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ORIGINAL RESEARCH ARTICLE

Development and validation of a novel non-contact monitor of nocturnal respiration for identifying sleep-disordered breathing in patients with heart failure

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Abstract

Aims At least 50% of patients with heart failure (HF) may have sleep-disordered breathing (SDB). Overnight in-hospital polysomnography (PSG) is considered the gold standard for diagnosis, but a lack of access to such testing contributes to under-diagnosis of SDB. Therefore, there is a need for simple and reliable validated methods to aid diagnosis in patients with HF. The aim of this study was to investigate the accuracy of a non-contact type IV screening device, SleepMinder™ (SM), compared with in-hospital PSG for detecting SDB in patients with HF.

Methods and results The study included 75 adult patients with systolic HF and suspected SDB who underwent simultaneous PSG and SM recordings. An algorithm was developed from the SM signals, using digital signal processing and pattern recognition techniques to calculate the SM apnoea-hypopnoea index (AHI). This was then compared with expert-scored PSG_{AHI}. The SM algorithm had 70% sensitivity and 89% specificity for identifying patients with clinically significant SDB (AHI $\geq$ 15/h). At this threshold, it had a positive likelihood ratio of 6.3 and a negative likelihood ratio of 0.16. The overall accuracy of the SM_{AHI} algorithm was 85.8% as shown by the area under a receiver operator characteristic curve. The mean AHI with SM was 3.8/h (95% confidence interval 0.5–7.1) lower than that with PSG.

Conclusions The accuracy of the non-contact type IV screening device SM is good for clinically significant SDB in patients with systolic HF and could be considered as a simple first step in the diagnostic pathway.

Keywords Heart failure; Sleep-disordered breathing; Apnoea-hypopnoea index; Diagnosis

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Introduction

Sleep-disordered breathing (SDB) is prevalent in patients with chronic heart failure (CHF), typically occurring in ≥50% of patients [1–4]. SDB is characterized by periods of apnoea (cessation or >90% reduction in ventilation) and hypopnoea (>30% reduction in ventilation associated with oxygen desaturation of ≥4%) [5]. Importantly, SDB is associated with

increased cardiovascular morbidity and mortality, an association that persists after adjustment for other clinical predictors of outcome [1,6–8].

There are two main types of SDB: obstructive sleep apnoea (OSA), arising from repeated partial or complete closure of the oropharyngeal airway during sleep leading to repetitive hypoxia and arousals, and central sleep apnoea (CSA), characterized by an instability in ventilatory control resulting in a temporary withdrawal of central respiratory drive leading to cessation of airflow and respiratory muscle activity. Both OSA and CSA have been documented in patients with CHF. CSA may be associated with Cheyne–Stokes respiration (CSR), which consists of crescendo–decrescendo oscillations of airflow alternating with central apnoea or hypopnoea [6].

OSA is known to be an independent risk factor for the development and progression of HF [9,10]. It may also contribute to the development of hypertension, cardiac arrhythmia, and myocardial ischaemia via a complex interplay of neurohormonal and inflammatory mechanisms [11]. CSA is generally thought to be a marker of increasing severity of CHF and may contribute to worsening symptoms of HF [12,13].

The Wisconsin Sleep cohort study estimated that 93% of women and 82% of men with symptomatic SDB were undiagnosed [14], and this proportion is likely to be similar in a HF population [15]. The current gold standard for diagnosing SDB is attended, overnight in-hospital polysomnography (PSG). This test is not always readily available and is expensive because of the need for well-equipped sleep laboratories and trained personnel who can interpret the results.

To reduce costs and provide timely diagnosis of SDB, a number of portable home monitors (PMs) have been developed but they only record a limited number of signals. PMs are usually used as an initial diagnostic or screening tool and in selected patients, if the test results are positive, can be used to recommend treatment, while ruling out additional testing if results were negative [16]. PMs can be used on an unattended basis, and several studies have reported a high diagnostic accuracy of up to 90% in detecting SDB [17–21]. High study failure rates because of their complex unattended design can, however, result in the need for repeated studies [16]. Overnight pulse oximetry and heart rate variability are some of the methods employed to screen for SDB with varying degrees of accuracy and usefulness reported [20,21].

The SleepMinder™ (SM) device is a novel non-contact type IV screening device [22] containing a biomotion sensor transceiver that uses ultra-low power radiofrequency signals to detect movement and breathing in a patient. It can be used in the patient's home, is easy to handle, and requires minimal engagement from the patient. It is also expected to have a lower failure rate because of its non-contact design. The SM has been validated in a number of studies for measuring apnoea-hypopnoea index (AHI) in patients with suspected OSA [23,24], for scoring sleep/wake patterns [25] and CSR patterns in patients with HF [26]. However, it has not been validated for screening for SDB in patients with HF.

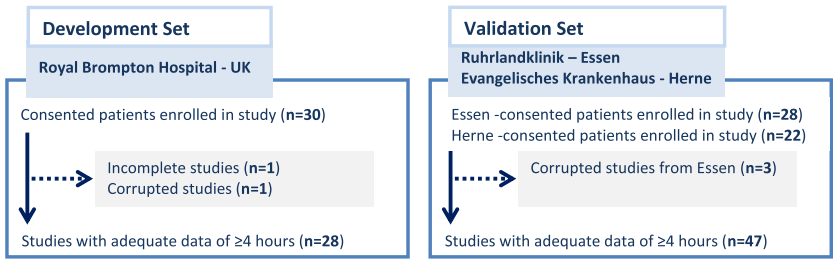
The aim of this study was to develop and evaluate an AHI algorithm for the SM device for screening patients with HF with suspected SDB.

Methods

Study population

Eighty adult patients underwent simultaneous SM and PSG studies as they were enrolled at three HF centres (30 patients from south-west London, UK; 28 patients from Essen and 22 patients from Herne, Germany). Five patients were excluded because of incomplete or corrupted data (one incomplete and one corrupted studies from London and three corrupted studies from Essen), leaving 75 patients with adequate paired data (>4 h) for analysis (*Figure 1*). No patient had undergone a diagnostic procedure for SDB before entering the study. All patients were referred to the study based on clinical judgement alone. UK volunteers were being followed up in a HF clinic and subsequently screened for SDB at the clinic. The German participants had been referred to a sleep laboratory for SDB screening. This is in accordance to the 2007 Clinical Guidelines of the portable monitoring task force of the American Academy of Sleep Medicine (AASM) [27] that

Figure 1 Patients flow through the study.



recommend that portable monitors should be used on patients with a high pre-test probability for OSA.

All patients had stable chronic systolic HF with a left ventricular ejection fraction (LVEF) of ≤45% based on transthoracic echocardiography. Patients with significant obstructive lung disease (forced expiratory volume in 1 s <50% of predicted) or those being treated with any form of positive airway pressure were excluded.

Patients gave written informed consent to participate in this study, and ethical approval was obtained from the relevant national and local committees. The study was conducted in accordance with the principles of good clinical practice and the Declaration of Helsinki.

Overnight polysomnography

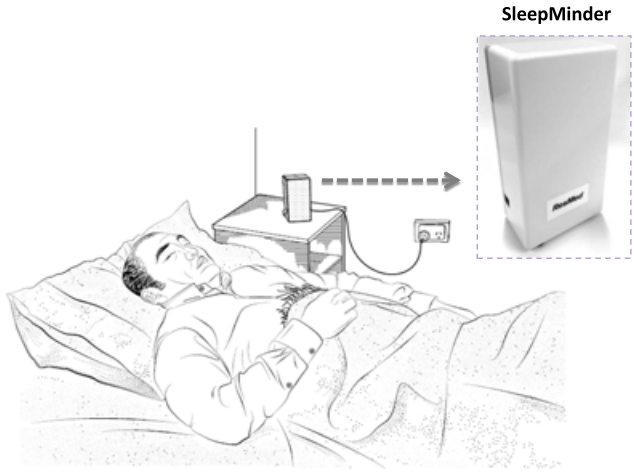
Attended in-hospital overnight PSG was performed in sleep laboratories using standard techniques. The PSG systems used were Somnoscreen PSG Tele (Somnomedics GmbH, Germany) in the UK, Rembrandt PSG (Embla Sys Inc., USA) in Essen, and Embla S7000 PSG (Embla Sys Inc., USA) in Herne. Outputs from these PSG recording systems were similar. Thoraco-abdominal motion was measured by respiratory inductance plethysmography, and nasal airflow was monitored by nasal pressure cannula and an oral flow thermistor. Arterial oxyhaemoglobin saturation (SaO_2) was monitored by pulse oximetry. Sleep was monitored using a standard neuro electrode placement system to include electroencephalogram references (C4/A1), (C3/A2), and (O1/A2). Submental and anterior tibialis electromyograms were also recorded. Electrooculogram electrodes placed on both lateral canthi measured rapid eye movements. The transducers and lead wires permitted normal positional changes during sleep as well as movement out of bed. Bedtime was at each subject's discretion, and PSG was terminated after final wakening.

Polysomnography scoring

Polysomnography studies were scored to determine PSG_{AHI} by a polysomnographic technologist using the AASM 2007 rules for respiratory events. The expert had no knowledge of the SM recordings as these were kept separate from the PSG studies. Apnoeas were defined as cessation of airflow or >90% reduction in airflow lasting ≥10 s. Hypopnoeas were defined as ≥30% reduction in airflow amplitude lasting ≥10 s with a >4% oxygen desaturation measured on pulse oximetry.

The classification of patients into CSA or OSA was based on criteria that were used in the SERVE-HF study [28,29]. Patients with clinically significant SDB (AHI ≥ 15/h) in whom greater than 50% of events were central, and central AHI ≥ 10/h were denoted as CSA patients; whereas if greater than 50% of events were obstructive, they were denoted as patients with OSA [28,29].

Figure 2 Patient with SleepMinder on bedside.



SleepMinder™ recording

The SM device (ResMed Sensor Technologies, Dublin, Ireland) is a non-contact bio-motion recording system interpreting body movement and breathing via two sinusoidal electromagnetic wave signals. The sensor operates in a licence-free band at 5.8 GHz and emits an average power of <10 mW. The signal is range gated to restrict the sensing range to a distance of 0.3 to 1.5 m.

On the night of the PSG study, the SM was positioned by the patient's bedside at a height of 10 and 100 cm from their chest (*Figure 2*). It was turned on at the start of the PSG recording and then turned off at final patient wakening. SM recordings were logged onto a Secure Digital card, which was downloaded at the end of the study. The SM_{AHI} was determined from algorithms developed from the SM signals.

Development of the SleepMinder™ AHI algorithm

The first step was to synchronize the SM and PSG signals as accurately as possible and to create a common timestamp as the signals were recorded separately and at different sampling frequencies (16 Hz for SM and 20 Hz/32 Hz for PSG). To achieve this, a cross correlation and sliding window method was used to align feature vector transformations of the signals [30].

The SM signal was pre-processed to filter noise and remove the baseline wander (also known as de-trending). Using pattern recognition and cycle interval techniques the respiratory signal was isolated. The sleep respiratory signal was then divided into segments for AHI algorithm development and analysis. These segments, also known as epochs, are the

time-defined segments upon which PSG is routinely scored and are typically 30 s.

An event-based algorithm was developed for the detection of SDB events (apnoeas and hypopnoeas) in the SM signals with the fundamental criteria being a ≥50% reduction in the amplitude of the non-contact measure of overall body breathing effort, lasting for ≥10 s. Severity of SDB was defined as mild (5/h ≤ AHI < 15/h), moderate (15/h ≤ AHI < 30/h), or severe (AHI ≥ 30/h).

Statistical analysis

The overnight study data were divided into two datasets. The data from the UK ($n = 28$) were used for SM AHI algorithm development. Results were then validated on patients from Essen and Herne ($n = 47$). The allocation of patients into development and validation sets based on geography potentially reduces the positive effect of any bias to site specific artefacts in the data and improves the likelihood of the test result being representative of true performance.

Normally distributed and continuous data are presented as means ± standard deviation (SD). The one-sample *t*-test was utilized to confirm the normal distribution of the data. The two-sample *t*-test was used to compare distributions of the non-paired continuous variables. For categorical variables, the chi-squared distribution was used. Algorithm development and signal analyses were performed using Matlab from Mathworks version 2011b. Statistical significance was defined as $p < 0.05$.

Correlation between the two AHI diagnostic tools was tested using Pearson's correlation coefficient, and the degree of agreement was tested using a Bland and Altman analysis examining for any systematic bias in scoring by either tool. Receiver operator characteristic curves were constructed to inspect the performance of the algorithm for a selection of SDB severities.

Results

Baseline characteristics

The demographic and clinical characteristics of the recruited patients are shown in *Table 1*. There was some variation in the mean LVEF across the recruiting centres, but all patients had an LVEF ≤ 45% and 59% of the patients had an LVEF ≤ 35%. Patients recruited in London had milder symptoms than those recruited in Germany. LVEF was almost equivalent in the validation dataset and the development dataset, with somewhat higher use of ACE inhibitors and ß-blockers, and lower use of mineralocorticoid receptor antagonists, in the validation dataset (*Table 2*). In the development set 36% (10 of 28) of the patients had SDB (25% with OSA and 11% with CSA), while in the validation set 43% (20 of 47) had SDB (34% with OSA and 9% with CSA). CSR patterns were noted in PSGs from 29% (8 of 28) of the patients in the development set, with 18% having AHI <15/h and 11% having AHI ≥15/h. In the validation set, 40% (19 of 47) had CSR with 17% having AHI <15/h and 23% having AHI ≥15/h.

Prevalence of sleep-disordered breathing

Based on PSG findings, the prevalence of clinically-significant SDB (AHI ≥ 15/h) was 44%, with a mean AHI of 20.4/h [95% confidence interval (CI) 15.9–24.9]; 27% of patients had no SDB (AHI ≤ 5/h).

AHI algorithm performance

Correlation

There was good correlation between SM_{AHI} and PSG_{AHI}, with correlation co-efficient values of 0.93 and 0.83 ($p < 0.001$) in the development and validation set of patients, respectively (*Figure 3*).

Table 1. Demographic characteristics and clinical features of overall patients and by study centre

	Overall ($n = 75$)	London ($n = 28$)	Essen ($n = 25$)	Herne ($n = 22$)
Age, years (range)	68 (34–90)	69 (34–90)	66 (41–84)	70 (52–81)
Male, *n* (%)	65 (87)	26 (93)	21 (84)	18 (82)
BMI, kg/m^2	29 ± 4.9	29 ± 5.5	29 ± 5.2	29 ± 3.8
LVEF, %	34 ± 8	34 ± 6.2	29.7 ± 11.1	38.0 ± 6.8
NYHA class ≥ III, *n* (%)	49 (65)	8 (29)	20 (80)	21 (95)
Ischaemic aetiology, *n* (%)	51 (68)	25 (89)	12 (48)	14 (64)
Diabetes mellitus, *n* (%)	17 (23)	6 (21)	7 (28)	4 (18)
COPD, *n* (%)	10 (13)	4 (14)	6 (24)	NA
Hypertension, *n* (%)	36 (48)	15 (54)	21 (84)	NA
ß-blockers, *n* (%)	60 (80)	21 (75)	21 (84)	18 (82)
ACEi, *n* (%)	56 (75)	15 (54)	23 (92)	18 (82)
MRA, *n* (%)	44 (59)	20 (71)	17 (68)	7 (32)

Values are mean ± standard deviation or (range), or number of patients (%).
ACEi; angiotensin converter enzyme inhibitor; BMI, body mass index; COPD, chronic obstructive pulmonary disease; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; NA, not available; NYHA, New York Heart Association.

Table 2. Demographic characteristics and clinical features of development and validation group of patients

	Development (n = 28)	Validation (n = 47)	P-value
Age, years (range)	69 (34–90)	68 (41–84)	0.931
Male, *n* (%)	26 (93)	39 (83)	0.223
BMI, kg/m^2	29 ± 5.5	29 ± 4.7	0.859
LVEF, %	34 ± 6.2	33.6 ± 10.1	0.951
NYHA class ≥ III, *n* (%)	8 (29)	41 (87)	<0.001
Ischaemic aetiology, *n* (%)	25 (89)	26 (55)	0.002
Diabetes mellitus, *n* (%)	6 (21)	11 (23)	0.843
ß-blockers, *n* (%)	21 (75)	39 (83)	0.403
ACEi, *n* (%)	15 (54)	41 (87)	0.001
MRA, *n* (%)	20 (71)	24 (51)	0.081
OSA, *n* (%)	7 (25)	16 (34)	0.411
CSA, *n* (%)	3 (11)	4 (9)	0.751
CSR, *n* (%)	8 (29)	19 (40)	0.300

Values are mean ± standard deviation or (range), or number of patients (%).
ACEi; angiotensin converter enzyme inhibitor; BMI, body mass index; CSA, central sleep apnoea; CSR, Cheyne-Stokes Respiration; LVEF, left ventricular ejection fraction; MRA, mineralocorticoid receptor antagonist; NYHA, New York Heart Association; OSA, obstructive sleep apnoea.

Figure 3 Pearson's correlation co-efficient plot for the validation set of patients (n = 47).

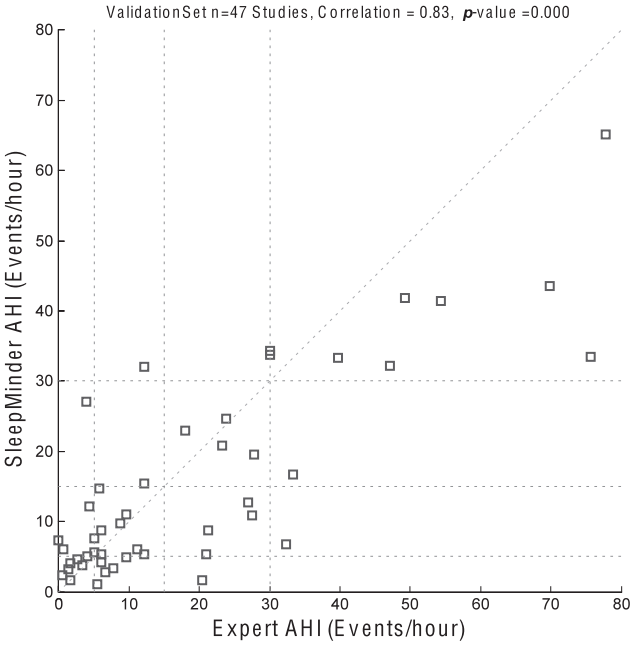
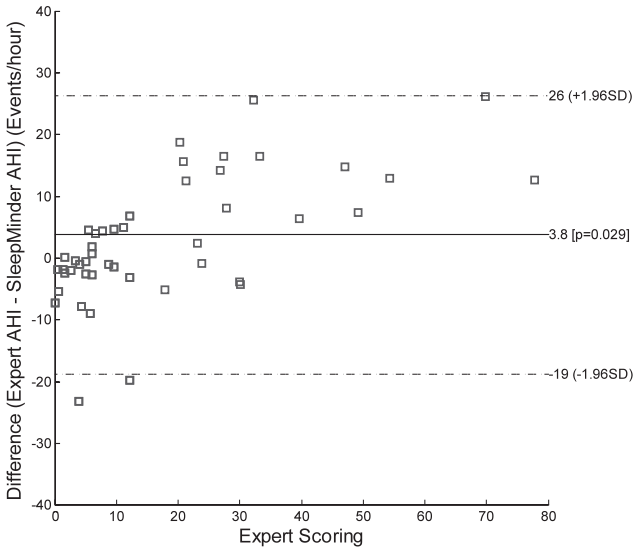


Figure 4 Bland-Altman Plot for validation set of patients (n = 47). (Shaded area represents agreement of both methods within 10 events per hour; lines represent mean difference and 95% upper and lower confidence intervals for the difference).



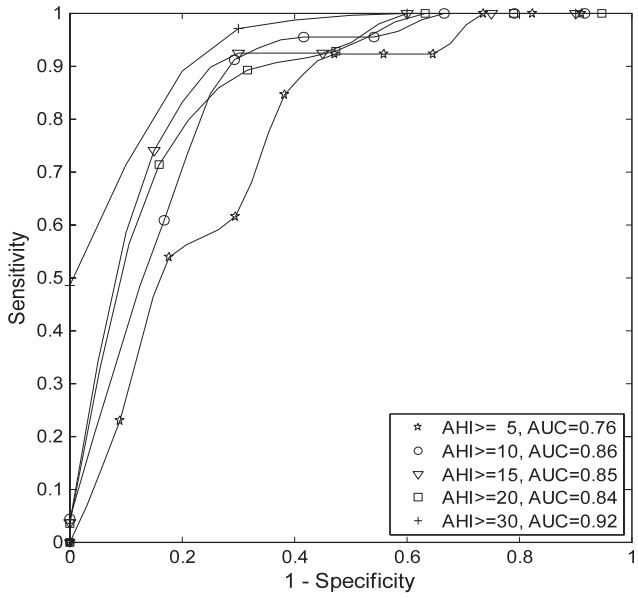
Test of agreement

A Bland–Altman plot was constructed to examine the agreement and systematic bias between the two AHI scoring techniques on the validation set (*Figure 4*). This plot demonstrated that the overall agreement between SM_{AHI} and PSG_{AHI} was fairly good for AHI values of <15/h. Conversely, there was consistent underscoring with SM for AHI values ≥15/h. The mean difference between the two scoring techniques was 3.8/h (95% CI 0.5–7.1, $p = 0.029$).

Screening accuracy

In terms of screening accuracy, the SM was 70% sensitive and 89% specific for identifying patients with clinically-significant SDB (AHI ≥ 15/h). It had a positive likelihood ratio (LR+) of 6.3 (positive predictive value 82.4%) and a negative likelihood ratio (LR−) of 0.16 (negative predictive value 80%). The overall accuracy of the SM AHI algorithm was 85.8% as represented by the area under a receiver operator characteristic curve constructed for this diagnostic threshold (*Figure 5*). The

Figure 5 Receiver operator characteristic curves for various thresholds of sleep-disordered breathing. AUC, area under curve; AHI, Apnoea-hypopnoea index.



performance of the algorithm was improved for the most severe disease threshold of AHI ≥ 30/h (sensitivity 82%, specificity 97%, LR+: 29, LR−: 0.03, AUC 92%).

Misclassification rate according to treatment

The misclassification rate (MRrx) of the SM device was calculated according to the commonly used treatment threshold of AHI ≥ 15/h. Compared with expertly scored PSG, SM misclassified 9/47 (19%) patients in the validation set [AHI < 15/h ($n = 6$) and AHI ≥ 15/h ($n = 3$)].

Discussion

This study describes the development and validation of an algorithm using signals obtained from the SM type IV screening device and showed that it is capable of identifying SDB (based on the AHI) with a good overall diagnostic accuracy in patients with chronic systolic HF and a prior suspicion of SDB. The prevalence of SDB (AHI ≥ 15/h) was 44%, which is comparable to that reported in previous studies [1–4]. SM/PSG signals from 80 patients were successfully collected. Exclusion of data from five patients was because of poor signal quality. The failure rate for SM was therefore 6.25%, which is comparable to contact devices used either at home, or in-hospital [16]. Refinement of the hardware and algorithm could lower the failure rate.

A positive SM result was associated with a high likelihood of clinically relevant SDB (LR+ 6.3). With the false positive rate of 6%, three patients in this cohort would have been incorrectly identified as having SDB. Conversely, only 16% of patients with a negative SM test actually had SDB (LR− 0.16). In general, portable monitors should be used to increase the pre-test probability to a sufficiently high post-test probability that one is very confident that the patient has OSA. As the relationship between the pre-test and post-test probability can be described by the LRs, it can be claimed that the SM device is capable of detecting SDB in patients with HF with a good accuracy at a threshold of AHI ≥ 15/h. The SM device may therefore have the greatest utility as a first step in identifying patients with a high probability of SDB and appropriately prioritize them for a formal PSG study.

In general, at an AHI threshold of <15/h, the SM performed well, although there was some underestimation (16%) at thresholds of ≥15/h. A potential explanation for this underestimation is the coincidence of body movements and respiratory events at higher AHI levels. The SM algorithm focuses on separating respiratory-related movement patterns from other movement-related patterns, e.g. limb movement (LM). Therefore, at higher apnoea/hypopnoea event rates, the SM algorithm may flag a number of events as LM rather than SDB events resulting in lower AHI values. However, the high sensitivity and specificity of the SM device at AHI values of ≥30/h indicate that even if AHI is underscored in some patients, the majority were still assigned to the appropriate SDB category. As an example, of the 47 studies in the validation set, 11 had an AHI of ≥30/h based on expert scoring of PSG data. Of these 11 studies, only two were misclassified by the SM algorithm (one as mild and one as moderate). The mild classification recording was entirely dominated by LMs of both legs, while the moderate classification recording was also affected by LMs, but to a lesser extent. As both of these studies had a high AHI and a large number of LMs, there were numerous instances where LMs coincided with breathing events. In disregarding LMs, the SM algorithm also therefore missed the overlapping breathing event.

The performance of SM in detecting and screening SDB in this HF cohort was comparable to other forms of portable/home contact screening devices [14–19]. However, most of these studies only included patients without HF. Two studies were performed on patients with a high clinical suspicion of OSA using ApneaLink™ as an SDB screening tool [31,32]. In both studies the accuracy of the automatic scoring of the contact-based type IV ApneaLink™ on patients without HF was comparable to that of the non-contact type IV SM performance reported in this paper (sensitivity-specificity of 64–94% and 89–60% for [31] and [32], respectively). An alternative study on patients with HF using oxygen saturations to determine the presence of SDB reported slightly better performance (sensitivity–specificity of 93–73%) [19].

A SM study is likely to be less costly and easier to perform than overnight in-hospital PSG and is more convenient for patients. Given that SM is non-contact, it is unlikely to influence

sleeping and is likely to be acceptable to patients. As a result, use of the SM for multiple nights could be possible as part of the diagnostic process for clinically important SDB. This means that the SM offers the potential to document variations in SDB metrics over much longer monitoring periods and improve understanding of how these physiological parameters influence or are influenced by the course of HF over time.

A future enhancement could be the ability to discriminate between obstructive and central events, which is important in the selection of an appropriate treatment. Treatment of predominant CSA in patients with HF has been shown to increase mortality [28]. At the current stage of development, limitations exist in using the SM technology for this application because the sensor is a movement detector and it can be difficult to separate respiratory movement from other sources of movement.

This study was a pilot study, and further studies are required to confirm the result in the wider HF population. This study included only patients with reduced LVEF, and so the results may not be generalizable to those who have HF with preserved ejection fraction, who also have a high incidence of SDB [33]. Future studies need to include a higher proportion of female samples given the predominantly male sample included in this trial.

In conclusion, in addition to being able to detect SDB in patients without HF, the results of this study show that the non-contact type IV screening device SM can be used to identify SDB in patients with systolic HF with good accuracy. By identifying patients with HF most likely to have SDB as suitable for referral for further diagnostic study (e.g. PSG), SM could reduce pressure on limited sleep service resources. This is particularly relevant if demand for these services increases based on the results of multinational randomized control trials investigating the morbidity and mortality benefits of treating SDB in patients with HF [29].

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Conflict of interest

H.S.'s salary was funded for research by ResMed through a grant to Imperial College. M.R.C. received research funding and consultancy fees from ResMed. H.T. receives a study grant, travelling expenses, and honorarium for invited lectures from ResMed. H.G. received financial support from ResMed only for medical talks. R.N.K., A.Z., M.C., C.H., S.F. and K.S. are all employed by ResMed. G.W., A.K.S. and M.D. have no conflicts of interest to declare.

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