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SCIENTIFIC INVESTIGATIONS

A comparison of 2 visual methods for classifying obstructive vs central hypopneas

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Study Objectives: Rules for classifying apneas as obstructive, central, or mixed are well established. Although hypopneas are given equal weight when calculating the apnea-hypopnea index, classification is not standardized. Visual methods for classifying hypopneas have been proposed by the American Academy of Sleep Medicine and by Randerath et al (Sleep. 2013;36[3]:363–368) but never compared. We evaluated the clinical suitability of the 2 visual methods for classifying hypopneas as central or obstructive.

Methods: Fifty hypopnea-containing polysomnographic segments were selected from patients with clear obstructive or clear central physiology to serve as standard obstructive or central hypopneas. These 100 hypopnea-containing polysomnographic segments were deidentified, randomized, and scored by 2 groups. We assigned 1 group to use the American Academy of Sleep Medicine criteria and the other the Randerath algorithm. After a washout period, re-randomized hypopnea-containing polysomnographic segments were scored using the alternative method. We determined the accuracy (agreement with standard), interrater (Fleiss's κ), and intrarater agreement (Cohen's κ) for obtained scores.

Results: Accuracy of the 2 methods was similar: 67% vs 69.3% for Randerath et al and the American Academy of Sleep Medicine, respectively. Cohen's κ was 0.01–0.75, showing that some raters scored similarly using the 2 methods, while others scored them markedly differently. Fleiss's κ for the American Academy of Sleep Medicine algorithm was 0.32 (95% confidence interval, 0.29–0.36) and for the Randerath algorithm was 0.27 (95% confidence interval, 0.23-0.30).

Conclusions: More work is needed to discover a noninvasive way to accurately characterize hypopneas. Studies like ours may lay the foundation for discovering the full spectrum of physiologic consequences of obstructive sleep apnea and central sleep apnea.

Keywords: hypopnea, obstructive sleep apnea, central sleep apnea

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Rules for defining apneas as obstructive or central are well established. Although apneas are given equal weight in the definition of sleep-disordered breathing, defining and classifying hypopneas remains controversial. Noninvasive, visual methods of hypopnea classification have been proposed by the American Academy of Sleep Medicine and by Randerath and colleagues.

Study Impact: We sought to evaluate the clinical suitability of these 2 methods with the thought that there is likely some merit in simply choosing 1 practical approach and standardizing the work that is being done in this area so as to begin to lay the foundation for discovering the full spectrum of physiologic consequences of obstructive sleep apnea and central sleep apnea and the clinical implications of categorizing hypopneas as obstructive or central.

INTRODUCTION

In the diagnosis of sleep-disordered breathing, the apnea-hypopnea index (AHI) is required to detect the presence and severity of disease. Both apneas (cessation of airflow) and hypopneas (reduction in airflow) are considered in the AHI. The rules for defining and classifying apneas (obstructive, central, mixed) using polysomnography (PSG) channels are well established and generally not controversial.[1] Although hypopneas are given equal weight in the calculation of the AHI, neither the definition nor their classification is straightforward and the current American Academy of Sleep Medicine (AASM) scoring manual does not mandate classification of hypopneas as obstructive or central. As the causes and treatments for obstructive sleep apnea (OSA) and central sleep apnea (CSA) differ, this lack of classification can potentially lead to inappropriate treatment.

The hypopnea has been redefined several times since it was originally described in 1976.[2] In 2007, the AASM published its first manual for scoring sleep events,[3] and the 2012 update to the scoring manual brought recommendations for the definition, detection, and classification of hypopneas that we still use today.[4] The 2012 "recommended" definition for hypopneas was a ≥ 30% reduction in flow from the nasal pressure transducer or positive airway pressure flow (during titration) from pre-event baseline lasting ≥ 10 seconds and accompanied by either a 3% oxygen desaturation or an arousal, while a 4% desaturation with airflow reduction was considered an "alternative" definition. For hypopnea detection, the AASM suggested basing the assessment of airflow reduction

Table 1—Previous studies of different methods of classifying hypopneas.

Authors	Year	Study Size	Definition of Hypopnea	Reference Standard Comparison	Method	Results
Argod et al[25]	1998	13 male participants, 100 hypopneas (74 obstructive, 26 central)	• ≥10 s • Decrease in oronasal airflow > 50% by pneumo-tachograph	Manual assessment of Pes	Pulse transit time measured from the ECG R-wave by 2 observers	CH: sen, 84.6%/80.8%; spec, 98.6%/97.3%; OH: sen, 98.6%/97.3%; spec, 84.6%/80.8%
Morgenstern et al[26]	2010	28 participants, 769 hypopneas	• ≥ 10 s and > 50% amplitude reduction of valid breathing measure from baseline or • ≥ 10 s and > 3% desaturation or arousal	Manual Pes assessment by 2 scorers excluding indeterminate events	Machine learning automatic invasive and noninvasive classifiers derived from Pes signal	Invasive: sen, 0.9; spec, 0.9; PPV, 0.91; NPV, 0.9; Acc, 0.9; noninvasive: sen, 0.72; spec, 0.71; PPV, 0.81; NPV, 0.6; Acc, 0.72
Mooney et al[8]	2012	20 participants, 300 hypopneas	• ≥10 s and nasal cannula airflow signal reduction of 10%–70%	Manual assessment of Pes	Ti and visual assessment of flow limitation using nasalvpressurevtransducer and oral thermistor	Presence of increased Ti or flow limitation to detect obstructive events: sen, 84%; spec, 77%; PPV, 71%; NPV, 88%
Randerath et al[6]	2013	41 participants, 1,170 hypopneas	• ≥10 s and reduction in nasal pressure signal of ≥ 50% with an arousal or 3% oxygen desaturation or • ≥10 s and reduction in nasal pressure of ≥ 30% with a desaturation of ≥ 4%	Manual assessment of Pes	Visual algorithm (**Figure 1**)	Acc, 68%; CH: sen, 77%; spec, 61%; PPV, 60%; NPV, 77%
Berry et al[7]	2018	65 participants, 325 hypopneas	• Does not explicitly say	AASM visual criteria	Chest wall surface EMG and inspiratory flattening of PAP signal	κ of 0.75 (95% CI, 0.66–0.85); ICC, 0.75 (95% CI, 0.71–0.80)

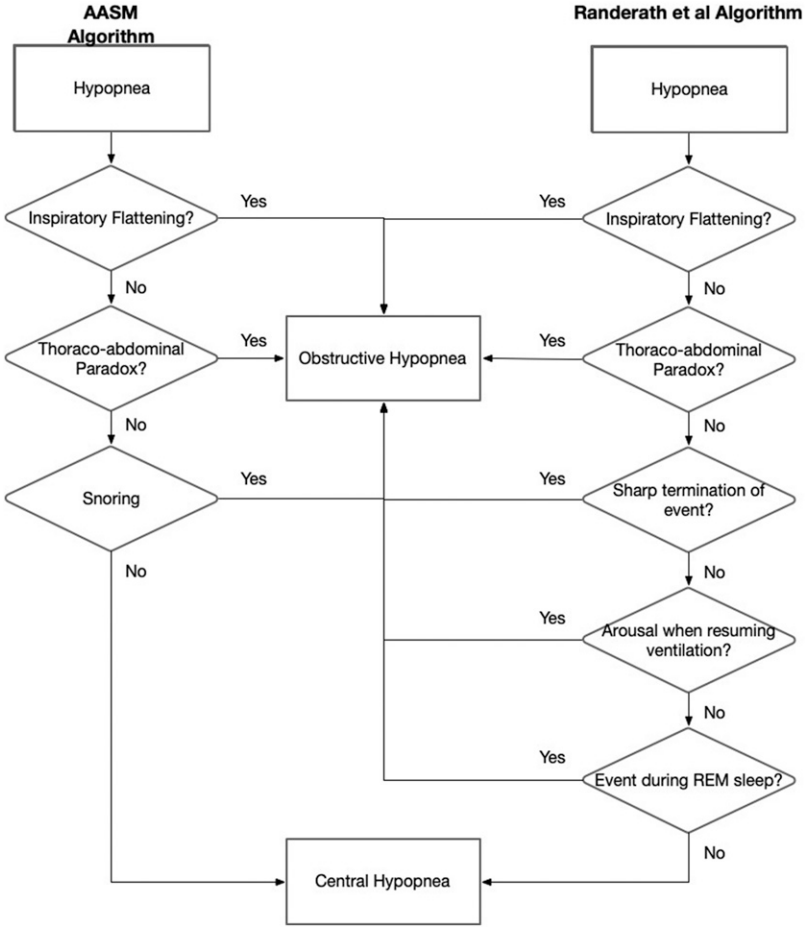
AASM = American Academy of Sleep Medicine, Acc = accuracy, CH = central hypopnea, CI = confidence interval, ECG = electrocardiograph, EMG = electromyography, ICC = intraclass correlation, NPV = negative predictive value, OH = obstructive hypopnea, PAP = positive airway pressure, Pes = esophageal pressure, PPV = positive predictive value, sen = sensitivity, spec = specificity, Ti = inspiratory time.

on the respiratory inductance plethysmography signals or from flattening of the inspiratory portion of the nasal pressure transducer waveform.[4] In discussing detection of respiratory effort as a means of classifying hypopneas, the AASM stated that measurement of esophageal pressure (Pes) was the gold standard for measuring respiratory effort but acknowledged that it was rarely used clinically due to the invasiveness and patient discomfort. Although the manual downplayed the importance of classifying hypopneas as obstructive vs central in most cases, it was recommended that a hypopnea should be classified as obstructive if it meets any of the 3 following criteria: snoring during the event, inspiratory flattening of the nasal pressureor positive airway pressure device flow compared with baseline breathing, and associated thoracoabdominal paradox during the event but not during prebreathing. If none of those criteria are met, then the hypopnea should be classified as central. However, although most clinical sleep laboratories report hypopneas using either the recommended and/or the alternative definition of a hypopnea (although there are both pragmatic arguments related to insurance coverage for therapy and scientific reasons to report both),[5] there is very little agreement about measuring and reporting the classification of hypopneas as obstructive or central using the AASM-recommended method.

Several alternative methods of hypopnea classification have been proposed (**Table 1**), but consensus has not emerged. One reason is that this work uses heterogeneous definitions of hypopnea, thus limiting the general relevance of most of the results, and some methods are not currently clinically applicable. Randerath et al[6] did develop a clinically applicable algorithm for classifying hypopneas as central or obstructive. It showed an overall accuracy of 68% compared with scoring with Pes as the gold standard. However, the authors drew attention to significant limitations of using Pes as a clinical reference.

If it is important to discriminate between obstructive and central hypopneas, then it would be because knowledge of the etiology of these respiratory events in a given patient may help improve the accuracy in differentiating OSA from CSA and in guiding treatment.[9] Believing that it may be clinically relevant to correctly and consistently categorize hypopneas as either obstructive or central, our main aim was to compare the AASM method with the method of Randerath et al[6] (**Figure 1**) in classifying hypopneas as either obstructive or central. We thought it would be important that the visual scoring method provide consistency between observers and result in classifications that would lead to accurate differentiation between patients with OSA vs those with CSA.

Figure 1—Comparison of the AASM method to the Randerath et al[6] stepwise hypopnea classification algorithm.



Adapted from Randerath et al.[6] AASM = American Academy of Sleep Medicine, REM = rapid eye movement.

METHODS

This study was approved by the Mayo Clinic Institutional Review Board (10-007230). We identified 100 hypopneas, a mixture of 50 central and 50 obstructive hypopneas, to serve as the test data set. To build this test data set, we reviewed the PSGs of patients who received diagnostic PSG at the Mayo Clinic Center for Sleep Medicine between 2016 and 2017, dividing patients into 2 cohorts, 1 with an obstructive phenotype and the other with a CSA phenotype. PSG was performed using a digital polygraph (Natus SleepWorks, Pleasanton, CA). We used the "acceptable" electroencephalogram montage, sampling and filter settings, and sleep staging and arousal scoring according to the recent AASM scoring handbook.[10] Respiratory signals were recorded from oronasal thermistors, nasal pressure transducers, and respiratory inductance plethysmography.[10] Hypopneas were scored using the alternative rule (30% reduction in flow signal using the nasal pressure transducer with a 4% oxygen desaturation). Patients were assigned to the obstructive cohort when their clinical diagnosis after PSG was OSA, their PSG demonstrated only obstructive apneic events (no central or mixed apneas), and the body mass index was > 30 kg/m^2. The central cohort had clinical diagnoses after PSG of CSA with Cheyne-Stokes respiration, idiopathic CSA, and opioid-induced CSA; an obstructive apnea index accounting for < 10% of the central apnea index on PSG; and a body mass index < 30 kg/m^2. Patients were excluded from the obstructive cohort if the ejection fraction was < 60% on echocardiography within 6 months of the PSG. Once we obtained our target of 100 hypopneas (50 for each type), we stopped building the cohorts.

When choosing hypopnea events for the test database, we selected 50 hypopnea segments each from the obstructive cohort PSGs (assumed to be obstructive hypopneas) and from the central cohort PSGs (assumed to be central hypopneas). The 100 hypopneas were taken from the diagnostic portion of the PSG and were preferentially selected if the channels were free of artifact, included all necessary information needed to follow the visual classification algorithms, and if there was event-free time before and after the hypopnea so as to allow the interpreter to see the baseline breathing pattern. Some events were not included in the final set because they were nearly identical in appearance to other events within the same PSG. This decision was made because many of the individual hypopneas from a single person's PSG had a near-identical appearance and we wanted to create the most varied set possible in order to enhance generalizability of the results.

Table 2—Characteristics of patients whose PSGs were used to obtain hypopnea segments.

	Age, y	Sex	BMI, kg/m^2	EF	AHI	CAI	OAI	MAI	HI
Central cohort	65	M	26.5	62%	15	10	0	0	5
	72	M	29.7	22%	70	59	0	4	7
	34	F	22.1	Unknown	25	21	0	0	4
	92	M	26.8	70%	62	47	1	1	13
	66	M	29.3	54%	35	28	0	1	6
	46	F	25.4	Unknown	38	32	0	0	6
	72	M	26.5	33%	51	43	1	1	7
Mean (SD)	63.9 (18.9)		26.6 (2.5)		42.3 (19.8)	32.8 (17.7)	0.3 (0.5)	1 (1.4)	6.9 (2.9)
Obstructive cohort	75	M	32.5	Unknown	56	0	30	0	26
	54	M	36.7	Unknown	51	0	24	0	27
	74	M	35.1	Unknown	44	0	33	0	11
	62	F	33.6	Unknown	30	0	21	0	9
	52	F	36.9	Unknown	15	0	7	0	8
	44	M	30.6	61%	42	0	24	0	18
	61	F	35.7	66%	94	0	42	0	52
Mean (SD)	60.3 (11.4)		34.4 (2.3)		47.4 (24.7)	0 (0)	25.9 (10.9)	0 (0)	21.6 (15.5)
P value	.69		< .001		.67				

AHI = apnea-hypopnea index, BMI = body mass index, CAI = central apnea index, EF = ejection fraction, F = female, HI = hypopnea index, M = male, MAI = mixed apnea index, OAI = obstructive apnea index, PSG = polysomnogram, SD = standard deviation.

The images of these 100 hypopnea-containing PSG segments (HCPSs) were deidentified, placed in randomized order, and sent to 2 groups of scorers. Each group had 6 members with varied training backgrounds including 2 registered polysomnographic technologists, 2 sleep medicine fellows, and 2 American Board of Internal Medicine–certified sleep medicine specialists. Although several of the participants had familiarity with the AASM classification criteria, this has not been a part of our practice and therefore none had exercised that knowledge to any extent prior to the study. One group scored the set using the AASM criteria and the other used the Randerath algorithm. For each method, scorers were given standard instructions and were provided with the algorithms shown in **Figure 1.** After a 2-week washout period, rerandomized HCPSs were scored by each group using the alternative method. For each scoring session, we collected the time that it took each individual to complete scoring of the set. We also collected qualitative feedback regarding each scoring method. Individuals and groups were blinded to each other's results and comments.

Statistical analysis

Each rater's HCPS classification using each visual method of classification was compared with the reference standard classification for that hypopnea. Accuracy was determined by measuring how often the scorers rated the HCPSs in a manner consistent with the reference classification. The accuracy was computed as the number of pairs with agreement/total number of pairs and expressed as a percentage. Accuracy results for groups are expressed as mean percentage (standard deviation). Cohen's κ was calculated to examine the interrater agreement for each of the methods. For each method, all 12 raters' responses were examined together and a multirater κ (Fleiss's κ) was calculated to assess agreement between raters—ie, how consistently multiple scorers came to the same conclusion about a given hypopnea segment using each method. Sensitivity (the proportion of times a method correctly detected a specific kind of hypopnea) and specificity (the proportion of times the method identified that the event was not the type of hypopnea of interest) were also calculated.

RESULTS

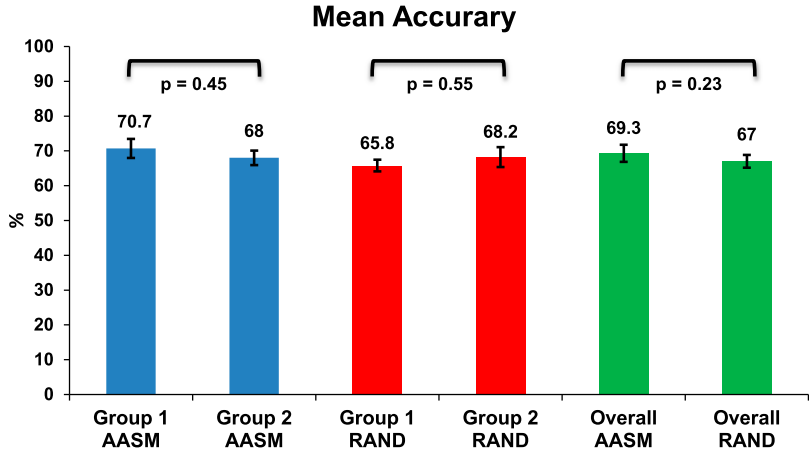
Reference standard cohorts

Our 100 HCPSs were obtained from 14 patients (7 in each cohort). Their characteristics are shown in **Table 2.** Ages were not significantly different (central cohort vs obstructive cohort: 63.9 ± 18.9 years vs 60.3 ± 11.4 years; $P = .69$). Both cohorts were predominantly male. The body mass index was lower in the central cohort, consistent with selection criteria (26.6 ± 2.5 kg/m^2 vs 34.4 ± 2.3 kg/m^2; $P < .001$). As designed, there were significant differences between the central apnea index and obstructive apnea index ($P = .006$ and $P < .001$, respectively). Mixed events did not reach a statistically significant difference between the cohorts ($P = .11$). The hypopnea index was significantly greater in the obstructive cohort (21.6 ± 15.5 vs 6.9 ± 2.9; $P = .05$).

Performance of the 2 methods

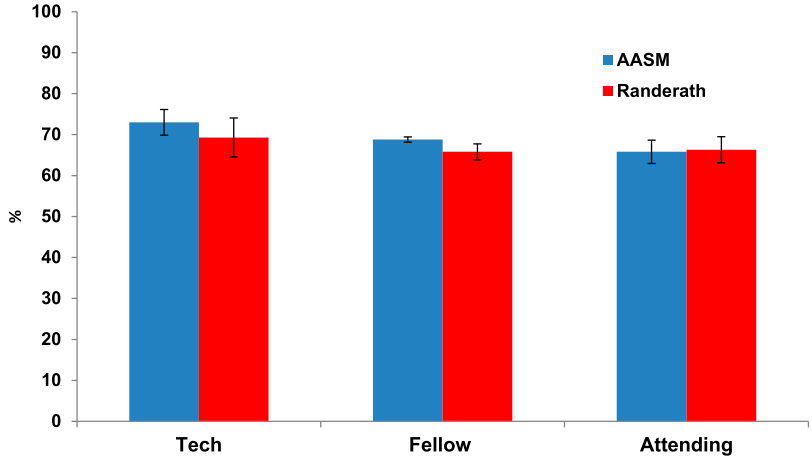
There was no significant difference in accuracy between the 2 methods, either according to which classification method was used first, according to scoring groups, or according to the level of training of the scorer (**Figure 2** and **Figure 3**). Cohen's κ was calculated for each individual rater to see how they agreed with themselves using the 2 methods. Among the 12 raters there was a wide range of Cohen's κ (0.01–0.75), demonstrating that some

Figure 2—Accuracy of the AASM and Randerath methods by scoring group and overall.



AASM = American Academy of Sleep Medicine, RAND = Randerath.

Figure 3—Accuracy of the AASM and Randerath scoring methods by level of training.



AASM = American Academy of Sleep Medicine, Attending = board-certified sleep medicine specialist, Fellow = sleep medicine fellow, Tech = registered polysomnographic technologist.

raters scored the HCPSs very similarly using the 2 methods, while others scored them markedly differently. The mean Cohen's κ was 0.34 (95% confidence interval [CI], 0.11–0.79). Additionally, the level of agreement between the 12 different raters using the same scoring method is of utmost interest. For this assessment, the multirater (Fleiss's) κ was calculated. The multirater κ for the AASM algorithm was 0.32 (95% CI, 0.29–0.36) and for the Randerath algorithm was 0.27 (95% CI, 0.23–0.30). The Cohen's κ for analysis of central events was 0.22 (95% CI, 0.13–0.31) for the AASM method and 0.17 (95% CI, 0.10–0.24) for the Randerath method. These values were significantly lower than the Cohen's κ values of the obstructive cohort (0.48 [95% CI, 0.36–0.59] for AASM and 0.46 [95% CI, 0.36–0.56] for Randerath).

When identifying obstructive events, both methods were equally sensitive (0.80) but were less specific. When identifying central events, both methods were equally specific (0.80) but showed less sensitivity. In other words, when an event was not obstructive, both methods were equally effective in recognizing that the event was not obstructive. When an event was identified as central, it likely was truly a central event.

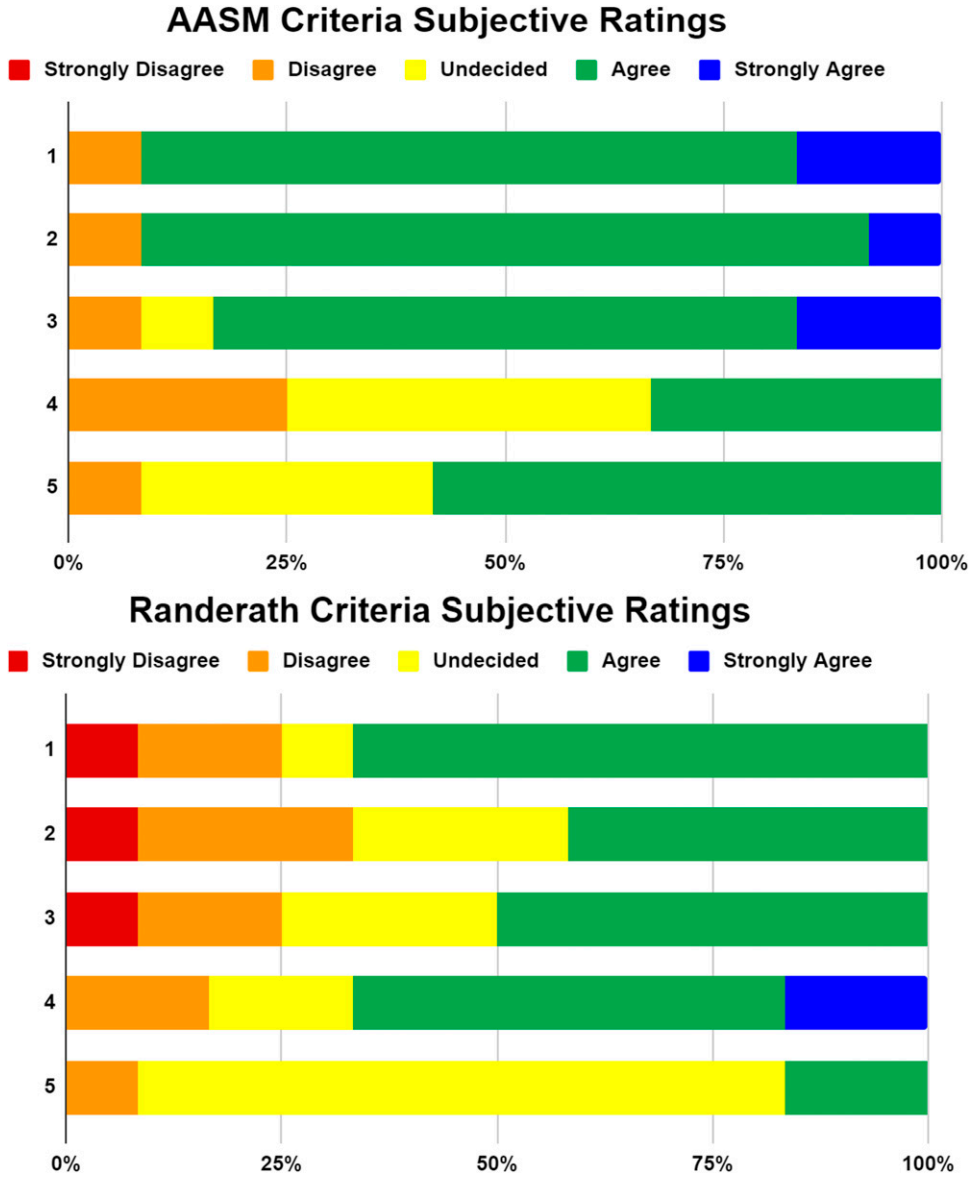
Efficiency and preference between the 2 methods

The raters were slightly faster using AASM (21.1 ± 8.6 minutes) than Randerath (26.9 ± 14.1 minutes), but this difference did not meet statistical significance ($P = .053$). Time taken to score was not dependent upon which method was used first, again supporting that the washout period was adequate to avoid a learning or recall effect. Qualitative feedback favored the AASM method (**Figure 4**). More raters felt that the Randerath criteria led them to score the events differently than they otherwise would have.

DISCUSSION

Both methods allowed for efficient classification of hypopneas as obstructive or central, which may suggest that classification of hypopneas during PSG should be routine, particularly given

Figure 4—Qualitative feedback from raters.



1 = easy to use, 2 = liking the scoring method, 3 = was efficient, 4 = led me to score differently than I otherwise would have, 5 = would use this method in my practice. AASM = American Academy of Sleep Medicine.

that approximately 50%–75% of the AHI is typically composed of hypopneas.[11,12] While generally deemed efficient and easy to use, both visual methods yielded an accuracy of only 67%–69.3% when compared with our reference standards. This similarity in accuracy is not entirely unexpected as both methods rely heavily on the presence of flattening of the inspiratory portion of the nasal pressure transducer signal and the presence of paradoxical breathing for classification of obstructive events. The interrater reliability was only fair using either method, with κ levels around 0.30. This was the first time these scorers had used these methods, and so one might anticipate improvement with practice, training, and iterative calibration. Nonetheless, this suggests that visual methods have limitations in reliability of classification and that automated methods may improve upon reliability by using traditional or novel signals. For example, Berry et al[13] applied signal processing to a surface electromyography to aid in automated classification of apneas. The automated technique, compared with a reference standard of visual scoring, had very good agreement, 89.5%, with a κ statistic of 0.83 and interclass correlation of 0.83. This was referenced against the use of respiratory inductance plethysmography, which is the current gold standard of effort detection during apnea. This method was later tested for classification of hypopneas as obstructive or central, using the AASM criteria as a reference standard.[7] The electromyography method agreed with the AASM method 93.2% of the time with a κ of 0.74 and intraclass correlation of 0.75. The results of the electromyography hypopnea study may offer some proof of concept for using novel signals to aid in automation of respiratory event classification.

Apart from the task of ascertaining an acceptable method of classifying hypopneas, the implications of doing so remain undiscovered. Although others have compared clinical outcomes of the 3% and the 4% rules, literature regarding the clinical impact of classifying hypopneas as obstructive or central is scarce.[14] OSA is associated with increased risk of systemic hypertension,[15] pulmonary hypertension, heart failure,[16] atrial fibrillation,[17] cardiovascular disease,[18] and stroke.[19] Many of these risks are felt to be consequences of negative transthoracic pressure generated by increased inspiratory effort in the setting of an obstructed airway, surges in sympathetic output from the nervous system inflammation, transient increases in blood pressure, and oxidative stress from intermittent hypoxia and reperfusion. CSA has been linked to activation of the sympathetic nervous system and oxidative stress,[20] but the specific physiologic implications are less defined compared with OSA.

Beyond physiology, there are potential clinical implications of classifying hypopneas. A subset of patients with predominantly obstructive events develop complex sleep apnea syndrome, also known as treatment-emergent CSA, the emergence of predominantly central events following commencement of continuous positive airway pressure therapy. But for people with mostly hypopneas and few apneas, failure to identify treatment-emergent hypopneas as central events could result in falsely underestimating the central component and erroneously classifying the disease as obstructive. Although most treatment-emergent CSA resolves over a period of weeks to months, about 30% of patients develop persistent treatment-emergent CSA and may require a respiratory assist device, such as adaptive servoventilation.[21] Another potential consequence of not classifying hypopneas relates to insurance coverage for indicated therapy. The Centers for Medicare & Medicaid Services requires that over half of the events constituting the AHI be central in nature to justify reimbursement of respiratory assist devices. If a patient's breathing events are primarily hypopneas, then it may be crucial to account for the nature of these events from the perspective of insurance coverage. There are also certain disease states where hypopnea classification may be vital. In patients with systolic heart failure, both OSA and CSA are common and may occur in the same patient. Ward et al[22] studied whether using the 3% vs 4% rule to define hypopneas might impact whether a patient was ultimately diagnosed with OSA or CSA using the AASM criteria to classify the hypopneas as obstructive or central. They found that using the 3% rule categorized more people as having mild and moderate sleep apnea but did not influence whether the nature of the disordered breathing was assessed to be predominantly obstructive or central. However, it might be expected that changing the classification of hypopnea might influence differentiation between OSA and CSA in many patients, along with their appropriate treatment.

Perhaps a starting point for developing an acceptable method of differentiation would be to create a foundation on which to base that assessment derived from the existing PSG signals. Both the Randerath and AASM methods incorporate paradoxical breathing and flattening of the nasal pressure transducer. But are there other signals within the existing PSG configuration that may, when added to these entities, improve accuracy? One might wonder if the presence of snoring might be an indicator of respiratory effort and add to the ability to discriminate between central and obstructive events. A major point of difference between the 2 visual methods discussed was the AASM algorithm's use of snoring, which, interestingly, did not seem to allow for a more accurate determination of the nature of the hypopnea. Although we can use the nasal pressure transducer to measure flow limitation, the traditional PSG lacks the ability to directly measure respiratory effort by noninvasive means. In the absence of a direct measurement of effort, Mooney and colleagues[8] found that inspiratory time when assessed in congress with flow limitation as demonstrated by the nasal pressure transducer signal yielded a relatively good sensitivity and specificity with a high negative-predictive value in predicting obstructive hypopneas (**Table 1**). This may suggest that a 2-tiered system of analysis may improve the accuracy of discrimination of hypopneas without directly measuring respiratory effort.

Limitations

We assigned reference standard hypopnea classifications based on the underlying phenotypes of the patients from whom they were derived instead of using Pes to define the references. We agree that it may have been better to use Pes, but several factors influenced our decision. First, pragmatically, we have not used Pes in our clinical practice routines for over a decade, and so it was not readily available. Second, while we considered building a reference library of hypopneas using Pes prospectively, there has not been uniform agreement that using Pes provides a reliable and reproducible interpretation. Randerath et al[6] found that only 64% of hypopneas could be classified using Pes due to various artifacts. In addition, patient acceptance is poor outside of a research setting. In addition, there is reason to believe that our strict criteria did create 2 very different cohorts. Javaheri[23] documented the proportion of obstructive apneas and hypopneas among 20 men with heart failure with reduced ejection fraction and severe CSA syndrome (similar to our CSA cohort). In his study, he found that the overall AHI averaged 57 ± 25 event/h, the central apnea index was 35 ± 24 events/h, and the purely obstructive AHI was only 0.1 ± 0.3 events/h. In a study of cardiac output in patients with heart failure with reduced ejection fraction and Cheyne-Stokes respiration/CSA, Inami et al[24] defined central hypopneas as events with a 50%–90% reduction in tidal volume from baseline for ≥ 10 seconds with in-phase thoracoabdominal motion and without airflow limitation on nasal pressure. Patients were entered into the study if they had an AHI of at least 15 events/h and with a central AHI > 10 events/h that alternated with a waxing-waning pattern of tidal volume.[24] Using these entry criteria, obstructive apneas and hypopneas made up only 19.3% of all events in their patients. However, the criteria used to define our central cohort were more selective, and the patients in our cohort all had an obstructive apnea index of <10% of the total AHI on PSG. It is therefore likely that only a very small percentage of respiratory events in our central cohort would have an underlying obstructive physiology. Finally, we thought that since the main purpose of classification of hypopneas was to ensure diagnostic accuracy, deriving the reference standard hypopneas from cohorts that were clearly defined as either obstructive or central

would likely provide clinically relevant standards. Fortuitously, the accuracy of our scoring methods was not significantly different from the accuracy obtained by Randerath et al,[6] who did use Pes to determine the reference standard. However, before our results are accepted generally, a broader spectrum of patient phenotypes might be tested against reference hypopneas categorized using either Pes or some other physiologic measures (**Table 1**). The Cohen's κ demonstrated more interrater variability in the assessment of central events using either method. This suggests that the difficulty in correct visual categorization of central hypopneas that we found is not an artifact of our cohort construct, but rather indicates that there is a need for more dependable categorization methods or criteria.

Interpretation

This evolving definition of the hypopnea and evolving opinion regarding detection and classification of hypopneas reflect the complex and heterogeneous nature of sleep-disordered breathing disorders. If we have not been able to identify a highly precise and accurate method of classifying hypopneas thus far, then there is likely some merit in simply choosing 1 practical approach and standardizing the work that is being done in this area so as to begin to lay the foundation for discovering the full spectrum of physiologic consequences of OSA and CSA and the clinical implications of categorizing hypopneas as obstructive or central. Like Schrödinger's famous cat, if we leave hypopneas in a box of ignorance and there is no one who dares to define what kind of hypopnea lies there, then the hypopnea ends up both obstructive and central (or neither). This conclusion seems unlikely to match the underlying pathophysiology. The presence or absence of obstruction is not merely an artifact of close observation; it is really there waiting to be found. If it is not possible to imminently have a high accuracy of classification for hypopneas, might the first step be to strive for consistency in defining and evaluating hypopneas?

ABBREVIATIONS

AASM, American Academy of Sleep Medicine
AHI, apnea-hypopnea index
CI, confidence interval
CSA, central sleep apnea
HCPS, hypopnea-containing PSG segment
OSA, obstructive sleep apnea
Pes, esophageal pressure
PSG, polysomnography/polysomnogram

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