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Accuracy of portable devices in sleep apnea using oximetry-derived heart rate increases as a surrogate arousal marker

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Abstract

Purpose Type 3 home study (HS) monitors do not detect cortical arousal-related hypopneas and may therefore underestimate the polysomnography (PSG)-based apnea-hypopnea index (AHI). Our aim was to test the hypothesis that scoring hypopneas using heart rate accelerations as a surrogate marker for cortical arousal (autonomic hypopnea; AnH) improves the accuracy of HS for OSA diagnosis, using PSG AHI as the diagnostic gold standard.

Methods We retrospectively identified patients referred for OSA who underwent complete PSG following an initial inconclusive HS. Respiratory events were scored using AASM research (Chicago) criteria with additional HS scoring for AnH, defined as hypopneas based on flow criteria associated with an increase in pulse oximetry-derived heart rate ≥ 6 beats/min.

Results A total of 178 patients met inclusion criteria, with mean (±SD) HS AHI = 4.4 ± 4.2/h, which increased to 8.5 ± 5.3/h with AnH scoring. The hypopnea arousal index on subsequent PSG was 7.6 ± 7.7/h, with total AHI 15.6 ± 11.9/h. Bland-Altman analysis showed improved agreement between HS and PSG AHI (mean difference 11.2/h (95%CI 33.6, − 11.1) without vs. 7.2/h (95%CI 29.6, − 15.4) with AnH scoring). Overall diagnostic accuracy was improved with AnH scoring as reflected by an increased area under the receiver-operating characteristic curve for AHI thresholds of 10 and 15 events/h.

Conclusions In this retrospective analysis, the diagnostic accuracy of type 3 HS was improved by the inclusion of hypopnea-associated heart rate increases as a surrogate marker of arousal. Prospective studies are warranted to evaluate the impact of AnH scoring on clinical decision-making and patient outcomes.

Keywords Sleep apnea · Ambulatory diagnosis · Autonomic control · Breath-related arousal · Pulse oximetry · Diagnostic accuracy

Abbreviations

AHI	Apnea-hypopnea index
AnH	Autonomic hypopnea
AUC	Area under the receiver operating characteristic curve
ECG	Electrocardiogram
EEG	Electroencephalogram
EMG	Electromyography
EOG	Electrooculography
HAI	Hypopnea with arousal index
HS	Home sleep study
NCM	Negative chronotropic medication
NoNCM	No negative chronotropic medication
ODI	Oxygen desaturation index
OSA	Obstructive sleep apnea
O_2	Oxygen
PAT	Peripheral arterial tonometry
PSG	Polysomnography
PTT	Pulse transit time
PWA	Pulse wave analysis

Summary In this retrospective analysis, the incorporation of hypopnea-associated heart rate increases as a surrogate marker for cortical arousal improved the accuracy of type 3 home studies for the diagnosis of OSA based on subsequent full polysomnography.

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REI	Respiratory event index
ROC	Receiver operating characteristic
TRT	Total recording time
TST	Total sleep time
SpO_2	Blood oxygen saturation

Introduction

Obstructive sleep apnea-hypopnea (OSA) is a highly prevalent condition characterized by complete or partial collapse of the upper airway during sleep [1]. The termination of obstructive events is frequently associated with immediate physiologic responses that include sustained sympathetic activity, reductions in parasympathetic tone, and cortical arousal [2]. Along with intermittent hypoxia and sleep fragmentation, they lead to worse cardiovascular, metabolic, and neurocognitive outcomes [3–5]. The characteristic modulation in autonomic activity following respiratory events was first described on electrocardiogram (ECG) recordings [6]. This cyclic variation of heart rate is frequently observed on portable monitoring tracings and correlates with breath-related arousal [7–9]. While post-obstructive cortical arousals are measured routinely in complete polysomnography (PSG) and contribute to diagnosis and clinical management, autonomic activity is less commonly measured. Heart rate increases are one marker of autonomic activation which could potentially improve diagnosis and clinical management of OSA, especially using sleep devices which do not record electroencephalogram (EEG) activity.

Full night in-laboratory PSG is the gold standard exam for OSA and yields the apnea-hypopnea index (AHI), a key measure in therapeutic decision-making. However, PSG is costly and time-consuming since it requires continuous monitoring by a trained technician. Portable monitoring, which has the potential to greatly reduce diagnostic costs and patient wait times, has come into increasing use. Type 3 home sleep studies (HS), which include a minimum of four channels, have been used successfully for unattended diagnostic studies, with good diagnostic accuracy compared to PSG in patients with high pretest probability of OSA and with minimal comorbidities [10, 11]. However, an important limitation is that HS do not record EEG and electrooculography (EOG), and so do not provide identification of sleep-wake state and arousals [12]. This has two important implications: total sleep time (TST) cannot be measured and respiratory events meeting flow reduction criteria for hypopneas but associated with microarousals rather than O_2 desaturation are not identified. Previous studies have demonstrated that therapeutic decision agreement between home recordings and PSG is lower specifically in patients who have mild-moderate OSA [13]. Thus, in OSA patients with events predominantly characterized by hypopneas with respiratory-related arousal, a surrogate marker of arousal which could be easily integrated into available portable devices could improve the agreement of HS with PSG AHI [14, 15].

To date, there have been several reports demonstrating the utility of autonomic markers in OSA diagnosis [16]. However, not all markers can be easily incorporated into portable devices. The variation of the heart rate after obstructive events can be either analyzed with ECG recording (R-R interval or spectral analysis) or with a pulse oximetry-derived signal [17, 18]. Diagnostic algorithms using automated heart rate variability calculations in unattended HS have shown good correlation compared to PSG but have not specifically evaluated event-by-event correlation with EEG arousal [11, 19]. Adachi et al. evaluated the diagnostic value of visual pulse oximetry heart rate increases in identifying arousals and found that it accurately identifies breath-related arousal with a sensitivity of 88% and a specificity of 86% [7]. To our knowledge, no study has yet formally evaluated the diagnostic accuracy of type 3 HS using pulse oximetry-derived heart rate increases as a surrogate marker of cortical arousal. In this retrospective analysis, we hypothesized that the incorporation of autonomic hypopneas (AnH, hypopnea associated with increase of ≥ 6 bpm) improves the accuracy of type 3 HS, using the PSG AHI as the diagnostic gold standard, in a population with an inconclusive OSA first test [7]. We also investigated the impact on AnH counts of negative chronotropic medications and comorbidities which could attenuate the heart rate responses to respiratory events.

Methods and materials

Participants

This study was performed with the approval of the McGill University Health Centre Research Institute Ethics Board. In a retrospective chart review of the Sleep Laboratory, we identified all patients who had undergone both a type 3 HS and an in-laboratory diagnostic PSG between January 2010 and August 2013. The inclusion criteria were: patients with a moderate-high pre-test probability of OSA following evaluation in our sleep clinics, in whom an initial type 3 HS was considered by the treating physician to be inconclusive, and who underwent subsequent in-laboratory full PSG. With the objective of evaluating AnH scoring specifically in non-diagnostic home recordings, we excluded patients with an initial HS considered by the treating physician to be conclusive for OSA, patients with a type 3 HS AHI ≥ 30 and patients with previously diagnosed or treated OSA. We also excluded patients whose medical chart was unavailable for review and subjects in whom either the HS was considered technically inadequate (nasal pressure airflow signal or SpO_2 data present for < 50% of the recording) or in whom HS recording time or PSG total sleep time was < 240 min.

Protocol

All HS were performed using either Embletta or Embletta Gold recorders (Natus, Embla, Mississauga Ont), with recording of finger oximetry, nasal cannula pressure signal, ribcage and abdominal respiratory belts and body position. Patients received instruction in the laboratory on how to apply the device, then self-recorded at home and returned the device for analysis. PSG recording using a Stellate PSG system with Harmonie v 7.0 software (Natus Inc., Mississauga, Ont.). Recording included 6 EEG channels (C3, C4, F3, F4, O1, and O2), EOG, submental electromyography (EMG), bilateral tibialis anterior and extensor digitorum EMG, and digital video. Respiratory inductance plethysmography was used for thoracoabdominal motion, and nasal pressure cannula measured airflow. Oxygen saturation (SpO_2) was continuously monitored with a finger oximeter. Both HS and PSG equipment use Nonin (XPOD 3012 or 3012LP) oximeter technology.

Scoring

Both PSG and type 3 HS sleep studies were scored manually by experienced polysomnographic technologists using AASM 2007 criteria, except for respiratory events which were scored using AASM research (Chicago) criteria [20]. Hypopneas were scored as an airflow decrease more than 50% from pre-event baseline for more than 10 s or with any clear airflow reduction accompanied by > 3% oxyhemoglobin desaturation or microarousal. Since EEG is not available on type 3 HS, we created an autonomic hypopnea (AnH) tag for every clear decrease in airflow (≥ 30% reduction for ≥ 10 s) associated with increase in pulse oximetry-derived heart rate ≥ 6 beats/min, as a surrogate marker for arousal. AnH were scored manually and integrated into the final AHI in the HS autonomic scoring protocol.

Statistical analysis

Demographic and clinical variables were summarized using standard descriptive statistics (mean ± standard deviation). PSG and HS sleep variables were compared using standard *t* test. Agreement and diagnostic accuracy measurements were performed in accordance with previous portable monitoring evaluation recommendations [12]. AHI mean difference between level 3 studies with or without AnH scoring and PSG studies was analyzed. Limits of agreement were calculated using Bland and Altman analysis. Diagnostic accuracy with respect to sensitivity, specificity, positive predictive value, negative predictive value, and the values for area under the receiver operating characteristic (ROC) curve (AUC) were calculated for the different polysomnographic AHI cut-off points (≥ 5, ≥ 10, and ≥ 15). Subgroup analyses were performed to evaluate the impact of medications and comorbidities. All analyses were performed on IBM SPSS Statistics 25.

Results

Study participants

During the period studied, we identified 244 patients who had undergone both HS and diagnostic PSG. Figure 1 outlines the selection process of the 178 patients included in the analysis. The mean age was 49 ± 13 years and 50% was female. Subject clinical characteristics are shown in Table 1. In the cohort, 21 patients were on cardiac negative chronotropic medication (NCM) and one had a permanent pacemaker. Twenty-two patients met our criteria for having cardiovascular disease defined as a history of at least one of the following: coronary artery disease, arrhythmia, heart failure, previous stoke, and valvular heart disease.

Sleep study results

Sleep and respiratory data from PSG and HS are shown in Table 2. The mean time interval between the two tests was 175 ± 145 days. The PSG total recording time (TRT) was 443 min and TST was 345 min. The type 3 HS TRT was 435 min.

In this group of patients, the AnH index alone detected more than half of PSG hypopneas with arousal normally missed on portable monitoring (AnH index: 4.4 events/h, PSG hypopnea arousal index 7.6 events/h) (Table 2). The HS AHI doubled with the inclusion of autonomic hypopnea scoring but was still significantly lower than the PSG AHI. These differences were less marked when total recording time rather than total sleep time (Respiratory Event Index, REI) was used in the calculation of PSG AHI, but were still significantly different. To approximate type 3 recording conventional scoring, we divided PSG total number of hypopneas with arousal by TRT (HAI TRT) and subtracted it from the REI (REI-HAI). The PSG REI-HAI was 6.2 events/h which was still significantly higher than the HS conventional AHI of 4.4 events/h. This was explained by a higher apnea index and oxygen desaturation index (ODI) on the PSG studies.

Agreement

Bland-Altman analysis (Fig. 2) demonstrated improved agreement between HS and PSG with the inclusion of AnH scoring, based on the smaller mean difference between PSG and HS AHI with (7.1 events/h) vs without (11.2 events/h, p 0. 01) AnH scoring. The Bland-Altman agreement between HS and PSG was further improved when the PSG REI (AHI using

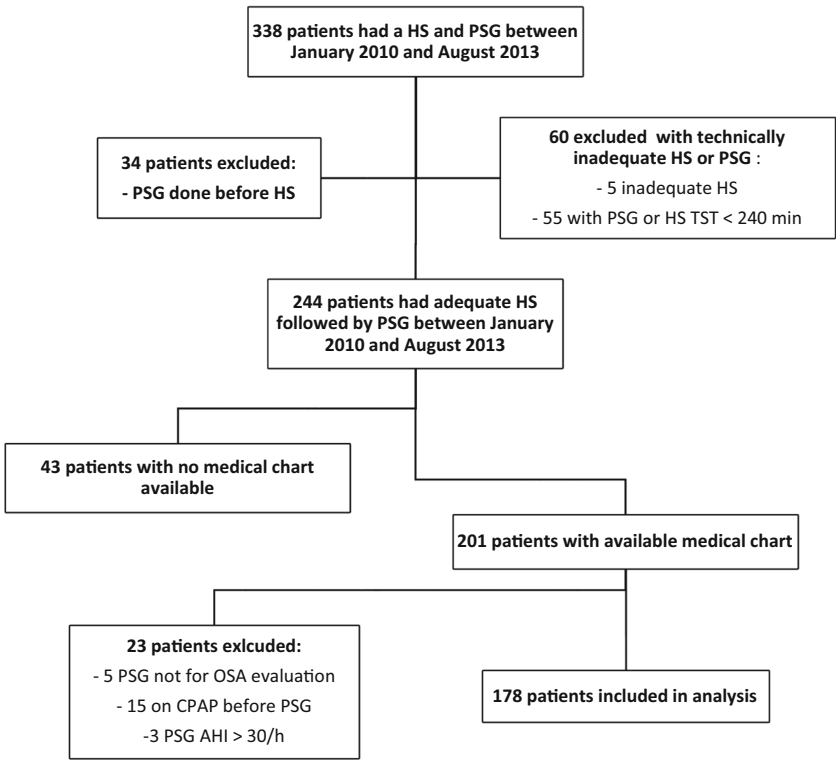

Fig. 1 Flow chart demonstrating selection of patients for inclusion in the retrospective analysis

TRT) was used with the mean difference falling to 3.6 (95% limits of agreement 20.5, − 12.8) with versus 7.6 (24.4, − 9.3) without AnH scoring.

Effect of negative chronotropic medication on autonomic arousal

In the 21 patients receiving NCM, 19 were taking β-blocker and 2 were on non-dihydropyridine calcium-channel blockers. Respiratory variables for subgroups with and without NCM (NoNCM) are shown in Table 3. HS maximal heart rate was significantly higher in the NoNCM group (99 vs 92 bpm), but the mean (67 vs 68 bpm), and minimum heart (52 vs 54 bpm), rate did not differ. The inclusion of AnH increased the HS AHI by 4.8 events/h among NoNCM group compared to only 2.1 events/h in the NCM group [p 0.01]. The HS AnH index was significantly closer in value to the PSG-based hypopnea arousal index in the NoNCM group than for the NCM group. The AHI mean difference between PSG and HS was again significantly lower with the autonomic scoring protocol compared to the conventional protocol in patients with NoNCM (11.4 vs 6.6 events/h). However, this difference was absent in the NCM subgroup.

Table 1 Patient clinical characteristics

Comorbidities		**Sleep symptoms**	
Hypertension, *n* (%)	59 (33)	Snoring, *n* (%)	150 (84)
Coronary artery disease, *n* (%)	13 (7)	Epworth Sleepiness Score	10 ± 6
Arrhythmia, *n* (%)	6 (3)	Sleepiness, *n* (%)	147 (83)
Pacemaker, *n* (%)	1 (1)	Witnessed apnea, *n* (%)	85 (48)
Cardiovascular disease[a], *n* (%)	22 (12)	Choking, *n* (%)	62 (35)
Diabetes, *n* (%)	15 (8)	Nocturia, *n* (%)	113 (63)
Hypothyroidism, *n* (%)	25 (14)	Leg movements, *n* (%)	29 (16)
Obstructive pulmonary diseases, *n* (%)	39 (22)	Habitual sleep duration (h)	7 ± 2
Negative chronotropic medication		**Physical exam**	
β-blocker, *n* (%)	19 (11)	Body mass index, kg/m^2	30 ± 6
Non-dihydropyridine ca$^+$-channel blockers, *n* (%)	2 (1)	Mallampati score	3 ± 1

[a] At least one of the following: coronary artery disease, arrhythmia, pacemaker, heart failure, stroke, and valvular heart disease

Table 2 Sleep characteristics

HS		PSG		*p* value*
TRT (min)	435 (72)	TRT (min)	443 (45)	0.16
		TST (min)	345 (52)	< 0.01
		Sleep Efficiency (%)	78 (11)	
		WASO (min)	69 (41)	
		# Awakenings	25 (13)	
		# Stage changes	117 (44)	
		Arousal Index *(n/h)*	26 (14)	
		% N1	9 (5)	
		% N2	60 (12)	
		% N3	17 (11)	
		% Stage REM	15 (7)	
		% TST supine	40 (29)	0.57
%TRT supine	41 (27)	% TRT supine	31 (24)	< 0.01
AHI with AnH included *(n/h)*	8.5 (5.3)	AHI *(n/h)*	15.6 (11.9)	< 0.01
		REI (AHI with TRT) *(n/h)*	11.9 (8.7)	< 0.01
Autonomic Hypopnea Index *(n/h)*	4.1 (3.6)	Hypopnea with arousal index (HAI) *(n/h)*	7.6 (7.7)	< 0.01
		HAI w/ TRT *(n/h)*	5.7 (5.5)	< 0.01
Conventional AHI (without AnH) *(n/h)*	4.4 (4.2)	AHI–HAI *(n/h)*	8.0 (7.7)	< 0.01
		REI–HAI with TRT *(n/h)*	6.2 (6.0)	< 0.01
Apnea Index *(n/h)*	1.6 (2.3)	Apnea Index *(n/h)*	4.5 (6.5)	< 0.01
		Apnea Index w/ TRT *(n/h)*	3.6 (5.2)	< 0.01
4% ODI *(n/h)*	2.9 (3.4)	4% ODI *(n/h)*	4.1 (5.2)	< 0.01
		4% ODI w/ TRT*(n/h)*	3.2 (4)	0.18
SaO_2 mean *(%)*	95 (2)	SaO_2 mean *(%)*	93 (5)	< 0.01
SaO_2 nadir *(%)*	86 (6)	SaO_2 nadir *(%)*	86 (6)	0.93

Values are expressed as mean (SD); *p value paired t test

TRT total recording time, *TST* total sleep time, *WASO* wake after sleep onset, *N1* stage 1 sleep, *N2* stage 2 sleep, *N3* stage 3 sleep

Indices for HS calculated using TRT, for PSG calculated using TST unless otherwise specified: *AHI* apnea-hypopnea index, *REI* respiratory event index (AHI calculated with TRT rather than TST), *HAI* hypopnea with arousal index, *AnH* autonomic hypopnea, *ODI* oxygen desaturation index, *SaO_2* saturation

Effect of comorbidities on autonomic arousal

We did not identify any effect of comorbidities independent of NCM on AnH counts, although diabetic patients tended to have lower AnH indices. Specifically, in 10/15 diabetic patients with NoNCM, the mean AnH index was 2.3 compared to 4.1 events/h in 157 patients with NoNCM ($p = 0.08$). In the 22 patients with cardiovascular disease, 16 patients were on NCM.

Home study diagnostic accuracy

The diagnostic accuracy of portable monitoring with and without AnH scoring for different AHI cut-off values is shown for the entire patient group in Table 4. Diagnostic sensitivity increased substantially with AnH scoring while specificity was somewhat reduced. The area under the receiver operating characteristic curve (AUC) reflecting overall diagnostic accuracy increased with AnH scoring for the AHI ≥ 10 and 15/h threshold values. The most notable improvement in accuracy measures was for the AHI ≥ 10 threshold value while no benefit was observed with AHI ≥ 5.

The data for the NoNCM and NCM subgroups are shown in Tables 5 and 6, respectively. Consistent with the findings above regarding the impact of NCM on autonomic arousals, there was no change in diagnostic accuracy by any parameter in the NCM group, while the values for the NoNCM subgroup were slightly improved compared to those for the entire group (Table 5 vs Table 4).

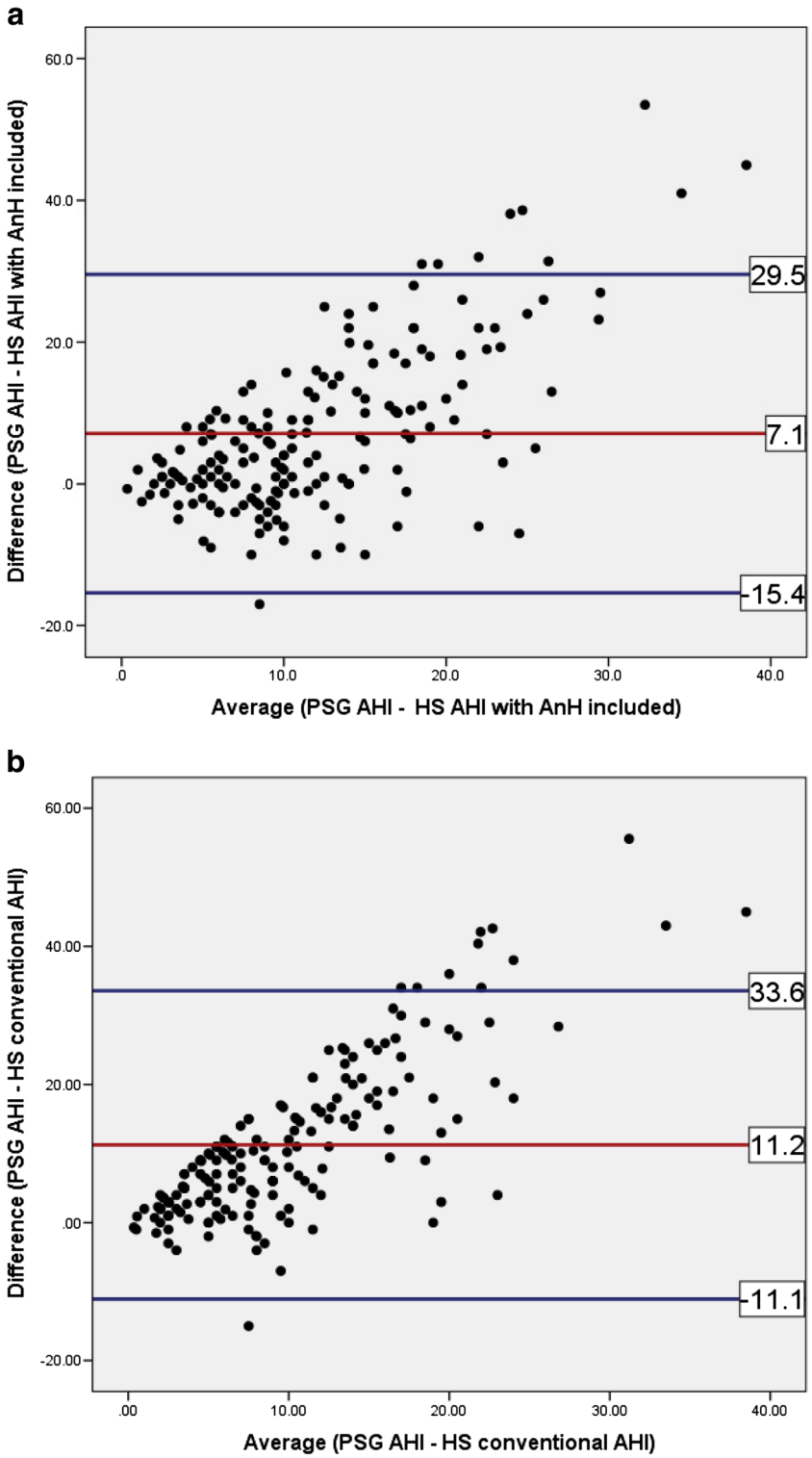

Fig. 2 Bland-Altman plot for agreement between PSG-based AHI and home study-based AHI with (**a**) and without (**b**) autonomic hypopnea (AnH) scoring

Discussion

Type 3 HS are playing an increasing role in OSA diagnosis. HS offer a simplified technology with good AHI agreement compared to PSG in patients with a high pretest probability of OSA [10] and moderate to severe OSA, with a predominance of apneas and hypopneas with desaturation [21, 22]. However, to date, few studies have specifically evaluated the diagnostic accuracy of type 3 HS in patients with mild to moderate OSA or patients with a predominance of respiratory-related arousal events [23]. Notably, Masa et al. reported that the therapeutic decision agreement between type 3 HS and PSG was much

Table 3 Effect of chronotropic medications on sleep respiratory variables

Study groups *N*	Total 178	NCM 21	NoNCM 157	*p* value*
Conventional HS AHI *(n/h)*	4.4 (4.2)	7.1 (7.0)	3.6 (3.6)	0. 01
HS AHI with AnH included *(n/h)*	8.5 (5.3)	9.2 (7.8)	8.4 (5.0)	0.51
Autonomic Hypopnea Index *(n/h)*	4.1 (3.6)	2.1 (2.3)	4.4 (3.6)	0. 01
PSG AHI *(n/h)*	15.6 (11.9)	19.8 (17.4)	15.0 (10.8)	0.08
PSG Hypopnea with arousal index *(n/h)*	7.6 (7.7)	9.4 (10.6)	7.3 (7.2)	0.23

Values are expressed as mean (SD); *p value: NCM vs NoNCM, independent t test

NCM negative chronotropic medications, *AHI* apnea-hypopnea index, *HAI* hypopnea with arousal index, *AnH* autonomic hypopnea

lower in patients with AHI < 30 events/h, which suggests that a higher number of false negative HS are present in this subgroup [13]. Mild-moderate OSA is very prevalent and may present with typical OSA symptoms leading to diagnostic testing with HS [24]. Approaches to increase the diagnostic accuracy of HS in this group are highly relevant [25].

Previous studies have assessed surrogate markers of cortical arousal that could improve HS diagnostic accuracy. For instance, the Spanish group assessed hyperventilation following flow reduction as a surrogate marker of arousal but did not find any changes in OSA diagnostic agreement with PSG [26]. Pulse transit time (PTT), pulse wave analysis (PWA), and peripheral arterial tonometry (PAT) are other measurements of autonomic activity that correlate with cortical arousals [27, 28]. Vat et al. demonstrated that HS conventional scoring showed better agreement with PSG AHI than scoring with a PWA drop algorithm, which overestimated EEG arousals [27]. Nevertheless, more recently, PTT has been shown to be increased in patients with inspiratory flow limitation [28]. These results suggest that autonomic markers may be more beneficial in specific subgroups. To our knowledge, ours is the first study to evaluate AnH as a surrogate marker of arousal to improve the diagnostic accuracy of type 3 HS in a population with mild to moderate OSA.

AnH scoring approximately doubled HS AHI values, with the mean AnH index = 4.1 ± 3.6 compared to the PSG hypopnea with arousal index of 7.6 ± 7.7 or 5.7 ± 5.5 events/h when the PSG index was calculated using total recording time (Table 2). These findings suggest that AnH scoring detected slightly more than half of hypopnea with arousal events not identified by conventional HS scoring. This is concordant with the results of Ayappa et al. who found that approximately 30% of hypopneas are missed (i.e., not meet scoring criteria) without cortical arousal monitoring, while over 50% of those missed events were identified with an autonomic arousal marker [29].

The differences between HS and PSG indices were reduced when PSG values were calculated using total recording time rather than total sleep time (Table 2, Bland-Altman analysis), as expected given the mean sleep efficiency of 78 ± 11% on PSG. However, PSG indices (REI-HAI, AI, and ODI) remained slightly higher than HS indices, and this was not accounted for by differences in supine recording times (Table 2). We conclude that this likely represents night-night variability, and potentially a contribution of other factors such as minor changes in weight or upper airway congestion, given the relatively long mean time between the two recordings. Indeed, Prasad et al. demonstrated on consecutive HS recording nights that night-to-night variability

Table 4 Diagnostic accuracy of HS vs PSG for the studied population (n = 178)

PSG AHI cut-off	PSG AHI ≥ 5		PSG AHI ≥ 10		PSG AHI ≥ 15	
n	150		111		73	
HS Scoring Method	Conventional	AnH included	Conventional	AnH included	Conventional	AnH included
Sens (%)	42	77	14	49	7	17
Spe (%)	89	57	94	76	99	94
PPV (%)	95	91	80	77	83	68
NPV (%)	22	32	40	47	60	62
AUC	0.66	0.61	0.54	0.61	0.53	0.56

Sens sensitivity, *Spe* specificity, *PPV* positive predictive value, *NPV* negative predicitive value, *AUC* area under the receiver operating characteristic curve

Table 5 Diagnostic accuracy of HS vs PSG for NoNCM patients ($n = 157$)

PSG AHI cut-off	PSG AHI ≥ 5		PSG AHI ≥ 10		PSG AHI ≥ 15	
n	133		97		62	
HS Scoring Method	Conventional	AnH included	Conventional	AnH included	Conventional	AnH included
Sens (%)	40	78	11	49	2	14
Spe (%)	91	54	97	75	99	94
PPV (%)	96	90	84	76	50	60
NPV (%)	22	31	40	48	61	63
AUC	0.66	0.66	0.54	0.62	0.50	0.54

is proportionally higher in patients with mild to moderate OSA [30]. In addition, it is possible that a cut-off of 6 bpm heart rate increase was not sufficiently sensitive [7]. Azarbarsin et al. demonstrated a linear relationship between the HR response and the arousal intensity [8]. A lower cut-off might therefore increase the diagnostic accuracy in patients with low arousal response.

Bland-Altman analysis demonstrated an improvement in HS and PSG agreement with AnH scoring based on a reduced mean AHI difference with a similar limits of agreement range. As shown in Tables 4 and 5, AnH scoring improved the diagnostic sensitivity and negative predictive value, and also slightly improved the AUC values from ROC analysis, reflecting overall improvement in diagnostic accuracy, for OSA defined by PSG AHI threshold values of 10 and 15 events/h. These results contrast with other trials which have demonstrated higher HS diagnostic accuracy (sensitivity ≥ 0.825) in populations with high probability of OSA [11]. However, our retrospective cohort was composed of patients with inconclusive HS, a higher proportion of females and for the most part mild-moderate OSA. Further larger, prospective studies will be required to determine the diagnostic utility of AnH scoring and its optimal application across the spectrum of OSA patients.

Our results demonstrate that AnH scoring improved the diagnostic accuracy of type 3 HS in patients not receiving NCM. As expected, autonomic activation was significantly blunted in patients receiving NCM, and there was no gain in diagnostic accuracy with AnH scoring in that subgroup. We did not identify any effect of comorbidities on AnH counts independent of NCM usage. However, the prevalence of comorbidities was low in this patient population targeted for HS.

This study represents to our knowledge a first evaluation of pulse oximetry-derived heart rate increases as a surrogate marker of arousal. Our results suggest that AnH scoring detected more than half of the missed hypopneas with arousal on portable monitoring in patients on NoNCM. This is based on the assumption that every AnH was linked with hypopnea with arousal events on the PSG, which due to the retrospective design, was not directly assessed in a real-time comparison. However, based on previous data, we expect that fewer than 10% of AnH events would not be associated with arousal or desaturation [29]. Nonetheless, this may have adversely affected diagnostic accuracy at the lowest AHI threshold. Given the design of this study, we were unable to assess the impact of AnH scoring on clinical decision-making by the treating physician. Prospective studies will be required to

Table 6 Diagnostic accuracy of HS vs PSG for NCM patients ($n = 21$)

PSG AHI cut-off	PSG AHI ≥ 5		PSG AHI ≥ 10		PSG AHI ≥ 15	
n	17		14		11	
HS Scoring Method	Conventional	AnH included	Conventional	AnH included	Conventional	AnH included
Sens (%)	76	76	36	36	36	36
Spe (%)	75	75	71	71	100	100
PPV (%)	93	93	71	71	100	100
NPV (%)	43	43	36	36	59	59
AUC	0.76	0.76	0.54	0.54	0.68	0.68

Sens sensitivity, *Spe* specificity, *PPV* positive predictive value, *NPV* negative predicitive value, *AUC* area under the receiver operating characteristic curve

determine whether AnH scoring can reduce the need for additional testing after HS, influence treatment initiation decisions, and ultimately improve patient clinical outcomes.

Conclusion

With the current obesity epidemic, OSA is a major public health issue. Access to sleep laboratory testing is limited and unable to meet the demand in many jurisdictions. Improved accuracy of portable testing has the potential to greatly facilitate OSA diagnosis and management. The findings of this study suggest that the use of pulse oximetry-derived heart rate increases, which can be readily scored as a surrogate marker of arousal on most available type 3 HS devices, has the potential to improve the diagnostic accuracy of these devices in patients not receiving NCM, even in mild-moderate OSA. Prospective studies are required to further evaluate the utility and clinical impact of AnH scoring on patient outcomes.

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Compliance with ethical standards

Ethical approval This study has been approved by McGill University Health Centre Research Institute Ethics Board.

Conflict of interest All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements) or non-financial interest (such as personal or professional relationships, affiliations, knowledge, or beliefs) in the subject matter or materials discussed in this manuscript.

Informed consent Informed consent was waived due to the retrospective study design.

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