events with a relative decrease in effort can be associated with unchanged resistance (proportional decrease in airflow) or increased resistance (larger decrease in flow relative to effort). The authors concluded that “a relative increase in resistance during a hypopnea may be the most appropriate measure for classifying hypopneas as obstructive, but this requires an invasive measurement not usually available in routine polysomnography.” The goal of this study was to evaluate the utility of

<table>
<thead>
<tr>
<th>Resistance Ratio</th>
<th>Resistance Ratio</th>
<th>RR1 Versus RR2</th>
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<tbody>
<tr>
<td>First Half (RR1)</td>
<td>Second Half (RR2)</td>
<td></td>
</tr>
<tr>
<td>Obstructive (n = 266)</td>
<td>3.99 ± 4.74</td>
<td>7.59 ± 7.24</td>
</tr>
<tr>
<td>Central (n = 59)</td>
<td>1.14 ± 0.51</td>
<td>1.27 ± 0.56</td>
</tr>
<tr>
<td>Obstructive versus central</td>
<td><em>P &lt; .001</em></td>
<td><em>P &lt; .001</em></td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation. Resistance ratio = deflection in CW-EMG-EF / deflection in PAP flow. RR1, RR2 are resistance ratios for the first and second half of hypopneas, respectively (normalized so that the RR of the pre-event breath was 1). AASM Scoring Manual = The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, CW-EMG-EF = effort signal derived from the chest wall electromyography, NS = not significant, PAP = positive airway pressure.
a noninvasive measurement of effort to provide the ability to visualize the flow versus effort relationship during hypopnea. We analyzed both a measure of inspiratory effort and flow amplitude to classify hypopneas as obstructive (disproportionate decrease in airflow). To allow a blinded comparison of classification with AASM Scoring Manual criteria, flattening was removed from the flow signal.

Several studies have evaluated noninvasive methods of classifying hypopneas. Mooney and coworkers found that a relative increase in the inspiratory time was a useful method of identifying high resistance versus low resistance hypopneas.14 Randerath and coworkers6 compared a classification algorithm based on flattening of the inspiratory airflow curve, paradoxical breathing, arousal position (delayed in central), sleep stage (REM favors obstructive), and breathing pattern at the end of the hypopnea (abrupt increase favors obstructive) versus a classification based on esophageal pressure. An overall accuracy of 68% was provided by the algorithm. Of note, they did not consider events without an increased inspiratory effort relative to pre-event breaths to be obstructive hypopneas. Our analysis defined obstructive hypopneas based on a relative increase in effort (compared to flow) over at least the last half of the event.

Luo et al.7 used both esophageal pressure and diaphragmatic EMG (esophageal electrodes) using a multipair esophageal electrode to detect respiratory effort during apnea. Approximately one-third of the central events as assessed by respiratory inductance plethysmography were not central using esophageal pressure deflections and diaphragmatic EMG. There was no difference in the number of central events diagnosed by esophageal pressure and diaphragmatic EMG. In our study, we used surface EMG monitoring because use of an esophageal electrode would not be practical for routine clinical monitoring.

Our study has a number of limitations. First, we used an effort signal based on a surface CW-EMG signal rather than esophageal manometry. Stoohs and coworkers7 compared esophageal manometry and surface diaphragmatic EMG in patients with obstructive sleep apnea. The ECG artifact in the EMG signal was minimized by a gating technique and the signal was rectified and a moving time average was determined. During obstructive events, changes in the EMG signal closely tracked the esophageal pressure signal in most patients. Over the course of the obstructive events the increase in esophageal pressure deflections and the EMG signal were similar as a percentage of baseline. This suggests that the surface EMG signal is an acceptable surrogate for esophageal pressure deflections to detect respiratory effort. However, it is possible that use of esophageal manometry could have changed the results of our study. As mentioned previously, a study of hypopnea classification using esophageal manometry found that a significant number of hypopneas could not be classified due to artifact in the esophageal signal. Thus, there are issues with the gold standard signal in addition to the fact that it requires invasive monitoring. Our results do not prove that the CW-EMG signal is more reliable given the significant percentage of patients did not have an acceptable signal. However, CW-EMG monitoring is noninvasive.

Second, we only evaluated patients on PAP. This allowed a retrospective analysis without concern about the accuracy of flow measurements based on nasal pressure and oronasal thermal flow. A study using an oronasal mask with a pneumotachograph (with simultaneous recording of nasal pressure) and CW-EMG is needed to extend our results to diagnostic studies. Until a study documents the utility of CW-EMG for classification of hypopneas during diagnostic studies, the value of recording CW-EMG for this purpose is unproven. A third limitation of our study was its retrospective nature. Although we identified consecutive patients meeting our study selection criteria, the retrospective nature may have affected our results. Because most of our patients had moderate to severe sleep apnea, the utility of the technique in patients with milder sleep apnea also should be evaluated.

Another limitation of the investigation is that an appreciable number of studies with 10 or more hypopneas during PAP titration that were reviewed for inclusion (82/147) did not have a technically adequate CW-EMG signal. There was no special protocol for changing electrode placement if the CW-EMG signal did not show inspiratory bursts with normal breathing. In addition, because these were routine clinical studies, the CW-EMG electrodes were not replaced if all other signals were satisfactory and the patient was asleep. Half of the studies excluded had inadequate CW-EMG electrode application (very high 60 Hz activity). We would contend that with better electrode application and placement, a higher proportion of patients would have a technically adequate CW-EMG signal. However, a prospective study with a systematic approach to verifying proper CW-EMG electrode placement and replacement of electrodes as needed during the study is essential to determine if the frequency of obtaining a satisfactory CW-EMG signal is high enough for the signal to be clinically useful.

Our study used custom software to remove ECG artifact and derive an amplitude signal reflecting inspiratory effort. In our previous study,8 we used a low-filter setting of 25 Hz to minimize ECG artifact. However, this reduces signal amplitude. To take full advantage of the CW-EMG in routine clinical studies, software vendors would need to include an option of ECG artifact removal, rectification, and integration of the signal.

Given the limitations of the study, a recommendation that the CW-EMG signal be routinely recorded for hypopnea classification is premature. The findings of this study, while promising, need to be followed by additional studies as previously discussed. Furthermore, classification of hypopneas is optional and not performed by many sleep centers. In the authors’ routine clinical practice, it has been found that the CW-EMG signal is most useful for classification of apnea (avoiding classification as central when the events are obstructive).8 Although CW-EMG recording has the advantage of being noninvasive, there are definite issues with 60 Hz artifact (poor electrode application or displacement during the study) as well as selecting the proper location for electrode placement. Documentation of the ability to obtain a good CW-EMG signal in most patients is crucial before the technique can be recommended for routine use. Of note, other sites for recording inspiratory muscle activity might provide a better signal in some patients. ECG artifact in the CW-EMG signal is likely to become a minor problem as an increasing number of sleep recording systems have the ability to reduce the ECG artifact in EMG channels. One may
also question the utility of the CW-EMG signal for hypopnea classification given the good agreement with AASM Scoring Manual criteria using routine sensor signals. That is, what does recording the CW-EMG signal really add to routine sensors? One could contend that the signal is useful when information from the other sensors is equivocal or their signal quality is not adequate for interpretation. If the CW-EMG technique can be refined there is the possibility of using a single effort sensor rather than two effort belts. In addition, using the resistance ratio as a surrogate of resistance may provide useful information about the relative importance of decreasing respiratory effort and increasing upper airway resistance as the etiology of obstructive hypopneas.

In summary, using a signal derived from recording of surface CW-EMG using methods similar to those routinely used to record chin and anterior tibial EMG appeared to be clinically useful for classification of hypopneas. Using this signal and airflow amplitude there was good agreement in classification of hypopneas compared to classification using AASM Scoring Manual criteria based on airflow flattening, chest/abdominal paradox, or snoring. Measurement of a surrogate for resistance (RR) showed that most obstructive hypopneas are associated with an increase in resistance during the event and that central hypopneas were characterized by no or a modest increase in the RR. Prospective evaluation of the CW-EMG-EF signal in a larger group of patients in both diagnostic and PAP titration studies would be useful to establish the clinical utility of this approach.

ABBREVIATIONS

CW-EMG, chest wall electromyography
CW-EMG-EF, effort signal derived from the chest wall electromyography
ECG, electrocardiogram
EDF, European data format
EMG, electromyography
PAP, positive airway pressure
PAP flow, airflow signal from PAP device
RR, resistance ratio (CW-EMG-EF / PAP flow)

REFERENCES


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DISCLOSURE STATEMENT

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