




## Sleep Medicine

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# Obstructive sleep apnea diagnosis and beyond using portable monitors

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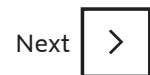
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## Highlights

- Several factors impact portable monitor performance in OSA diagnosis.
- Individual physiological parameters & their ability to detect OSA are assessed.
- Recent innovative parameters for portable OSA quantification are highlighted.
- Portable monitors can be used in OSA endotyping and assessing health impacts.
- Portable OSA endotyping can transform OSA diagnosis and treatment approaches.

## Abstract

Obstructive sleep apnea (OSA) is a chronic sleep and breathing disorder with significant health complications, including cardiovascular disease and neurocognitive impairments. To ensure timely treatment, there is a need for a portable, accurate and rapid method of diagnosing OSA. This review examines the use of various physiological signals used in the detection of respiratory events and evaluates their effectiveness in portable monitors (PM) relative to gold standard polysomnography. The primary objective is to explore the relationship between these physiological parameters and OSA, their application in calculating the apnea hypopnea index (AHI), the standard metric for OSA diagnosis, and the derivation of non-AHI metrics that offer additional diagnostic value. It is found that increasing the number of parameters in PMs does not necessarily improve OSA detection. Several factors can cause performance variations among different PMs, even if they extract similar signals. The review also highlights the potential of PMs to be used beyond OSA diagnosis. These devices possess parameters that can be utilized to obtain endotypic and other non-AHI metrics, enabling improved characterization of the disorder and personalized treatment strategies. Advancements in PM technology, coupled with thorough evaluation and validation of these devices, have the potential to revolutionize OSA diagnosis, personalized treatment, and ultimately improve health outcomes for patients with OSA. By identifying the key factors influencing performance and exploring the application of PMs beyond OSA diagnosis, this review aims to contribute to the ongoing development and utilization of portable, efficient, and effective diagnostic tools for OSA.



## Keywords

OSA; Polysomnography (PSG); Sleep study; At-home sleep test; Apnea-hypopnea index (AHI); Non-AHI metrics; Endotypes; Phenotypes

## 1. Introduction

Obstructive sleep apnea (OSA) is a sleep and respiratory disorder characterized by repetitive partial or complete collapse of the upper airway during sleep [1]. This narrowing or collapse of the upper airway is primarily attributed to factors such as poor upper airway anatomy, reduced responsiveness of dilator muscles, and hypertrophy of airway soft tissues, resulting in decreased airflow, oxygen desaturations and increased effort to breathe during sleep [[2], [3], [4]]. OSA is associated with a range of negative health outcomes, including daytime sleepiness, neurocognitive impairment, cardiovascular diseases, and increased mortality [[5], [6], [7], [8]]. It affects a substantial portion of the general population, with prevalence rates ranging from 9 to 38% [9].

Given the detrimental effects associated with OSA, there is a critical need for early and accurate diagnosis. Polysomnography (PSG) is currently considered the gold standard for OSA diagnosis,

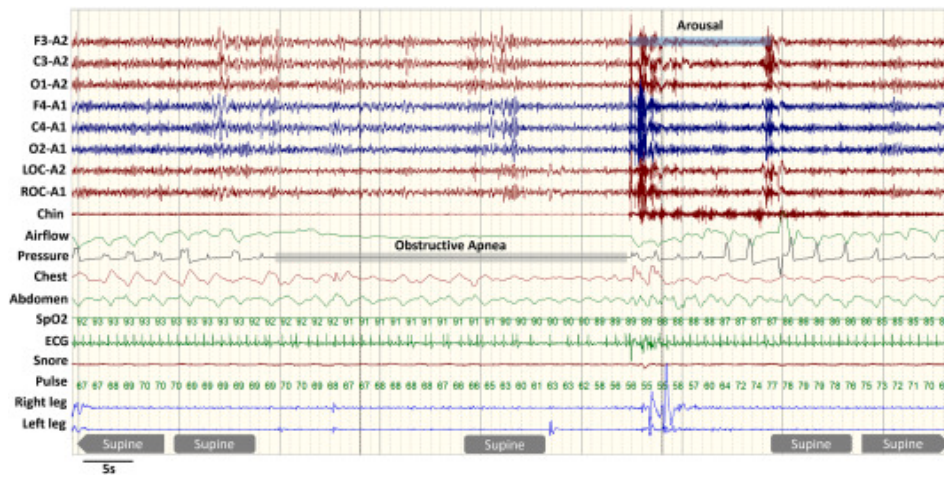
where the apnea-hypopnea index (AHI) is determined. The AHI serves as the standard diagnostic metric for OSA, quantifying the frequency of airflow reduction or cessation associated with the upper airway during sleep [1]. PSG involves a comprehensive sleep laboratory assessment that measures various physiological parameters, including respiration, snoring, cardiovascular measures, and brain activity [10]. To capture these measurements, PSG employs a multitude of sensors, such as pulse oximeter, respiratory inductance plethysmography (RIP) belts, nasal pressure transducer, oronasal thermistor, electroencephalogram (EEG), electrooculogram (EOG), chin and limb electromyogram (EMG), video and microphone [11]. Trained technicians attach these sensors to the patient's body within a controlled clinical setting.

However, the requirement for a clinical setting, technical expertise, and the attachment of multiple sensors to the body limit the practicality and accessibility of PSG. In response to these limitations, portable monitors (PMs) have been developed for the detection of OSA, particularly in symptomatic patients having no underlying cardiovascular diseases [12]. PMs utilize one or more of the physiological signals typically acquired during PSG but are applied outside of the clinic [12,13].

This review aims to discuss the various physiological signals used in PSG and PMs, their relevance to OSA, and their acquisition and utilization in calculating the AHI. Furthermore, it examines the performance of PMs in detecting OSA compared to PSG, considering sensitivity, specificity, and accuracy, while also exploring factors that may influence their performance. The review also delves into the concept of endotypic metrics, including upper airway collapsibility, muscle responsiveness, loop gain, and arousal threshold, which are derived from PSG and/or non-PSG signals and used to stratify OSA patients into treatment subgroups. Additionally, it describes other non-AHI metrics, such as hypoxic burden index and obstruction severity, and highlights their potential use for predicting specific effects of OSA. Finally, the review discusses the applicability of non-AHI metrics in PMs and the role of modified or advanced PMs in OSA endotyping.

## 2. Apnea hypopnea index (AHI) and polysomnography (PSG)

PSG involves monitoring various physiological signals during sleep, including EEG, EOG, limb and chin EMG, oxygen saturation, respiratory movement of the chest and abdomen and body position (Fig. 1). EEG with or without EOG and chin EMG are used to detect REM and NREM sleep stages and determine total sleep time [[14], [15], [16], [17], [18]]. Limb EMG is used to monitor periodic limb movement, which can interfere with the overall accuracy of OSA diagnosis [19]. Oxygen saturation levels throughout sleep are monitored by a pulse oximeter, often attached to the finger [17]. Sleep position, such as lateral or supine, is tracked by an accelerometer and video to distinguish between position-dependent and position-independent OSA [17,20,21].



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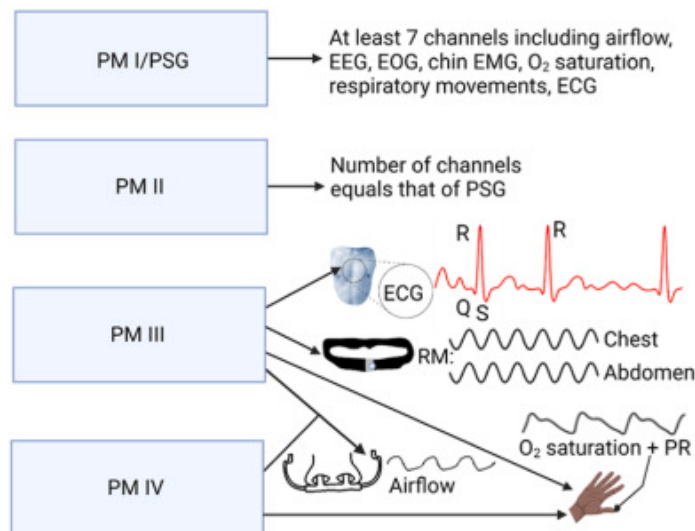
Fig. 1. A sample of the common physiological parameters acquired during polysomnography. Obstructive apnea and hypopnea events are defined by cessation or reduction, respectively, in airflow/pressure signals despite persistent movement of the chest/abdomen and are accompanied by decrease in oxygen saturation (SpO<sub>2</sub>). Arousal may occur at the end of the obstructive event or following airflow restoration. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

The PSG data are analyzed alongside nasal airflow to detect and score airflow reduction (hypopnea) and airflow cessation (apnea). Apnea is scored as a  $\geq 90\%$  decrease in airflow from pre-event baseline, and hypopnea as a  $\geq 30\%$  decrease in airflow from pre-event baseline with  $\geq 3\%$  oxygen desaturation or arousal lasting for  $\geq 10$ s [22]. Arousal is indicated by sudden change in the frequency of the EEG waveform (Fig. 1) [18]. The AHI is then computed as the sum of apneas and hypopneas per hour of sleep time for different sleep stages and sleep positions [11]. To use AHI in the diagnosis of “obstructive” sleep apnea, respiratory effort measured by RIP-monitored chest and abdomen movements must persist during the OSA events (Fig. 1) [23]. The presence of effort discriminates OSA from central apnea, in which there is no effort to breathe and airflow ceases [24]. AHI is required to assess the severity of OSA, where  $5 \leq \text{AHI} \leq 15$  events/hr is considered mild,  $15 \leq \text{AHI} \leq 30$  events/hr is moderate and  $\text{AHI} > 30$  events/hr is severe [1].

### 3. Portable monitors

PMs have emerged as a simplified alternative to PSG for certain cases, addressing the complex and technical challenges associated with PSG, which is typically limited to sleep clinics and specialized centers [11]. PMs are essentially OSA testing devices that can be used outside of the sleep clinic environment [17]. Depending on their type, PMs employ simplified methods and connections to the body to acquire the necessary physiological parameters for detecting respiratory events, often requiring minimal or no assistance from a trained technician [25,26]. PMs are generally classified

into three main types: PM II, III and IV [13], based on the number of channels used to capture the relevant physiological parameters (Fig. 2). It is worth noting that a fourth monitor type, referred to as PM I, is recognized as PSG itself [12].

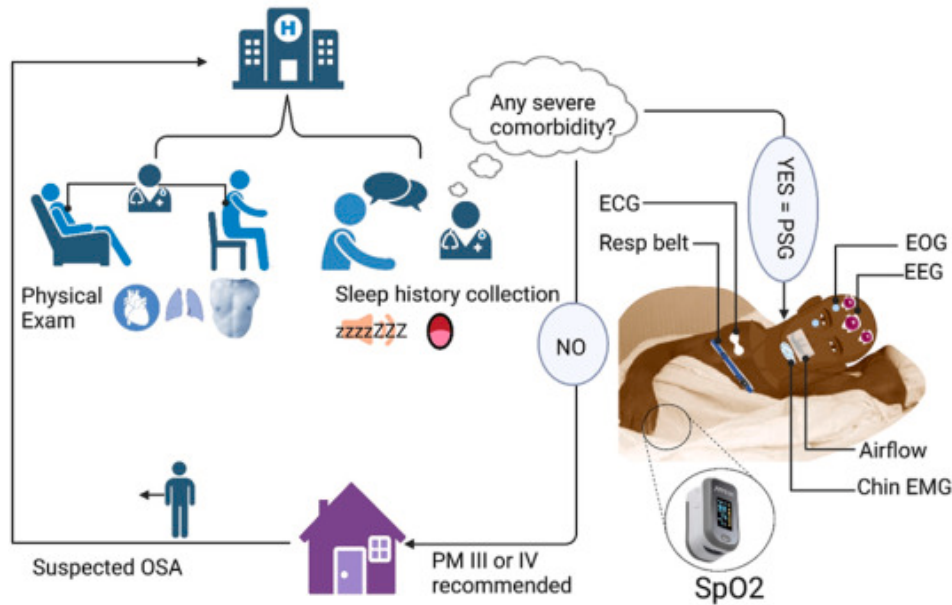


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Fig. 2. A summary of the number and type of signals used in portable monitors (PMs) I-IV. PM II, known as unattended PSG, uses the same number of channels (at least 7) as standard PSG (PM I). PM III requires at least 4 physiological signals, while PM IV only needs one or two signals. RM, respiratory movement. PR, pulse rate.

Prior to considering the use of PMs for OSA diagnosis, a pre-diagnostic clinical examination is typically conducted [27]. During this examination, the clinician evaluates the patient's sleep history and assesses their respiratory and cardiovascular health. Symptoms such as regular loud snoring, nocturnal gasping or choking may indicate increased risk of OSA. PMs II-IV are recommended for home sleep apnea studies in symptomatic patients who do not suffer from potential respiratory muscle weakness, severe cardiopulmonary disease, or critical illness or immobility [12,27]. Otherwise, clinical PSG is the preferred diagnostic approach. Fig. 3 provides an overview of the decision-making process for selecting the appropriate PM [27].



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Fig. 3. Procedure for portable monitor prescription. Upon suspicion of OSA, the clinician physically examines the patient, and enquires about the sleep history. In the absence of severe comorbidities, portable monitors III and IV are recommended. Otherwise, PSG (i.e., PM I) is required.

PM I, also known as standard PSG, employs a minimum of 7 channels, including EEG, chin EMG, EOG, ECG, airflow, effort, and oxygen saturation [12,28]. Although rarely prescribed [12,29], PM II utilizes the same number of channels as PM I, but is intended for unattended use outside of the laboratory setting [27]. Patients are initially set up with a PM II device in the clinic and then sent home, where they initiate data acquisition on the device upon going to bed. Alternatively, the setup can be conducted directly at the patient's home [29]. PM III utilizes at least 4 channels, incorporating two respiratory parameters such as airflow and respiratory movements, a cardiac parameter such as ECG or heart rate, and oxygen saturation [13,27]. PM IV, on the other hand, utilizes one or two channels, typically focusing on heart rate and oxygen saturation or solely airflow [27].

For a potential OSA patient, a standard ambulatory diagnostic device, either PM III or IV, is typically prescribed [30,31]. At the clinic, the patient receives education on the proper use and setup of the PM before taking it home. The patient then wears the PM to bed at home, collecting the necessary sleep data for the instructed number of nights. Subsequently, the patient returns the PM to the clinic, where the acquired sleep data is processed for diagnosis. These clinically oriented PMs require prescriptions from a sleep practitioner. In contrast, consumer-focused PMs can be directly obtained from the manufacturer. These PMs utilize cloud algorithms to process a user's sleep data and present OSA-related information on a user-friendly graphical user interface (GUI)-enabled

device upon request [32,33]. Table 1 provides a select list of both clinically oriented and consumer-focused PMs that are currently available.

Table 1. Types of portable monitors (PMs).

Clinical PMs	Type	Description
Alice 5 Respiroics(Murrysville PA, USA) [34,35]	I	The physiological signals acquired by different sensors are received by a computer, which is connected to the Alice base station set up at the clinic through wires or wirelessly, or a network switch. The computer displays these signals for use by the clinician. The base station stores and digitizes these signals received from the sensors connected to a patient.
SOMNO sleep lab PSG (SOMNOmedicsGmbH, Germany) [36,37]	I	SOMNO HD eco allows transfer of acquired physiological data to the clinician or physician's tablet and smart phone, in addition to display on the computer, thus enhancing monitoring in the sleep lab.
SleepStudy PSG (Medeia, Santa Barbara, CA93101, US) [38]	II	SleepStudy home PSG acquires 8 channels, while Embletta MPR PG offers 12–28 channels for data acquisition.
Embletta MPR PG (Natus Medical Inc., Middleton, WI 53562 USA) [39]	II	
Nox-T3 (Nox Medical) [40]	III	NOX-T3 has bipolar channels for ECG, EEG, EMG and EOG as in PSG, however these select channels are not commonly used [40]. Hence, it is here categorized type III.
BREAS SC20(Breas Medical AB, Sweden) [41]	III	Both BREEAS SC 20 and ApneaLink Air are entirely cardiorespiratory devices recording at least airflow, respiratory movements, oxygen saturation, pulse rate. Snoring is additionally recorded by ApneaLink Air, while ambient light intensity and limb movements are also monitored by
ApneaLink Air(ResMed) [42]	III	
SleepView monitor (CleveMed) [43]	III	BREAS SC20. SleepView monitor basically records respiratory movement, nasal airflow, pulse rate and oxygen saturation.
Apnomonitor (Chest M.I. Inc, Tokyo) [44]	IV	Apnomonitor records airflow, pulse rate, oxygen saturation and body position. It is classified as a type IV device as it does not measure respiratory movement [44].
Belun sleep platform (ring) [45,46]	IV	An FDA-approved ring, it provides physicians with a patient's AHI as well as vitals like SpO <sub>2</sub> , heart rate and response of autonomic nervous system to heart rate variability.
WatchPAT [47,48]	–	WatchPAT records 7 physiological signals, including the propriety peripheral arterial tonometry (PAT), SpO <sub>2</sub> , heart rate, snoring, body

Clinical PMs	Type	Description
		movement and position, and chest motion, but it does not belong to any class of PMs due to its use of PAT [27]
AccuPebble SA100 [49], [50]	–	AccuPebble is a small pebble-shaped device attached to the neck. It is only commercially available to the clinician and can provide full remote diagnostics reports to the clinician.
Consumer-focused PMs	Type	Description
Firefly App (ResMed) [51]	–	This proprietary application runs on mobile phone and eliminates the need for buying extra sensors [51].
Apnea App [52]	–	A proprietary application, Apnea App runs on a mobile phone, eliminating the need for additional sensors [52].
Withins Sleep Analyzer [32,53]	–	CE-marked for consumer use, contactless, and displays OSA information using a custom operating system or Android App on a mobile phone.
GT 2 Watch, Huawei [33,54]	IV	The physiologic data measured by GT2 smartwatch are not for medical use, as indicated by the manufacturer. Nonetheless, a recent clinical trial has shown the watch's potential for AHI estimation [33].

Data acquired by clinical PMs are processed only by clinicians, whereas the OSA information can be provided to the user of a consumer-focused PM upon a request to the manufacturer's cloud system. Apnea App and Firefly App are potential consumer focused PMs requiring only users' mobile phone. “-” is used where PM does not fit into the types I-IV, e.g., Watch PAT uses peripheral arterial tonometry (PAT) in addition to pulse oximetry [47] and does not fit into any of PMs I-IV [27].

#### 4. Physiologic parameters used in OSA detection and portable monitors

Certain physiological parameters, such as SpO<sub>2</sub>, heart rate and respiratory rate, can be derived from one physiological signal measured from a single channel of pulse oximetry [33]. Accordingly, PMs are grouped into different categories based on the number of common parameters measured or derived, rather than the number of channels. Table 2 provides a summary of various PM devices along with their physiological parameters and performance characteristics in terms of AHI detection.

Table 2. Different combinations of physiological parameters in portable OSA detection based on PSG-AHI cutoffs of 5, 15 and 30.

Categories	Device name or authors	Sensitivity (%)			Specificity (%)			Accuracy (%)			Number of patients
		AHI ≥5	AHI ≥15	AHI ≥30	AHI ≥5	AHI ≥15	AHI ≥30	AHI ≥5	AHI ≥15	AHI ≥30	
		Respiration	ResMed ApneaLink [55]	90	79	66.7	76.9	88.2	95.5	-	
	Nasal flow wizard [34]	85	-	80	92	-	89	-	-	-	193
	APScan CIED [56]	-	80	83.3	-	60	60	-	-	-	21
SpO2	WISM, CloudCare Healthcare [57]	98	92	-	100	100	-	98	93	-	196
	Gutiérrez-Tobal et al. [58]	96.6	92.5	88.5	50	73.5	65.5	92.9	87.4	78.7	320
SpO2+heart rate	Belun Sleep Platform Ring [59]	55.8	62.1	50	88.6	79.6	67.2	70.5	73.1	64.1	78
Airflow+SpO2+heart rate	SleepView [60]	80.3	87.0	94.9	95.45	84.62	92.59	-	-	-	93
Airflow+SpO2+ heart rate+PPG amplitude change	Sommermeier et al. [31]	100	90	87	60	86	96	-	-	-	66
Airflow+SpO2+heart rate+sound+body position	Apnomonitor Mini [44]	82.8	-	75.8	76	-	80.4	89.7	-	80.6	387
Airflow+SpO2+heart rate+sound+body position+body movement+effort	Nox T3 [40]	95	93	63	69	85	93	-	-	-	77
Effort	IR-UWB radar [61]	100	93	100	92	84	100	-	-	-	94
	Coronel et al. [62]	-	-	-	-	-	-	90	-	-	52

Categories	Device name or authors	Sensitivity (%)			Specificity (%)			Accuracy (%)			Number of patients
		AHI ≥5	AHI ≥15	AHI ≥30	AHI ≥5	AHI ≥15	AHI ≥30	AHI ≥5	AHI ≥15	AHI ≥30	
		Effort+SpO2	Coronel et al. [25]	80			91			88	
Effort+body movement	SleepMinder [63]	86.1	88.7	-	46.4	92.1	-	-	-	-	157
	Apnea App [52]	-	-	-	-	-	-	86.5			37
Effort+body movement+sound	Firefly App [51]	-	80	-	-	88.3	-	-	84.2	-	128
Sound	Rodriguez- Villegas et al. [64]	77.1			99.7			-			30
Sound (cardiac and breath)	AcuPebble SA100 [49]	-	92.7	-	-	96.8	-	-	95.3		150
Sound+effort	Ghahjaverestan et [65]	93.3	91.7	83.3	33.3	55.6	86.7	76.2	77.2	85.7	21
Position+sound+body movement	Clebre [66]	96			76			86			30
Effort+heart rate+sound+body movement	Within sleep analyzer [32]	88.7	88	86	75	88.6	91.2	-	-	-	118
SpO2+heart rate+Respiratory rate	GT2, Huawei Device Co [33]	76.5	85.7	80	100	100	80	88.3	92.9	80	20
Effort+SpO2+heart rate+PAT+sound+body movement+position	WatchPAT [47]	-	85	-	-	70	-	-	-	-	84
Effort+SpO2+heart rate+RR+body movement+position	Zavanelli et al. [26]	100			-	-	-	-	-	-	9
Heart rate variability+respiratory rate+PPG-sleep	Papini et al. [67]	72	59	41	71	90	98	-	-	-	502

Categories	Device name or authors	Sensitivity (%)			Specificity (%)			Accuracy (%)			Number of patients
		AHI	AHI	AHI	AHI	AHI	AHI	AHI	AHI	AHI ≥30	
		≥5	≥15	≥30	≥5	≥15	≥30	≥5	≥15		
pattern+body movement											
Effort+heart rate+SpO2+ sound+body position	ApneaLink Air, ResMed [42]	-	-	-	-	-	-	†80	-		20
Mandibular movements	Pepin et al. [68]	91	92	-	94	84	-	92	88	-	376
EEG	Zhao et al. [69]	-	-	-	-	-	-	#	##	###95.2	30
								80.4	84.9		

Note that portable monitor (PM)-AHI is different from the set cutoffs, e.g., PM-AHI of 10.3 events/h satisfies the AHI cutoff of 5 with a sensitivity of 82.8% and specificity of 76.0% [44]. Sound=breathing sound/snoring. Respiration=airflow or tidal volume; RR=respiratory rate. #, ##, ### refers to % accuracy for classifying breathing during sleep into OSA, central apnea and normal breathing rather than with respect to the AHI cutoffs under which they appear in the table. † refers to AHI ≥10. Note that where information is missing in the table for parameters, these were not presented in the respective original work. Some sensitivities/specificities/accuracies in the table do not fall under a particular AHI cutoff but entered under AHI ≥ 5 over all AHI categories, as they were simply reported as overall performance in the associated work.

The minimum essential physiological signals recommended for OSA detection are airflow, respiratory effort, and oxygen saturation [12]. However, other parameters have also been utilized for detecting respiratory events (apnea and hypopnea) with varying performance characteristics compared to standard PSG (Table 2) [12]. Upon closer examination of the different PMs, it is evident that the sensitivity, specificity and accuracy of a PM do not necessarily increase with an increased number of parameters. For example, some devices relying on respiratory movement (effort) alone or both effort and body movement tend to perform better than a device incorporating effort, body movement and breath sound [[51], [61], [63]]. On the other hand, when two PM devices share common physiological parameters, such as oxygen saturation and heart rate, the device that includes an additional parameter like respiratory rate or at least three additional parameters such as effort, peripheral arterial tonometry and sound, has shown superior performance compared to a PM lacking these additional parameters (Table 2) [33], [47], [59]. Specifically, for a pulse oximeter, adding SpO2 to pulse-to-pulse interval and pulse-derived respiratory effort improved the performance for OSA classification compared to the separate use of the last and both the latter parameters, which had equal performance [70]. This observation supports the notion that

incorporating some specific physiological parameters can enhance the overall performance of a PM, as similarly acknowledged by Alarcon et al. [71].

The reasons for variations in performance among PMs is not completely understood. There may be differences in the algorithms used to process physiological signals, which can impact the accuracy and performance of a PM [69], [72]. Additionally, several factors, such as the definition of apnea, inclusion or exclusion of central apnea, patient exclusion criteria, lab or home setting, and the study sample size, contribute to variations in PM performance at different AHI cutoffs compared to PSG (Table 2) [19,59,[44], [45], [52], [67]].

PSG utilizes various respiratory events, including central, obstructive, and mixed apnea, as well as hypopnea, in computing AHI [52,73]. OSA patients with predominant central apnea, as confirmed by simultaneous PSG data, are excluded from PM evaluation studies [44], given this has been shown to improve PMs performance as opposed to including such patients [45]. While some studies did not explicitly state whether they included or excluded central apnea when evaluating their PMs, suspected central apnea is a criterion that contraindicates using PMs for the diagnosis of suspected OSA, as the accuracy of PMs in detecting central apneas requires further investigation [12,27]. Two studies evaluating the Belun sleep PM, one involving OSA patients with heart failure, who are commonly more predisposed to central apnea [74], and another without, demonstrated a better performance in the absence of heart failure, as compared to its presence [45], [59]. Specifically, the studies showed that the sensitivity/specificity for  $AHI \geq 15$  was 93.1/73.5% without heart failure versus 62.1/79.6% with heart failure, while the sensitivity/specificity for  $AHI \geq 30$  was 74/95.3% without heart failure versus 50/67.2% with heart failure. This performance difference was attributed to the inability of the PM to detect central apneas [45].

It should be noted that multiple entries in Table 2 have performance metrics that were determined in the sleep lab and measured against simultaneous PSG. However, these performance metrics may differ if the PMs were tested at home in comparison to PSG [32,47,60]. Indeed, studies have shown that the performance of home-tested PMs is poorer compared to lab-tested PMs deployed simultaneously with PSG and has lower AHI correlation to PSG AHI [55,75]. For instance, ResMed ApneaLink had sensitivity/specificity of 79/88.2% for in-lab testing and 73.7/85.3% for home testing at  $AHI \geq 15$  [55]. This may be due to various factors, such as the presence of bed partners and the patient's habits, including drinking alcohol prior to bed at home and spending more time sleeping in the supine position in PSG than at home [75,76]. On the other hand, AccuPebble, tested in a home setting, showed high sensitivity and specificity to OSA detection, but this might partly be due to its evaluation relative to a home-deployed PM III rather than PSG [49]. It is worth noting that the use of a PM III, rather than PSG, as a reference precludes sleep staging and can therefore affect the accuracy of the reference total sleep time used [49], [75]. Additionally, the effects of night-to-night variability in the diagnosis of OSA severity is likely to play a major role in any comparison between devices conducted on separate nights and needs to be taken into serious consideration [77].

The composition and characteristics of sample size in PM studies can also potentially influence the reported PM performance. Large sample sizes can reduce the probability of reporting false negatives and positives [78] and contribute to the overall performance of PMs using similar physiological signals, such as sound (Table 2) [49,64]. Having a predominance of males in the sample could also impact performance. Crinion et al. [75] suggested that the presence of more males in a study sample may improve the performance of a PM. For example, at AHI >15, the accuracy for males was 72.3% versus 65% for females. According to the authors, this difference may be due to variations in physical body characteristics, particularly the abdomen and chest, which are the basis for respiratory event detection in some PMs [19]. In contrast, significantly less sleep efficiency in males compared to females can reduce the total sleep time, leading to an overestimation of AHI and reduced PM performance [79]. Also, especially in PMs that detect respiratory events based on heart rate variability, age-related reductions in sleep efficiency and frequent arousals, which are positively associated with age, can accentuate false positives and reduce the overall performance of PMs [67,79,80].

Therefore, several factors can jointly affect the performance of PMs. In the following sections, we review the parameters used in these PMs, which may be combined however necessary, based on how they are acquired and how they are related to OSA to calculate AHI. The limitations associated with harnessing some of these parameters are also highlighted.

## 5. Physiological parameters for deriving AHI

### 5.1. Nasal airflow

Nasal airflow during breathing is the volume of air passing per second through the nose and is measured directly using a pneumotachometer with differential pressure transducer and nasal mask or indirectly using an oronasal thermistor or nasal pressure transducer [81]. Although the pneumotachometer is considered the gold standard for airflow monitoring, the discomfort caused to subjects by wearing a mask has led to the use of nasal pressure transducer and oronasal thermistor for the clinical detection of hypopnea and apnea [22,82]. When using the airflow signal alone for OSA detection, a  $\geq 30\%$  decrease in airflow from baseline for  $\geq 10$ s is defined as hypopnea, while a  $\geq 90\%$  decrease in airflow from baseline for  $\geq 10$ s is an apnea [22]. PMs using only airflow can detect OSA with 60–90 % sensitivity and specificity with respect to AHI cut offs of 15 and 30 (Table 2) [34], [55].

### 5.2. Tidal volume

Tidal volume refers to the volume of air that is inspired or expired during a respiratory cycle. It is typically derived by integrating the nasal airflow signal or by detecting tracheal breath sound or chest movement, and intrathoracic electrical impedance oscillations [[56], [65], [83], [84]]. While tidal volume is not used for event detection in PSG, in portable monitoring, hypopnea events are

defined as a reduction of  $\geq 50\%$  or  $74\%$  in tidal volume from baseline and apnea events as absence of tidal volume for at least 10s [56,85].

Barbieri et al. [56] reported sensitivity of 80% and specificity of 60% for the APScan CIED PM based on changes in tidal volume. However, the AHI estimated by the device was poorly correlated to PSG-AHI ( $R=0.41$ ,  $p=0.07$ ). This poor correlation was attributed to the device's low success rate (42.8%) in detecting apnea events in patients with PSG-diagnosed central sleep apnea, who constituted  $\sim 50\%$  of the study population.

### 5.3. Respiratory rate

Respiratory rate (RR) is the number of breaths per minute. It can be obtained from various respiratory signals such as airflow, electrocardiogram (ECG), photoplethysmography (PPG), ballistocardiograph (BCG)-derived respiratory signals, chest bioimpedance, respiratory inductance plethysmography, impedance pneumography, chest piezoelectric signal and breath sound [[86], [87], [88], [89], [90], [91], [92]].

To automatically detect the respiratory rate, two consecutive peaks of inspiration in the respiratory signal are identified within a specific time interval (in seconds). The instantaneous respiratory rate is calculated as the reciprocal of this interval [90,93]. In OSA, where airflow decreases or ceases, the respiratory rate signal is disrupted. In such cases, an advanced approach such as characteristic moment waveform, is required to identify the peaks [89]. Using the approach, Fang et al. [89] defined a threshold respiratory rate to be stable RR – stable RR/60, such that sub-threshold RR lasting  $\geq 10$ s is scored as apnea and otherwise as a hypopnea when the subthreshold RR lasts  $< 10$ s. This method has not been compared with PSG for hypopnea and apnea detection but has a reported respiratory rate detection accuracy of 98.4% relative to manual counting [89].

### 5.4. Oxygen saturation

Oxygen saturation ( $SaO_2$ ) is the percentage of oxygen-containing hemoglobin in the blood, measured invasively via oximetry of arterial blood [94]. Peripheral oxygen saturation ( $SpO_2$ ) is a non-invasive measure obtained by pulse oximetry.  $SpO_2$  is commonly used in PSG given it has strong agreement with  $SaO_2$  [[94], [95], [96]]. In sleep-related breathing studies, oxygen saturation less than 90% is an indicator of hypoxemia, whereas restoration of  $SpO_2$  has been used as a measure of positive response to OSA treatment [96,97].

Recently,  $SpO_2$  features have been used to estimate AHI through machine learning-based detection of apnea and hypopnea events. This approach showed promising results with relatively high classification accuracy for OSA severity ( $\sim 70\text{--}90\%$ ) [58], [98]. However, non-OSA related desaturations can increase the number of false positives in an OSA patient and thus limit the performance of a PM that adopts only the  $SpO_2$  signal for hypopnea and apnea detection [98].

Monitoring SpO<sub>2</sub> in OSA patients has shown that nadir SpO<sub>2</sub> < 90% during sleep is associated with an increased risk of myocardial infarction of up to double, particularly in patients with a mean heart rate >73 beats per minute or minimum heart rate >60 [99]. In women, but not men, nadir SpO<sub>2</sub> is associated with the incidence of cardiovascular disease in OSA patients [100]. Further research is needed to understand why SpO<sub>2</sub> features could determine the potential for female OSA patients, but not males, in developing cardiovascular diseases.

## 6. EEG

The EEG, or electroencephalogram, measures the combined electrical activity of inhibitory and excitatory post-synaptic potentials from various pyramidal neuronal populations via electrodes placed on the human scalp [101]. It is used in PSG for detecting cortical arousal and sleep staging [18]. Arousal is defined as a sudden change in EEG frequencies greater than 16Hz (excluding spindles), lasting at least 3s and preceded by at least 10s of stable sleep. In individuals with OSA, a respiratory related arousal occurs at the end of a hypopnea or apnea event, followed by recovery of airflow and oxygen saturation level [102]. The magnitude of the arousal, known as arousal intensity, is associated with increased pre-arousal respiratory effort during an OSA event [103].

EEG has also been used independently to detect respiratory events and categorize OSA based on selected features, such as sample entropy and variance of different frequency sub-bands (alpha, theta, delta, beta, and sigma) of the EEG signal, using machine learning techniques [69]. These methods have achieved accuracies of ~80–99% for distinguishing OSA from normal breathing, central sleep apnea and mixed apnea [69], [72]. However, additional studies are needed to determine the performance of these techniques for classifying OSA severity. Currently, only one study has shown the sensitivity and specificity of EEG in detecting mild OSA (86 and 84.4%, respectively), although this study used body movement and pulse oximetry in addition to EEG [104].

### 6.1. Respiratory effort

Respiratory effort refers to the activity undertaken by the muscles of respiration, including the rib cage muscles, diaphragm and abdominal muscles, to drive ventilation [105,106]. It is typically measured using esophageal manometry, which involves passing an esophageal balloon or pressure transducer-tipped catheter through the nose into the esophagus to measure intrathoracic pressure [22,107,108]. Alternatively, respiratory effort can be estimated by measuring epiglottic pressure [109]. However, due to the invasiveness of esophageal manometry (Peso) and epiglottic catheter (Pepi), respiratory effort is often measured non-invasively using respiratory inductance plethysmography belts that indicate volumetric changes due to chest and abdominal movements or from suprasternal pressure or by accelerometer on the chest [22,23,110]. Other approaches for detecting respiratory effort are non-contact monitoring of the movement of the abdomen and/or

chest by 3D time of flight (TOF) infrared cameras, and using doppler-effect based thoracoabdominal movement detection by radiofrequency, ultrawide radar and sonar systems [19], [52], [61], [62].

During a respiratory event, the nadir Peso or Pepi becomes more negative, indicating greater effort in breathing and that the sleep apnea is obstructive rather than central [23]. In scoring these obstructive sleep apnea events, the effort surrogate “thoracoabdominal movement” has been used to determine apnea and hypopnea events as corresponding to at least a 90% and 50% decrease in baseline abdominal or chest movement lasting  $\geq 10$ s, respectively [61,62]. Using only respiratory movements, sensitivity and specificity up to 93% and 84%, respectively, at an AHI of at least 15 events/hr and overall accuracy of 90% have been achieved for classifying respiratory events as apneas and hypopneas [61,62]. In fact, a recent meta-analysis concluded that non-contact PMs, some of which used respiratory movements for detecting OSA, had an overall low risk of bias for sleep apnea assessment in terms of their validity relative to PSG [111].

## 6.2. Snoring/breath sound

Snoring is the inspiratory and/or expiratory noise associated with narrowing and vibration of upper airway soft tissue structures during sleep, including the soft palate, pharyngeal walls, epiglottis, and tongue [112,113]. These loud breathing noises can occur without apnea or hypopnea [114]. Snoring and breath sound can be detected with a microphone placed above or under the bed [115,116], underneath a mat using digital stethoscope-based technology [117] or using tracheal sound sensor attached to the suprasternal notch [118]. Both breath cessation and reduction are reflected in the breath sound amplitude and snoring intensity [118]. Snoring intensities are usually measured in decibels as the mean maximal snoring intensity and used to classify snoring as mild (40–45 dB), moderate (45–55 dB), severe (55–60 dB) and very severe ( $\geq 60$  dB) [119].

Breath/snoring sound and the pressure at the trachea can be simultaneously detected by a suprasternal sound/pressure sensor, allowing for the distinction of obstructive apnea from central apnea, with the suprasternal/tracheal pressure serving as a surrogate for effort [118]. Additionally, tracheal movements and breath signals fed into a multivariate linear regression model and synergized with snoring features have been used to determine AHI [65]. Tracheal movements are the range of motions that the trachea undergoes during breathing, and they can be estimated via accelerometer measurements [84], although imaging would provide more accurate readings [120].

Snoring features have been used to detect respiratory events and classify OSA severity. These features include mel-cepstability, running variance, apneic phase ratio, pitch, pitch density, inter-respiratory event silence duration, formant frequency distribution and volume information, which are described further in the following references [65,115,121]. These features are commonly calculated for OSA patients and then combined in a multi-variate regression model to be correlated to PSG-AHI [65,115]. A combination of mel-cepstability, running variance, apneic phase ratio, pitch density, inter-respiratory event silence duration has achieved OSA detection with sensitivity and specificity of 87% and 80%, respectively, at AHI  $> 10$ , and sensitivity and specificity of 89% and 78%

for AHI >20 [115]. Montazeri Ghahjaverestan et al. [65] showed that incorporating melcepstability, running variance, pitch density to breath sound and tracheal movements improved sensitivity and accuracy of OSA detection at AHI >15—~87% and 84% from ~67% to 79% (before the addition).

Dips in the snoring sound dB-power time series have been used to detect respiratory events, based upon which AHI was defined as the total number of dips per hour of snoring time [122]. This approach was shown to attain sensitivity of 70% and specificity of 94% at AHI  $\geq 15$ , with an overall strong correlation with the PSG-AHI ( $R=0.94$ ). Similarly, snoring episode index, defined as the number of snoring episodes per hour of snoring, was significantly correlated to AHI ( $R=0.85$ ), although it was not intended for OSA detection [123].

Some snore features such as formant frequency and volume information features of snoring have been reported to have poor correlation with the snore-specific AHI (ssAHI; correlations  $<0.3$ ) [121]. ssAHI is the total number of apneas and hypopneas during the hour prior to the start of a snoring episode. Despite its poor association with formant frequency and volume information, the new metric ssAHI showed the potential of classifying snoring as mild or severe. In another study, the formant distribution was used to create a map of snore types, including type 1 called monosyllabic low-frequency snore, type 2 called duplex low-and-mid frequency snore, type 3 called the duplex low and high-frequency snore, and type 4 called the triplex low, mid and high-frequency snore [124]. Types 3–4 were shown to be significantly correlated to AHI ( $R=0.52$ ,  $p=0.026$ ), although the study was conducted on only males using small sample size of 20.

Limitations associated with the use of snoring-based PMs for detection of OSA include BMI and neck circumference, which can attenuate snoring features such as intensity [124]. Although the snore features described here are not intensity-dependent, except sound power, further study on the influence of BMI and neck circumference on the snoring-based PM performance is required. Another challenge in using snoring signal in the detection of OSA is that signal loss due to change in sleep positions may occur when a non-contact microphone is used to acquire the signals. The latter requires that the sensor be attached to the neck, at the level of the trachea for instance, to minimize the aforementioned potential signal loss [65].

### 6.3. Heart rate variability and pulse rate variability

Heart rate variability (HRV) can be measured from the ECG signal [125], while pulse rate variability (PRV) can be derived from photoplethysmography (PPG). PPG involves the transmission and reflection of red and infra-red light to sense the pulsatile blood volume changes in microvascular beds [[126], [127], [128]]. PRV uses the pulse-to-pulse interval (PPI) of the PPG signal [129]. HRV is estimated by analyzing the variation of a set of R–R intervals (RRI) of the ECG signal over  $\leq 5$ -min epoch [130]. PRV has been proposed as a close estimate of HRV [131].

The decrease in heart rate during respiratory events and the increase in heart rate following the events with or without arousals contribute to heart rate variability [103,[132], [133], [134]]. Lower

HRV is a characteristic of more severe OSA [135], and night-to-night decrease of HRV can suggest worsening OSA severity [136]. In addition to sleep staging, features such as arousal probability, frequency analysis, sample entropy extracted from HRV have been used to detect respiratory events and determine AHI using machine learning [67,80]. Chang et al. [137] achieved sensitivity of 69% and specificity of 100% for the detection of OSA at AHI >15 using the low-frequency component of HRV with or without other HRV features. OSA detection by HRV may be affected by gender and age, thus requiring wide testing of HRV-based PM for thorough performance evaluation [79].

## 6.4. Mandibular movements

Mandibular movement refers to the peak-to-peak movement of the mandible that has been shown to occur during obstructive events or arousal [138]. This movement can be measured with magnetometer attached to chin and forehead, or an accelerometer/gyroscope attached to chin [138,139]. Mandibular movement opening (MMO) is defined as the lowering of the mandible by  $\geq 0.3$  mm lasting for at least two respiratory cycles, while sharp upward mandibular movement (MMS) is defined as movement amplitude  $> 1$  mm and/or  $\geq$  twice the previous or the following mandibular movement amplitude [138]. The authors showed that a significant association exists between MM and effort, as well as AHI, and MMS is associated with arousal following MMO.

Further studies have shown that mandibular movements can be utilized as a surrogate of esophageal pressure, due to the existence of pattern match between the two signals, to measure and discriminate between different respiratory efforts in relation to obstructive apnea, mixed apnea, central apnea, central hypopnea and obstructive hypopnea [139,140]. For OSA detection at AHI cutoff of 15, Pepin et al. reported high sensitivity (92%), specificity (84%) and accuracy (88%) using an MM-based PM [68]. However, according to the authors, this method may be limited by excess fat on the neck and friction between MM sensors and pillow or mattress as patients change sleep positions.

## 7. Physiological parameters not used to independently derive AHI

There are several physiological parameters that are not used separately in determining AHI. However, they change in response to respiratory events and can facilitate the events identification.

### 7.1. Heart rate and pulse rate

Heart rate is measured as the number of times the heart beats per minute, and it is conventionally measured from ECG [125]. Another method for detecting heart rate is ballistocardiography [141], which measures the movement of the body's center of mass caused by acceleration of blood into the great vessels in the body due to motion of the heart [142]. Pulse rate is a measure of heart rate that is derived from the PPG signal [127]. To calculate heart rate or pulse rate in beats per minute, the R-R interval or P-P interval, respectively, is measured in seconds on the corresponding waveform and used to divide 60 [143].

While heart rate and pulse rate are related, they are not the same. Pulse rate is generally considered to be a more peripheral measure of heart rate, whereas ECG-derived heart rate is considered to be more representative of the heart's activity [144]. However, in many cases, the terms heart rate and pulse rate are used interchangeably, as they are highly correlated in both people with and without OSA (correlation coefficient=0.95–1) [145,146].

During a respiratory event, heart rate typically decreases and then increases when airflow resumes or after arousal [132,147]. This change in heart rate is used in conjunction with peripheral arterial tonometry to score these events and assess their severity [148].

## 7.2. Peripheral arterial tonometry

Peripheral arterial tonometry (PAT) is a non-invasive method that involves applying a uniform pressure field (e.g., 40mmHg) to the finger and measuring the pulsatile volume changes in the vascular beds of the finger using optical sensors [149]. The pressure applied is aimed at increasing the amplitude of the pulse wave signal without blocking venous return or causing the device to slide off the finger [149,150]. PAT signal amplitude decreases due to vasoconstriction, which can occur with or without arousal following respiratory events [133,134]. Based on this premise, apnea and hypopnea events can be scored when decrease in PAT amplitude is accompanied by an increase in heart rate or body activity count, or when a decrease in PAT amplitude occurs together with  $\geq 3$  or 4% OD [148].

## 7.3. Body movement

Body movement is a useful indicator of sleep state and can serve as a sleep surrogate for EEG measurements [17]. It provides information that helps discriminate between wakefulness and sleep and is used to determine the effective total sleep time for AHI computation [17,151]. By measuring body movement, physiological signals, such as airflow and SpO<sub>2</sub>, can be segmented and accurately scored as sleep events, while arousals or gross body movement can be excluded. Body movement can be measured by 3D accelerometer placed on the wrist [47,67].

## 7.4. Body position

Positional body orientations during sleep, including supine, prone and lateral positions, can be measured by accelerometer and/or gyroscope attached to the neck, wrist, or chest [[151], [152], [153], [154]]. Supine position is known to be associated with a higher incidence of OSA than non-supine positions [21]. If a patient's AHI is at least twice as high in the supine position compared to the lateral position, they are classified as having positional OSA [21]. This is related to the increased susceptibility of the upper airway to collapse under the effect of gravity in the supine position [155]. Accurate tracking of sleep positions is therefore important to prevent overestimation of AHI [75].

## 8. Advantages and limitations of physiological parameters

Physiological parameters can be measured from one or more body parts, depending on the type of sensor used and measurement required. For instance, ECG can only be acquired from the chest, while PPG signals can be obtained from different parts of the body, including the finger, wrist, or forehead [126]. Moreover, PPG signals have multiple applications, including the measurement of pulse rate, pulse rate variability, cyclic variability of heart rate, and oxygen saturation.

Nonetheless, all sensors have some limitations, such as susceptibility to surrounding noise, motion artefacts, or causing discomfort to the patients. Therefore, it is important to consider both the advantages and limitations of each sensor to choose the appropriate one for a particular application. A summary of the advantages and limitations can be found in Table 3.

Table 3. Physiological parameters used for OSA detection.

Parameters	Sensors	Body location	Advantage	Disadvantage
Pulse rate	Pulse oximeter (from PPG)	Mostly finger, wrist, back of the neck, or forehead [126].	The signal can be obtained from many parts of the body.	Cardiac arrhythmia and heart failure disease can affect the effectiveness of the device for OSA detection [47,67]; clips affixing the sensor to the finger may be disturbing [49]; susceptible to motion artefacts and surrounding noise [156].
Heart rate signals	ECG	On the chest	Restricted to the chest.	Requires patients taking off their tops; placement of electrodes on the chest may cause skin rashes or irritation.
Oxygen saturation	Pulse oximeter; arterial bloodgas spectrophotometry.	finger, wrist, or forearm.	Non-invasive determination of oxygen desaturation; and used to estimate hypoxic burden.	SpO2 may be overestimated in smokers [94]. Non-OSA related desaturations can increase false positives [98].
Sound	Microphone	Suprasternal notch or place above	Both snoring and breath sound signals can be	Anthropometric factors such as height, neck

Parameters	Sensors	Body location	Advantage	Disadvantage
		patient near the head.	simultaneously acquired.	circumference, or BMI may affect signal quality [157].
Respiratory movement	Timeofflight camera, sonarsystem, accelerometerfor breathing movement.	Thoracoabdominal region [52,110]	Breathing movement detectable without contact to the body using cameras or radio frequency signals.	Attaching accelerometer to the abdomen/thorax may initially cause discomfort; camera sensing may be particularly more sensitive to central apnea [62].
Body movement	Accelerometer;sonar system.	Wrist, thoracoabdominal movement [47,52,67]	Body movement detectable from multiple parts, with/without contact.	Discomfort may be initially associated with fixing an accelerometer to a body part.
Airflow	Nasalpressure transducer;oronasal thermistor;suprasternal microphone.	Nose,mouth, neck [118].	Use of two types of the transducers ensures discrimination between apnea and hypopnea.	Respiratory effort cannot be obtained from only nasal pressure and thermistor [55].

The sensors commonly used to acquire the signals and the location on the body, advantages and limitations are summarized.

### 9. Non-AHI metrics

Non-AHI metrics are important physiological measures that can provide additional information about the pathophysiology and impact of OSA beyond the standard measure of AHI and help guide individualized treatment decisions. Some of these metrics include the now well-known OSA endotypes (upper airway collapsibility, muscle responsiveness, arousal threshold and loop gain), cyclic heart rate variability, oxygen desaturation index, apnea hypopnea time and hypoxic burden index. Many of these parameters show strong associations with AHI, which are summarized in [Table 4](#). The relationship between non-AHI metrics with OSA is briefly described below.

Table 4. Correlation between non-AHI metrics and AHI.

Non-AHI metric	AHI	Number of patients	Authors
CVHR index	0.95 <sup>a</sup>	63	Hayano et al. [158]
	0.81 <sup>a</sup>	41	Hayano et al. [159]
	0.65 <sup>b</sup>	119	Hsu et al. [132]
ODI	0.93 <sup>c</sup>	65	Rosa et al. [160]
Obstruction severity	0.78 <sup>a</sup>	19	Kulkas et al. [161]
HBI	0.70 <sup>b</sup>	2743	Azarbarzin et [162]
	0.69 <sup>b</sup>	459	Chen et al. [163]
ArTh	0.37 <sup>a</sup>	127	Edwards et al. [109]
Pcrit	0.66 <sup>c</sup>	25	Wellman et al. [164]
Loop gain	0.88 <sup>c</sup>	25	Wellman et al. [164]

Superscripts for correlation: a=linear regression; b=Spearman rank correlation; c=Pearson correlation; CVHR=cyclic variation of heart rate; ODI=oxygen desaturation index; AHT=apnea hypopnea time; HBI=hypoxic burden index; ArTh=arousal threshold. Pcrit=critical pressure. All correlations are significant. Loop gain was associated with AHI at atmospheric Pcrit [164].

## 9.1. Metrics for OSA endotyping

Endotyping is an approach that categorizes OSA (obstructive sleep apnea) patients based on their underlying pathophysiological traits. These traits include highly collapsible upper airway (Pcrit), poor upper airway muscle responsiveness, high ventilation instability (loop gain), and low arousal threshold. The endotyping approach, described by Eckert et al. helps in classifying OSA patients into different subgroups [165]. For instance, a group with relatively low Pcrit (-5 to -2 cmH<sub>2</sub>O) may have the highest loop gain and arousal threshold, but the least muscle responsiveness. On the other hand, a group with Pcrit > +2cmH<sub>2</sub>O may have the least arousal threshold and the highest muscle responsiveness.

By understanding these endophenotypes of OSA, patients can be stratified as responders and non-responders to specific treatments. For example, some patients may respond to treatments such as eszopiclone (a sedative) or supplemental oxygen, while others may not [166,167]. Furthermore, the knowledge of endophenotypes has implications for treatment selection. OSA patients with better passive upper airway collapsibility and lower loop gain have been found to achieve a significant reduction in AHI (apnea-hypopnea index) of approximately 67% in response to mandibular advancement treatment [168]. This indicates that treatment outcomes can vary depending on the

specific endophenotypic characteristics of the individual patient. The limitations of the treatment interventions applied to manage these endotypes have been discussed elsewhere [169].

The four endo/phenotypic parameters generally require CPAP and/or invasive upper airway muscle activity recording to be determined [165,170]. Therefore, efforts have been made to develop simpler and less-invasive approaches to acquire these parameters, which are described in the following subsections.

## 9.2. Upper airway collapsibility

Upper airway collapsibility is typically measured by the critical closing pressure, or Pcrit, which is defined as the nasal pressure at which the pharyngeal airway collapses, and results in the cessation of airflow [3]. A higher Pcrit value indicates greater tendency for the upper airway to collapse. To measure Pcrit, nasal pressure is gradually decreased using a modified CPAP device to induce narrowing of the upper airway [3,20].

Initially, a holding nasal pressure is titrated for the participant to eliminate flow-limitation and reduce neuromuscular activity. Next, the nasal pressure is abruptly reduced in increments of 1–2 cmH<sub>2</sub>O until flow-limitation occurs for 5 breaths, after which the pressure is returned to the holding pressure for 1–2 min. The process is repeated with additional CPAP drops in steps of 1–2 cmH<sub>2</sub>O, recording flow-limitation until zero or near zero flow is achieved. The nasal pressure value at zero flow (extrapolated from the obtained flow-pressure data) is known as the passive Pcrit. To obtain active Pcrit, flow-limitation following pressure drops is left to persist for 10 min during NREM sleep and further drops are then applied until recurrent obstructive apneas occur or sleep is no longer sustained. In ensuring accurate determination of Pcrit, algorithms have been developed to accurately detect breaths including inspiration and expiration onsets from airflow signals, while minimizing the effect of noise and baseline drifts during CPAP maneuvers [171].

Pcrit is significantly and positively correlated with AHI (Pearson correlation=0.66), making it an indicator of OSA severity [3,164]. However, determining Pcrit with the above standard method is time-consuming, laborious, and not clinically feasible. To simplify the process, a method has been recently developed by Azarbarzin et al. that utilizes traditional PSG data to derive mid-inspiratory and peak inspiratory airflows from pneumotachograph [172]. The method showed a significant correlation between active Pcrit and peak inspiratory flow ( $R=-0.71$ ,  $p<0.05$ ) and mid-inspiratory flow ( $R=-0.64$ ,  $p<0.05$ ), respectively. Another study has also formulated a model for estimating Pcrit from percentage peak inspiratory flow (PIF) with a significant  $R^2=0.71$  [173]. Percentage PIF was defined as the average PIF of all 3–5 breaths without arousal at each of CPAP drops to zero level expressed as percentage of the average PIF of all 5 baseline breaths preceding each of the CPAP dial-downs.

However, both methods described above are limited because they are estimated only during sleep. A wakefulness/daytime method of estimating the pharyngeal collapsibility has been presented by

Osman et al. requiring brief application of negative airway pressure pulses of about  $-12$  cmH<sub>2</sub>O [174]. The authors showed that wakefulness upper airway collapsibility index (UACI), derived from nadir choanal and corresponding epiglottic pressures, was significantly correlated with Pcrit evaluated at NREM sleep ( $R=0.8$ ). Translation of this approach to portable monitors is also limited because choanal and epiglottic pressures are invasive and are not routinely measured during PSG.

Alternatively, nasal pressure-transducer-derived ventilation was synchronized with absence or presence of EEG arousals and shown as a portable surrogate of CPAP for estimating the upper airway collapsibility [175]. This method defined upper airway collapsibility during sleep as passive ventilation, i.e., the reduced ventilation at normal ventilatory drive, indicating a more or less collapsible airway. Ventilatory drive is the intended level of ventilation as influenced by the blood oxygen-carbon dioxide changes and response to arousal input [176]. Sands et al. used passive ventilation to categorize airway collapsibility into high, moderate, and low endotypes, with high percentage value of passive ventilation indicating less airway collapsibility [175]. The authors showed that passive ventilation strongly correlated with peak diaphragm-EMG ( $R=0.83$ ), which is the gold standard for measuring ventilatory drive.

### 9.3. Arousal threshold

Arousal threshold is the negative intrathoracic pressure related to triggering cortical arousal [103,165], which is commonly measured as the nadir esophageal or epiglottic pressure immediately prior to arousal [177]. A low arousal threshold is typically defined as an epiglottic pressure between 0 and  $-15$  cmH<sub>2</sub>O, while a high arousal threshold is considered to be an epiglottic pressure less than  $-15$  cmH<sub>2</sub>O [166]. Alternatively, non-invasive methods such as nasal pressure ventilation and screening scores have been used for the prediction of arousal threshold. The nasal pressure-estimated arousal threshold was defined as the median ventilatory drive immediately preceding EEG arousals [178]. The screening-score method used the criteria of at least two of  $AHI < 30$ , nadir  $SpO_2 > 82.5\%$ , and  $> 58.3\%$  hypopneas in all respiratory events to predict a low arousal threshold with 80.4% sensitivity and 88% specificity in  $\sim 84\%$  of the study population [109].

Individuals with a low arousal threshold experience repeated apnea and hypopnea events because the events, when they occur, are terminated quickly at a low breathing effort [102,166]. Although arousals help to restore oxygen saturation levels after obstructive events, frequent arousal deprives patients of deep sleep [102]. Additionally, frequent arousal can potentially cause upper airway muscle disturbance since arousal leads to the activation of upper airway muscle [103]. On the other hand, OSA patients with high arousal threshold require higher respiratory effort to be cortically aroused during apnea and hypopnea events and are thus predisposed to blood gas imbalance (i.e., CO<sub>2</sub> and O<sub>2</sub>) [166]. Therefore, understanding an individual's arousal threshold can help personalize their treatment, e.g., the use of sedatives like eszopiclone to reduce AHI in OSA patients with low arousal threshold as eszopiclone increases/improves the patients' arousal threshold [166].

## 9.4. Loop gain

Loop gain is a respiratory control parameter that quantifies the ventilatory response to a disturbance in airflow caused by an obstructive respiratory event [176]. Thus, it is calculated as the ratio of the ventilatory response to the disturbance in ventilation, typically measured through drops in CPAP to cause airway narrowing/obstruction followed by CPAP restoration [170]. High loop gain is undesirable as it leads to ventilation instability [179]. Loop gain has been correlated with upper airway collapsibility. That is, it has been reported that loop gain in OSA patients with  $P_{crit} < -2 \text{ cmH}_2\text{O}$  is about two times as much as those with  $P_{crit} > +2 \text{ cmH}_2\text{O}$ , suggesting that high loop gain is indicative of an upper airway with less collapsibility [165]. It has further been shown that OSA patients with high loop gain and response to supplemental oxygen, such that their AHI was reduced by  $\geq 50\%$ , demonstrated less airway collapsibility [167]. In fact, Edwards et al. [180] showed that acetazolamide significantly reduced loop gain by  $\sim 41\%$  in OSA patients with high baseline loop gain, while also decreasing their AHI but without significantly changing airway collapsibility [180]. Terrill et al. have developed a simplified method to quantify loop gain (or a representative measure) from standard PSG data, promoting regular calculation of the metric [176].

## 9.5. Upper airway muscle responsiveness

Upper airway muscle responsiveness refers to the ability of upper airway muscles, such as the genioglossus and tensor palatini, to adjust their activity in response to respiratory stimuli [103]. Studies have shown that in individuals with OSA, these muscles exhibit decreased EMG activity during cyclic breathing compared to stable breathing, and during OSA events in REM relative to stage 2 sleep [181].

Passive ventilation of healthy subjects with CPAP has been shown to decrease EMG activity in both the genioglossus and tensor palatini during transitions from wake to sleep and when comparing NREM and REM sleep to sleep onset in the tensor palatini [182]. On the other hand, activity in these muscles is increased approximately 2–3 folds in response to reduced CPAP levels [103,183]. Failure of these muscles to respond to the respiratory stimulus increases airway collapsibility [3,184].

Measuring upper airway responsiveness or compensation involves invasive techniques such as EMG, which limit its determination non-invasively or outside of the laboratory. This may be alternatively assessed using the Sands et al.'s nasal pressure transducer approach described earlier under upper airway collapsibility [175]. The authors defined muscle compensation as the difference between passive ventilation and active ventilation, which is the level of ventilation at the arousal threshold and showed that it had a significant correlation with the peak diaphragm-EMG ( $R=0.76$ ). Greater baseline muscle compensation with better passive collapsibility and/or high loop gain are predictors of responders to the supplemental oxygen treatment [167]. Additionally, the timing of respiratory-related arousals (i.e. inspiration vs. expiration) could potentially be incorporated as a predictor of muscle responsiveness, particularly for the tensor palatini [177].

## 9.6. Cyclic variability of the heart rate index

Cyclic variability of the heart (CVHR) refers to cyclical pattern of heart rate changes during apnea and hypopnea events in individuals with OSA. During apnea and hypopnea events, the heart rate decreases, followed by an increase in the heart rate when the events are terminated with resumption of airflow [132]. This pattern is identified as an R–R dip distinct from other pseudo-dips within a threshold window, typically with an average width of 6min, along the R–R interval waveform [[158], [159], [185]].

The CVHR index is calculated as the number of CVHRs per hour of sleep [158]. The correlation between CVHR index and AHI is very high ( $R=0.95$ ), not significantly affected by presence/absence of heart failure, and the CVHR amplitude (effective RRI dip) can predict heart failure-related mortalities [185,186]. Home-based CVHR has significant correlation with tau protein level, as a measure of neuro degenerative disease in OSA patients ( $R=0.53$ ), such that the protein level is significantly higher in severe OSA compared to normal-to-moderate severity [187]. Further research was suggested to determine how habits such as cigarette smoking and alcohol intake will impact the tau level and thus the CVHR-tau protein correlation. It is also important to note that CHVR can be impacted by periodic limb movements [185].

## 9.7. Oxygen desaturation index

Oxygen desaturation (OD) refers to the decrease in the oxygen saturation ( $SpO_2$ ) signal from the baseline (mean  $SpO_2$ ) before a respiratory event to the lowest  $SpO_2$  following the event [22]. To score hypopnea, at least a 30% decrease in baseline nasal airflow lasting  $\geq 10s$ , accompanied with  $\geq 3\%$  OD, is recommended [22]. The number of times that desaturation events occur per hour of sleep is known as the oxygen desaturation index (ODI). While a 3% OD is commonly used, higher ODIs based on 5% OD have been shown to offer protection against the hazard of developing cardiovascular disease in women with OSA (but not men) [100]. ODI has also been used to predict OSA in children, elderly individuals, and obese patients [[160], [188], [189]].

Recent studies have presented and defined additional indices of oxygen desaturations, which are obstruction severity and desaturation severity indices [161,190]. Obstruction severity was defined as the sum of all the products of area under the desaturation curve and the corresponding hypopnea or apnea duration per unit hour of sleep [161]. Desaturation severity, which has similar meaning as hypoxic burden index (described in [subsection 9.8](#)), was defined by the authors as the sum of desaturation areas associated to respiratory events per unit hour of sleep.

Kainulainen et al. [190] found that a 10% increase in desaturation severity is a risk factor for moderate daytime sleepers to fall asleep in 5–10min following laying down to sleep [190]. They also showed that a 10% increase in both desaturation and obstruction severities is a risk factor for severe daytime sleepers to fall asleep in  $\leq 5$ min [191]. Additionally, a 10% increase in desaturation depth or desaturation severity significantly raised the odds for having a slow reaction time to psychomotor

vigilance tasks. These findings present a new paradigm for evaluating daytime sleepiness and physical alertness in OSA patients, although they may be limited by some non-OSA related desaturations [98].

## 9.8. Hypoxic burden index

The hypoxic burden index (HBI) is a metric that considers the respiratory event-related desaturation durations and depths in the SpO<sub>2</sub> waveform. HBI is defined as the sum of all the individual areas under the desaturation curves associated with hypopnea and apnea events per hour of sleep [162]. With focus on hypoxia, typically described as SpO<sub>2</sub> < 90%, Chen et al. defined HBI as the summation of all the different areas below 90% saturation line which is bounded by the desaturation curves [163]. It is important to note that HBI differs from desaturation severity, because an increase of 10% is required to define the physiological implication of desaturation severity on daytime sleepiness [190].

HBI has been shown to be associated with AHI, nadir SpO<sub>2</sub>, as well as the total time spent sleeping during which SpO<sub>2</sub> < 90% (Spearman correlation=0.690, 0.733 and 0.801, respectively) and is considered a risk factor for cardiovascular diseases [163]. A prospective cohort study is required to establish causality between hypoxic burden before and after cardiovascular disease occurrence [163]. In addition, OSA patients with HBI >1 have been found to have significantly elevated levels of vascular endothelial growth factor (VEGF-A) following myocardial infarction, which may suggest cardiac remodeling in response to myocardial infarction [192]. Lastly, HBI has been linked to cardiovascular disease-related deaths in a prospective cohort study involving individuals with OSA, who exhibited a negative HBI-to-survival probability relationship [162].

## 9.9. Apnea hypopnea time

The apnea-hypopnea time (AHT) is a metric that was recently introduced and described in a paper by Ma et al. [193]. AHT represents the total duration of apnea and hypopnea events per hour of sleep, expressed as a percentage. The metric was shown to be significantly correlated (R=0.889) to the total sleep time spent with SpO<sub>2</sub> levels below 90% and therefore a useful tool for evaluating nocturnal hypoxemia in patients with OSA.

In addition, a study of 17 OSA patients who underwent surgical treatment showed a significant average increase in mean-SpO<sub>2</sub> (by 2.8%) following a significant mean reduction in obstructive apnea index by 36/h and apnea duration per event by 8.5s [194]. This finding supports the usefulness of the event duration captured by AHT. Note that the obstructive apnea index is defined as the total number of apneas per hour of sleep.

## 10. Discussion and future direction

This review article has discussed the physiological parameters involved in calculating the AHI and diagnosing OSA. PSG makes use of a variety of signals for the clinical measurement of AHI, including airflow, effort, oxygen saturation, EEG, EOG, and EMG. PMs have emerged as a potential alternative to in-lab PSG for diagnosing OSA. PMs can use as many or as few parameters as PSG for symptomatic patients, with the first three parameters being the minimum recommended [12,13,22,28]. The number of parameters used by a PM does not necessarily influence their performance [[51], [61], [63]]. However, the inclusion of certain parameters such as respiratory rate, effort, oxygen saturation, peripheral arterial tonometry and sound can enhance PM accuracy [33,59,47].

The choice of PM can help discriminate different types of sleep apnea. PM III, with its chest/abdomen channels, can score obstructive apneas and obstructive hypopneas by breathing effort [23]. It can identify obstructive hypopnea by presence of flow limitation and out-of-phase chest/abdomen oscillations on the airflow and chest/abdomen channels, respectively, and detect central hypopnea by absence of flow limitation and presence of in-phase oscillations [23,195]. Although PM IV, comprising an airflow channel, can also detect presence/absence of airflow limitation, PM III, unlike PM IV, which lacks the chest/abdomen channel for effort detection [13], can additionally distinguish obstructive apnea from central apnea in which there is no effort [23].

According to the American Academy of Sleep Medicine recommendation, the presence of dominant central apnea should not be used as a criterion for diagnosing OSA using PMs [12,27]. However, PM III, unlike PM IV, has the ability to identify central apnea, which makes it potentially useful for quickly excluding central sleep apneic patients from PM-based diagnosis, especially in situations where PSG is unavailable. Additionally, PM III is set as a quicker and simpler choice for rapid OSA diagnosis compared to PM II, as PM II requires setup by a clinical technician [29]. With PM III, patients can set up the device themselves after brief instruction from the clinician [30].

Beyond OSA diagnosis, the physiological signals typically acquired in PSG have been utilized to stratify OSA patients based on their pathophysiology and enhance personalized treatment. This stratification is achieved through the evaluation of the four endotypic traits [165]: upper airway collapsibility, muscle responsiveness, arousal threshold and loop gain. While these four traits are traditionally obtained from CPAP and/or invasive measurements during PSG, recent advancement have allowed for their non-invasive derivation from nasal pressure and EEG [175,178].

A PM III device (ResMed's ApnearLink Plus/Air), without EEG, has been utilized to estimate loop gain from the ventilatory signal [196]. This estimation is facilitated by monitoring breathing effort and oxygen saturation through the chest/abdomen and pulse oximetry channels. By doing so, ventilatory disturbances such as apnea/hypopnea can be classified as obstructive or central, and the ventilatory response to blood gas imbalances due to these disturbances can be observed [167,175]. Orr et al. conducted a study comparing loop gain values derived from PM III with those from PSG and found that for a specific average loop gain there was no significant bias between the two methods [196]. Additionally, PM III-estimated loop gains showed a significant moderate correlation

( $R=0.47$ ) with PSG-derived loop gain. Interestingly, this correlation coefficient increased even further when sleep stages and arousal were removed from the PSG data to approximate portable home sleep apnea monitoring ( $R=0.66$ ), which typically does not include EEG. Moreover, it is worth noting that no significant differences were observed in the respiratory events and oxygen saturation levels detected using both PM and PSG approaches. This suggests that PM III can be a reliable alternative when EEG data is unavailable for loop gain estimation and monitoring respiratory events during sleep.

A more recent study by Schmickl et al. has provided a promising approach for PM assessment of loop gain [197]. They developed a regression model based on the measured AHI and fraction of hypopneas present in the overall respiratory events. The model was moderately successful at predicting loop gain ( $R=0.48$ ), with no significant bias with the PSG-derived loop gain. The model also demonstrated a fair power of 73% to discriminate between high and low loop gains. However, it is essential to acknowledge that the accuracy of this approach may be limited due to the variability among loop gain estimates using different hypopnea scoring criteria. These criteria include  $\geq 3\%$  ODI with  $\geq 30\%$  or 50% decrease in airflow from baseline or  $\geq 4\%$  ODI with  $\geq 50\%$  decrease in airflow. Indeed, hypopneas and apneas constitute the ventilatory instabilities measured in loop gain [176]. This highlights the importance of considering potential sources of variability when applying the regression model in the assessment of loop gain.

The remaining three OSA endotypic traits can also be assessed using conventional PMs or modified PM versions. For the determination of arousal threshold, the work of Edwards et al. serves as the foundation upon which PM III or IV can gauge low arousal thresholds based on two out of three scoring criteria [109]. These criteria involve a calculated AHI  $< 30$  events/hr, nadir  $SpO_2 > 82.5\%$ , and greater than 58.3% of respiratory events being hypopneas. To effectively determine arousal threshold, upper airway collapsibility and muscle responsiveness portably, the approach outlined by Sands et al. [175,178] can be employed, provided that EEG data are available to assess sleep state and arousal. In this case, arousal threshold is defined as the ventilatory drive at arousal. Passive ventilation serves as a  $P_{crit}$  surrogate, representing the ventilation at eupneic ventilatory drive. Upper airway muscle responsiveness can be effectively estimated by comparing the active ventilation (ventilation at ventilatory drive associated with the arousal threshold) with the passive ventilation. Both the sleep state and arousal requirements can be met by a portable EEG head band and patches adapted to a home sleep testing device [198,199]. These advancements in PM technology and methodologies offer promising opportunities for the portable assessment of crucial OSA traits in a more convenient and accessible manner.

Even without modifying PMs to include EEG, regular PMs can still be used to estimate arousal threshold, upper airway collapsibility and muscle responsiveness. This is possible using a single-lead ECG and a pulse oximeter, which have been demonstrated as portable means of detecting arousal and sleep stages from heart rate variability and PPG signal, respectively [[200], [201], [202]]. In this case, ECG or PPG-derived arousal can be utilized as an EEG arousal surrogate, and then the

four traits can be estimated using the Sands et al. approach [175,178]. While the single-lead ECG showed minimal agreement with the PSG method of arousal detection ( $\kappa=0.47$  and arousal/no arousal discriminative power of 0.59) [200], the PPG study achieved a much higher arousal discriminative power of 0.91 [201]. The difference in the performance of these two portable assessments of arousal may be attributed to the use of arousal probability threshold of 30% with the ECG method and 25% with the PPG approach.

Other non-AHI parameters, including HBI, CVHR, AHT and obstruction severity, obtained from PSG signals, provide valuable insights into various pathophysiological states in patients with OSA, such as cardiovascular diseases [162], [185], daytime sleepiness [190], and psychomotor alertness [191]. PM III, with its pulse oximetry and ECG channels [27], can be utilized to derive all these parameters. Interestingly, in PM IV, HBI, AHT and obstruction severity can be determined from the SpO<sub>2</sub> feature of pulse oximetry and airflow channels, while CVHR can be derived from the PPG component of pulse oximetry [159], [162], [190], [193]. However, further research is needed to thoroughly evaluate the performance of PMs III and IV in both home and laboratory settings for the measurement of these non-AHI metrics.

Regardless of the type, any PM can include a channel for detecting snores given that sound channel is not used in the classification of PMs [32], [42], [44]. Analyzing snoring signals can provide information about the sites of airway obstruction, aiding in determining appropriate treatment options and identifying contraindications. For example, invasive drug induced endoscopy has shown that hypoglossal nerve stimulation treatment may be contraindicated in OSA patients with concentric collapse of the retropalatal airway [203]. Interestingly, snoring signals have been shown to non-invasively predict obstruction sites with a sensitivity  $\geq 73\%$  at the levels of velum, oropharynx, tongue and epiglottis [204], with an accuracy of  $\sim 86\%$  [205].

Overall, PMs have the potential to streamline the diagnosis and management of OSA by providing accessible, unattended, and portable monitoring options. With further advancements and research, PMs can significantly contribute to early diagnosis of OSA, including the quantification of endotypic and other non-AHI related metrics, personalized treatment, and ultimately improved health outcomes for OSA patients.

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.


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## References

- [1] AASM  
Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research  
Sleep, 22 (5) (1999), pp. 667-689  
[Google Scholar ↗](#)
- [2] M. Younes  
Contributions of upper airway mechanics and control mechanisms to severity of obstructive apnea  
Am J Respir Crit Care Med, 168 (6) (2003), pp. 645-658  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [3] S.P. Patil, H. Schneider, J.J. Marx, E. Gladmon, A.R. Schwartz, P.L. Smith  
Neuromechanical control of upper airway patency during sleep  
J Appl Physiol, 102 (2) (1985), pp. 547-556  
2007  
[Google Scholar ↗](#)
- [4] Y. Li, N. Lin, J. Ye, Q. Chang, D. Han, A. Sperry  
Upper airway fat tissue distribution in subjects with obstructive sleep apnea and its effect on retropalatal mechanical loads  
Respir Care, 57 (7) (2012), pp. 1098-1105  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [5] T.D. Bradley, J.S. Floras  
Obstructive sleep apnoea and its cardiovascular consequences  
Lancet, 373 (9657) (2009), pp. 82-93  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [6] F.J. Nieto, T.B. Young, B.K. Lind, *et al.*  
Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study. Sleep Heart Health Study  
JAMA, 283 (14) (2000), pp. 1829-1836  
[Google Scholar ↗](#)

- [7] M. Partinen, C. Guilleminault  
Daytime sleepiness and vascular morbidity at seven-year follow-up in obstructive sleep apnea patients  
Chest, 97 (1) (1990), pp. 27-32  
 [View PDF](#) [View article](#) [Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [8] M.L. Jackson, M. Cavuoto, R. Schembri, *et al.*  
Severe obstructive sleep apnea is associated with higher brain amyloid burden: a preliminary pet imaging study  
J Alzheim Dis, 78 (2020), pp. 611-617  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [9] C.V. Senaratna, J.L. Perret, C.J. Lodge, *et al.*  
Prevalence of obstructive sleep apnea in the general population: a systematic review  
Sleep Med Rev, 34 (2017), pp. 70-81  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [10] G.S. Hamilton, R. Gupta, D. Vizcarra, G. Insalaco, F. Escobar, H. Kadotani  
Endorsement of: “clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American academy of sleep medicine clinical practice guideline” by the World Sleep Society  
Sleep Med, 79 (2021), pp. 152-154  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [11] J.V. Rundo, R. Downey 3rd  
Polysomnography  
Handb Clin Neurol, 160 (2019), pp. 381-392  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [12] N.A. Collop, W.M. Anderson, B. Boehlecke, *et al.*  
Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients  
J Clin Sleep Med, 3 (7) (2007), pp. 737-747  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [13] A.L. Chesson, R.B. Berry, A. Pack  
Practice parameters for the use of portable monitoring devices in the investigation of suspected obstructive sleep apnea in adults  
Sleep, 26 (7) (2003), pp. 907-913  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [14] M. Shokrollahi, S. Krishnan, D. Kumar, S. Arjunan  
**Chin EMG analysis for REM sleep behavior disorders**  
Paper presented at: 2012 ISSNIP biosignals and biorobotics conference: biosignals and robotics for better and safer living (BRC); 9-11 jan. 2012 (2012)  
[Google Scholar ↗](#)
- [15] H.A. Al-Angari  
**Evaluation of chin EMG activity at sleep onset and termination in obstructive sleep apnea syndrome**  
Paper presented at: 2008 computers in cardiology; 14-17 sept. 2008 (2008), [10.1109/CIC.2008.4749132 ↗](#)  
[Google Scholar ↗](#)
- [16] J. Virkkala, J. Toppila, P. Maasilta, A. Bachour  
**Electro-oculography-based detection of sleep-wake in sleep apnea patients**  
Sleep Breath, 19 (3) (2015), pp. 785-789  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [17] N.A. Collop, S.L. Tracy, V. Kapur, *et al.*  
**Obstructive sleep apnea devices for out-of-center (OOC) testing: technology evaluation**  
J Clin Sleep Med, 7 (5) (2011), pp. 531-548  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [18] C. Iber, S. Ancoli-Israel, A.L. Chesson, S. Quan  
**The AASM manual for the scoring of sleep and associated events: rules, terminology and technical specifications**  
American Academy of Sleep Medicine, Westchester, IL (2007)  
[Google Scholar ↗](#)
- [19] A. Zaffaroni, B. Kent, E. O'Hare, *et al.*  
**Assessment of sleep-disordered breathing using a non-contact bio-motion sensor**  
J Sleep Res, 22 (2) (2013), pp. 231-236  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [20] J.P. Kirkness, L.A. Peterson, S.B. Squier, *et al.*  
**Performance characteristics of upper airway critical collapsing pressure measurements during sleep**  
Sleep, 34 (4) (2011), pp. 459-467  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [21] A. Oksenberg, D.S. Silverberg, E. Arons, H. Radwan  
**Positional vs nonpositional obstructive sleep apnea patients: anthropomorphic, nocturnal polysomnographic, and multiple sleep latency test data**

Chest, 112 (3) (1997), pp. 629-639

 [View PDF](#) [View article](#) [Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[22] R.B. Berry, R. Budhiraja, D.J. Gottlieb, *et al.*

Rules for scoring respiratory events in sleep: update of the 2007 AASM manual for the scoring of sleep and associated events. Deliberations of the sleep apnea definitions task force of the American academy of sleep medicine

J Clin Sleep Med, 8 (5) (2012), pp. 597-619

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[23] M. Glos, A. Sabil, K.S. Jelavic, C. Schöbel, I. Fietze, T. Penzel

Characterization of respiratory events in obstructive sleep apnea using suprasternal pressure monitoring

J Clin Sleep Med, 14 (3) (2018), pp. 359-369

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[24] W. Randerath, J. Verbraecken, S. Andreas, *et al.*

Definition, discrimination, diagnosis and treatment of central breathing disturbances during sleep

Eur Respir J, 49 (1) (2017)

[Google Scholar ↗](#)

[25] C. Coronel, C. Wiesmeyr, H. Garn, *et al.*

3D camera and pulse oximeter for respiratory events detection

IEEE J Biomed Health Inform, 25 (1) (2021), pp. 181-188

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[26] N. Zavanelli, H. Kim, J. Kim, *et al.*

At-home wireless monitoring of acute hemodynamic disturbances to detect sleep apnea and sleep stages via a soft sternal patch

Sci Adv, 7 (52) (2021), Article eabl4146

[View in Scopus ↗](#) [Google Scholar ↗](#)

[27] V.K. Kapur, D.H. Auckley, S. Chowdhuri, *et al.*

Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American academy of sleep medicine clinical practice guideline

J Clin Sleep Med, 13 (3) (2017), pp. 479-504

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[28] C.A. Kushida, M.R. Littner, T. Morgenthaler, *et al.*

Practice parameters for the indications for polysomnography and related procedures: an update for 2005

Sleep, 28 (4) (2005), pp. 499-521

- [View in Scopus ↗](#) [Google Scholar ↗](#)
- [29] Home sleep apnea testing for obstructive sleep apnea in adults, UpToDate (2021)  
<https://www.uptodate.com/contents/home-sleep-apnea-testing-for-obstructive-sleep-apnea-in-adults> ↗, Accessed 20th Apr 2023  
[Google Scholar ↗](#)
- [30] M.T. Saletu, S.T. Kotzian, A. Schwarzinger, S. Haider, J. Spatt, B. Saletu  
Home sleep apnea testing is a feasible and accurate method to diagnose obstructive sleep apnea in stroke patients during in-hospital rehabilitation  
J Clin Sleep Med, 14 (9) (2018), pp. 1495-1501  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [31] D. Sommermeyer, D. Zou, L. Grote, J. Hedner  
Detection of sleep disordered breathing and its central/obstructive character using nasal cannula and finger pulse oximeter  
J Clin Sleep Med, 8 (5) (2012), pp. 527-533  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [32] P. Edouard, D. Campo, P. Bartet, *et al.*  
Validation of the Withings Sleep Analyzer, an under-the-mattress device for the detection of moderate-severe sleep apnea syndrome  
J Clin Sleep Med, 17 (6) (2021), pp. 1217-1227  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [33] Y. Chen, W. Wang, Y. Guo, H. Zhang, Y. Chen, L. Xie  
A single-center validation of the accuracy of a photoplethysmography-based smartwatch for screening obstructive sleep apnea  
Nat Sci Sleep, 13 (2021), pp. 1533-1544  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [34] L.M. Rofail, K.K. Wong, G. Unger, G.B. Marks, R.R. Grunstein  
The utility of single-channel nasal airflow pressure transducer in the diagnosis of OSA at home  
Sleep, 33 (8) (2010), pp. 1097-1105  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [35] Respiroics. Respiroics Alice 5 and user manual.  
<https://www.manualslib.com/manual/1296902/Respiroics-Alice-5.html?page=12#manual> ↗  
Accessed February 15, 2023..  
[Google Scholar ↗](#)
- [36] A. Sabil, M. Glos, A. Günther, *et al.*

## Comparison of apnea detection using oronasal thermal airflow sensor, nasal pressure transducer, respiratory inductance plethysmography and tracheal sound sensor

J Clin Sleep Med, 15 (2) (2019), pp. 285-292

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[37] S.O.M.N.O. Somnomedics

### HD eco

[https://somnomedics.de/solutions/sleep\\_diagnostics/stationary\\_sleep\\_lab\\_psg/somno-hd-eco-stationary-sleep-lab-psg/](https://somnomedics.de/solutions/sleep_diagnostics/stationary_sleep_lab_psg/somno-hd-eco-stationary-sleep-lab-psg/) ↗, Accessed 13th Mar 2023

[Google Scholar ↗](#)

[38] SleepStudy Wireless

### Polysomnography type 2 (PSG)

[https://www.sleepstudy.com/s\\_products\\_type\\_2\\_sleep\\_monitor.html](https://www.sleepstudy.com/s_products_type_2_sleep_monitor.html) ↗, Accessed 13th Mar 2023

[Google Scholar ↗](#)

[39] Natus. Embletta® MPR Sleep System. <https://natus.com/products-services/embletta-mpr-sleep-system> ↗. Accessed March 13, 2023..

[Google Scholar ↗](#)

[40] L. Xu, F. Han, B.T. Keenan, *et al.*

### Validation of the nox-T3 portable monitor for diagnosis of obstructive sleep apnea in Chinese adults

J Clin Sleep Med, 13 (5) (2017), pp. 675-683

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[41] R. Nunez, J. Rey de Castro, E. Socarras, *et al.*

### [Validation study of a polygraphic screening device (BREAS SC20) in the diagnosis of sleep apnea-hypopnea syndrome]

Arch Bronconeumol, 39 (12) (2003), pp. 537-543

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[42] R. Bhattacharjee, A. Benjafield, A. Blase, *et al.*

### The accuracy of a portable sleep monitor to diagnose obstructive sleep apnea in adolescent patients

J Clin Sleep Med, 17 (7) (2021), pp. 1379-1387

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[43] SleeView. Home Sleep Testing with SleepView Monitor + Web Portal.

<https://clevedmed.com/sleepview/> ↗. Accessed March 13, 2023..

[Google Scholar ↗](#)

- [44] Y. Yamada, S. Kasagi, H. Tamura, *et al.*  
Clinical utility of a type 4 portable device for in-home screening of sleep disordered breathing  
Ann Palliat Med, 9 (5) (2020), pp. 2895-2902  
[Crossref ↗](#) [Google Scholar ↗](#)
- [45] W. Gu, L. Leung, K.C. Kwok, I.C. Wu, R.J. Folz, A.A. Chiang  
Belun Ring Platform: a novel home sleep apnea testing system for assessment of obstructive sleep apnea  
J Clin Sleep Med, 16 (9) (2020), pp. 1611-1617  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [46] Belun\_Technology. Belun ring. <https://beluntech.com/belun-ring/> ↗. Accessed 13 March, 2023..  
[Google Scholar ↗](#)
- [47] G. Pillar, M. Berall, R. Berry, *et al.*  
Detecting central sleep apnea in adult patients using WatchPAT-a multicenter validation study  
Sleep Breath, 24 (1) (2020), pp. 387-398  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [48] A. Tanphaichitr, A. Thianboonsong, W. Banhiran, V. Vathanophas, K. Ungkanont  
Watch peripheral arterial tonometry in the diagnosis of pediatric obstructive sleep apnea  
Otolaryngol Head Neck Surg, 159 (1) (2018), pp. 166-172  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [49] N. Devani, R.X.A. Pramono, S.A. Imtiaz, S. Bowyer, E. Rodriguez-Villegas, S. Mandal  
Accuracy and usability of AcuPebble SA100 for automated diagnosis of obstructive sleep apnoea in the home environment setting: an evaluation study  
BMJ Open, 11 (12) (2021), Article e046803  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [50] Acurable. AcuPebble®SA100. <https://acurable.com/en/products/acupebble-SA100> ↗. Accessed March 13, 2023.  
[Google Scholar ↗](#)
- [51] R. Tiron, G. Lyon, H. Kilroy, *et al.*  
Screening for obstructive sleep apnea with novel hybrid acoustic smartphone app technology  
J Thorac Dis, 12 (8) (2020), pp. 4476-4495

- [Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [52] R. Nandakumar, S. Gollakota, N. Watson  
Contactless sleep apnea detection on smartphones  
GetMobile: Mobile Comput Commun, 19 (2015), pp. 22-24  
[Crossref ↗](#) [Google Scholar ↗](#)
- [53] Withins. Withins Sleep Analyzer. <https://www.withings.com/eu/en/sleep-analyzer> ↗.  
Accessed March 13, 2023..  
[Google Scholar ↗](#)
- [54] Huawei. [HUAWEI] measuring SpO2 on your watch  
<https://consumer.huawei.com/en/support/content/en-us00754833/> ↗, Accessed 13th Mar 2023  
[Google Scholar ↗](#)
- [55] B. Oktay, T.B. Rice, C.W. Atwood Jr., *et al.*  
Evaluation of a single-channel portable monitor for the diagnosis of obstructive sleep apnea  
J Clin Sleep Med, 7 (4) (2011), pp. 384-390  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [56] F. Barbieri, W. Dichtl, A. Heidbreder, *et al.*  
Sleep apnea detection by a cardiac resynchronization device integrated thoracic impedance sensor: a validation study against the gold standard polysomnography  
PLoS One, 13 (4) (2018), Article e0195573  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [57] Y.X. Xu, Q. Ou, Y.L. Cheng, M.C. Lao, G. Pei  
Comparative study of a wearable intelligent sleep monitor and polysomnography monitor for the diagnosis of obstructive sleep apnea  
Sleep Breath, 27 (1) (2022), pp. 205-212  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [58] G.C. Gutierrez-Tobal, D. Alvarez, A. Crespo, F. Del Campo, R. Hornero  
Evaluation of machine-learning approaches to estimate sleep apnea severity from at-home oximetry recordings  
IEEE J Biomed Health Inform, 23 (2) (2019), pp. 882-892  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [59] E. Yeh, E. Wong, C.W. Tsai, *et al.*  
Detection of obstructive sleep apnea using Belun Sleep Platform wearable with neural network-based algorithm and its combined use with STOP-Bang questionnaire

PLoS One, 16 (10) (2021), Article e0258040

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[60] J. Zou, L. Meng, Y. Liu, *et al.*

Evaluation of a 2-channel portable device and a predictive model to screen for obstructive sleep apnea in a laboratory environment

Respir Care, 60 (3) (2015), pp. 356-362

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[61] S. Kang, D.-K. Kim, Y. Lee, *et al.*

Non-contact diagnosis of obstructive sleep apnea using impulse-radio ultra-wideband radar

Sci Rep, 10 (1) (2020), p. 5261

[View in Scopus ↗](#) [Google Scholar ↗](#)

[62] C. Coronel, C. Wiesmeyr, H. Garn, *et al.*

Measurement of respiratory effort in sleep by 3D camera and respiratory inductance plethysmography

Somnologie, 23 (2) (2019), pp. 86-92

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[63] A. Zaffaroni, P. de Chazal, C. Heneghan, P. Boyle, P.R. Mppm, W.T. McNicholas

SleepMinder: an innovative contact-free device for the estimation of the apnoea-hypopnoea index

Annu Int Conf IEEE Eng Med Biol Soc, 2009 (2009), pp. 7091-7094

[View in Scopus ↗](#) [Google Scholar ↗](#)

[64] E. Rodriguez-Villegas, G. Chen, J. Radcliffe, J. Duncan

A pilot study of a wearable apnoea detection device

BMJ Open, 4 (10) (2014), Article e005299

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[65] N. Montazeri Ghahjaverestan, S. Saha, M. Kabir, B. Gavrilovic, K. Zhu, A. Yadollahi

Sleep apnea severity based on estimated tidal volume and snoring features from tracheal signals

J Sleep Res, 31 (2) (2022), Article e13490

[View in Scopus ↗](#) [Google Scholar ↗](#)

[66] M. Młyńczak, T.A. Valdez, W. Kukwa

Joint apnea and body position analysis for home sleep studies using a wireless audio and motion sensor

IEEE Access, 8 (2020), pp. 170579-170587

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [67] G.B. Papini, P. Fonseca, M.M. van Gilst, J.W.M. Bergmans, R. Vullings, S. Overeem  
Wearable monitoring of sleep-disordered breathing: estimation of the apnea-hypopnea index using wrist-worn reflective photoplethysmography  
Sci Rep, 10 (1) (2020), Article 13512  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [68] J.L. Pepin, C. Letesson, N.N. Le-Dong, *et al.*  
Assessment of mandibular movement monitoring with machine learning analysis for the diagnosis of obstructive sleep apnea  
JAMA Netw Open, 3 (1) (2020), Article e1919657  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [69] X. Zhao, X. Wang, T. Yang, *et al.*  
Classification of sleep apnea based on EEG sub-band signal characteristics  
Sci Rep, 11 (1) (2021), p. 5824  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [70] M. Jayawardhana, Pd Chazal  
Enhanced detection of sleep apnoea using heart-rate, respiration effort and oxygen saturation derived from a photoplethysmography sensor  
Paper presented at: 2017 39th annual international conference of the IEEE engineering in medicine and biology society (EMBC); 11-15 july 2017 (2017)  
[Google Scholar ↗](#)
- [71] Serrano Alarcón Á, N. Martínez Madrid, R. Seepold  
A minimum set of physiological parameters to diagnose obstructive sleep apnea syndrome using non-invasive portable monitors  
A Systematic Review. Life (Basel) (2021), [10.3390/life11111249;11\(11\) ↗](#)  
[Google Scholar ↗](#)
- [72] B. Gholami, M.H. Behboudi, A. Khadem, A. Shoeibi, J.M. Gorriz  
Sleep apnea diagnosis using complexity features of EEG signals  
(2022)  
Cham  
[Google Scholar ↗](#)
- [73] A.R. Spector, D. Loriaux, A.E. Farjat  
The clinical significance of apneas versus hypopneas: is there really a difference?  
Cureus, 11 (4) (2019), Article e4560  
[Google Scholar ↗](#)
- [74] M. Fudim, I. Shahid, S. Emani, *et al.*  
Evaluation and treatment of central sleep apnea in patients with heart failure

Curr Probl Cardiol, 47 (12) (2022), Article 101364

 [View PDF](#) [View article](#) [View in Scopus](#) [Google Scholar](#)

[75] S.J. Crinion, R. Tiron, G. Lyon, *et al.*

**Ambulatory detection of sleep apnea using a non-contact biomotion sensor**

Journal of Sleep Research, 29 (1) (2020)

[Google Scholar](#)

[76] S.W. Yo, S.A. Joosten, H. Wimalaswaran, *et al.*

**Body position during laboratory and home polysomnography compared to habitual sleeping position at home**

J Clin Sleep Med, 18 (9) (2022), pp. 2103-2111

[Crossref](#) [View in Scopus](#) [Google Scholar](#)

[77] B. Lechat, G. Naik, A. Reynolds, *et al.*

**Multinight prevalence, variability, and diagnostic misclassification of obstructive sleep apnea**

Am J Respir Crit Care Med, 205 (5) (2022), pp. 563-569

[Crossref](#) [View in Scopus](#) [Google Scholar](#)

[78] C. Sammut-Powell, C. Reynard, J. Allen, *et al.*

**Examining the effect of evaluation sample size on the sensitivity and specificity of COVID-19 diagnostic tests in practice: a simulation study**

Diagnostic and Prognostic Research, 6 (1) (2022), p. 12

[Google Scholar](#)

[79] S. Redline, H.L. Kirchner, S.F. Quan, D.J. Gottlieb, V. Kapur, A. Newman

**The effects of age, sex, ethnicity, and sleep-disordered breathing on sleep architecture**

Arch Intern Med, 164 (4) (2004), pp. 406-418

[View in Scopus](#) [Google Scholar](#)

[80] C. Nakayama, K. Fujiwara, Y. Sumi, M. Matsuo, M. Kano, H. Kadotani

**Obstructive sleep apnea screening by heart rate variability-based apnea/normal respiration discriminant model**

Physiol Meas, 40 (12) (2019), Article 125001

[Crossref](#) [View in Scopus](#) [Google Scholar](#)

[81] R. Mehra, K. Strohl

**Evaluation and monitoring of respiratory function**

Sleep Disorders Medicine (2009), pp. 188-197

 [View PDF](#) [View article](#) [Google Scholar](#)

[82] J.V. Rundo

## Polysomnography

Technique (Phila) (2014), pp. 930-935

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[83] E. Sivieri, K. Dysart, S. Abbasi

### Evaluation of pulmonary function in the neonate

<https://doi.org/10.1016/B978-0-323-35214-7.00074-3.754-765.e753> ↗ (2017)

[Google Scholar ↗](#)

[84] N. Montazeri Ghahjaverestan, M.M. Kabir, S. Saha, *et al.*

### Relative tidal volume and respiratory airflow estimation using tracheal sound and movement during sleep

J Sleep Res, 30 (4) (2021), Article e13279

[View in Scopus ↗](#) [Google Scholar ↗](#)

[85] C. Voscopoulos, D. Ladd, L. Campana, E. George

### Non-invasive respiratory volume monitoring to detect apnea in post-operative patients: case series

J Clin Med Res, 6 (3) (2014), pp. 209-214

[Google Scholar ↗](#)

[86] O. Linschmann, S. Leonhardt, A. Vehkaoja, C. Hoog Antink

### Estimation of the respiratory rate from ballistocardiograms using the Hilbert transform

Biomed Eng Online, 21 (1) (2022), p. 54

[View in Scopus ↗](#) [Google Scholar ↗](#)

[87] C. Qiu, M.R. Yuce

### A wearable bioimpedance chest patch for IoHT-connected respiration monitoring

Annu Int Conf IEEE Eng Med Biol Soc, 2021 (2021), pp. 6924-6927

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[88] I.Q. Chung, J.T. Yu, H. Wei-Chih

Automatic estimation of respiration rate from oronasal pressure using Ensemble empirical mode decomposition, Butterworth filter, and data fusion, vol. 14, IET Science, Measurement & Technology (2020)

[Google Scholar ↗](#)

[89] Y. Fang, Z. Jiang, H. Wang

### A novel sleep respiratory rate detection method for obstructive sleep apnea based on characteristic moment waveform

J Healthc Eng, 2018 (2018), Article 1902176

[View in Scopus ↗](#) [Google Scholar ↗](#)

- [90] V. Jeyhani, T. Vuorinen, M. Mäntysalo, A. Vehkaoja  
Comparison of simple algorithms for estimating respiration rate from electrical impedance pneumography signals in wearable devices  
Health Technol, 7 (2016)  
[Google Scholar ↗](#)
- [91] E. De la Hoya, J. Cota-Ruiz, R. Gonzalez-Landaeta  
Respiratory rate detection by a time-based measurement system  
Rev Mexic Ingen Biomed, 37 (2016), pp. 91-99  
[Google Scholar ↗](#)
- [92] M.I.T.C. Data, P.H. Charlton, M. Villarroel, F. Salguiero  
Waveform analysis to estimate respiratory rate  
Secondary analysis of electronic health records, Springer Copyright, Cham (CH) (2016), pp. 377-390  
The Author(s). 2016  
[Google Scholar ↗](#)
- [93] R.K. Raji, X. Miao, A. Wan, L. Niu, Y. Li, A. Boakye  
Knitted piezoresistive smart chest band and its application for respiration patterns assessment  
Journal of Engineered Fibers and Fabrics, 14 (2019), Article 1558925019868474  
[Google Scholar ↗](#)
- [94] M. Ascha, A. Bhattacharyya, J.A. Ramos, A.R. Tonelli  
Pulse oximetry and arterial oxygen saturation during cardiopulmonary exercise testing  
Med Sci Sports Exerc, 50 (10) (2018), pp. 1992-1997  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [95] R. Kirszenblat, P. Edouard  
Validation of the withings ScanWatch as a wrist-worn reflective pulse oximeter: prospective interventional clinical study  
J Med Internet Res, 23 (4) (2021), Article e27503  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [96] N. Rauniyar, S. Pujari, P. Shrestha  
Study of oxygen saturation by pulse oximetry and arterial blood gas in ICU patients: a descriptive cross-sectional study  
JNMA J Nepal Med Assoc, 58 (230) (2020), pp. 789-793  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [97] D.T. Kent, K.A. Carden, L. Wang, C.J. Lindsell, S.L. Ishman

Evaluation of hypoglossal nerve stimulation treatment in obstructive sleep apnea  
JAMA Otolaryngol Head Neck Surg, 145 (11) (2019), pp. 1044-1052

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [98] S. Nikkonen, I.O. Afara, T. Leppänen, J. Töyräs  
Artificial neural network analysis of the oxygen saturation signal enables accurate diagnostics of sleep apnea

Sci Rep, 9 (1) (2019), Article 13200

[View in Scopus ↗](#) [Google Scholar ↗](#)

- [99] Z. Huang, Y. Wu, K. Huang, P. Chen, J. Chen, L. Wang  
The nadir oxygen-specific heart rate response in sleep apnea links with the occurrence of acute myocardial infarction

Front Cardiovasc Med, 9 (2022), Article 807436

[View in Scopus ↗](#) [Google Scholar ↗](#)

- [100] K. Sutherland, N. Sadr, Y.S. Bin, *et al.*  
Comparative associations of oximetry patterns in Obstructive Sleep Apnea with incident cardiovascular disease

Sleep, 45 (12) (2022)

[Google Scholar ↗](#)

- [101] A.M. Beres  
Time is of the essence: a review of electroencephalography (EEG) and event-related brain potentials (ERPs) in language research

Appl Psychophysiol Biofeedback, 42 (4) (2017), pp. 247-255

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [102] D.J. Eckert, M.K. Younes  
Arousal from sleep: implications for obstructive sleep apnea pathogenesis and treatment

2014

J Appl Physiol, 116 (3) (1985), pp. 302-313

[Google Scholar ↗](#)

- [103] J. Amatoury, A. Azarbarzin, M. Younes, A.S. Jordan, A. Wellman, D.J. Eckert  
Arousal intensity is a distinct pathophysiological trait in obstructive sleep apnea

Sleep, 39 (12) (2016), pp. 2091-2100

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [104] A. Guillot, T. Moutakanni, M. Harris, P.J. Arnal, V. Thorey  
0616 validation of a sleep headband for detecting obstructive sleep apnea

Sleep (2020), p. 43

[Google Scholar ↗](#)

- [105] H. de Vries, A. Jonkman, Z.H. Shi, A. Spoelstra-de Man, L. Heunks  
Assessing breathing effort in mechanical ventilation: physiology and clinical implications  
Ann Transl Med, 6 (19) (2018), p. 387  
[Google Scholar ↗](#)
- [106] A. Aliverti  
The respiratory muscles during exercise  
Breathe, 12 (2) (2016), pp. 165-168  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [107] K.J. Maddison, K.L. Shepherd, V.A. Baker, *et al.*  
Effects on upper airway collapsibility of presence of a pharyngeal catheter  
J Sleep Res, 24 (1) (2015), pp. 92-99  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [108] E. Akoumianaki, S.M. Maggiore, F. Valenza, *et al.*  
The application of esophageal pressure measurement in patients with respiratory failure  
Am J Respir Crit Care Med, 189 (5) (2014), pp. 520-531  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [109] B.A. Edwards, D.J. Eckert, D.G. McSharry, *et al.*  
Clinical predictors of the respiratory arousal threshold in patients with obstructive sleep apnea  
Am J Respir Crit Care Med, 190 (11) (2014), pp. 1293-1300  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [110] H. Sumbul, A.H. Yuzer, K. Sekeroglu  
A novel portable real-time low-cost sleep apnea monitoring system based on the global system for mobile communications (GSM) network  
Med Biol Eng Comput, 60 (2) (2022), pp. 619-632  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [111] C. Khalil, S. Zarabi, K. Kirkham, *et al.*  
Validity of non-contact methods for diagnosis of Obstructive Sleep Apnea: a systematic review and meta-analysis  
J Clin Anesth, 87 (2023), Article 111087  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [112] C. Guilleminault, R. Stoohs, S. Duncan  
Snoring (I). Daytime sleepiness in regular heavy snorers

Chest, 99 (1) (1991), pp. 40-48

 [View PDF](#) [View article](#) [Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[113] D. Pevernagie, R.M. Aarts, M. De Meyer

### The acoustics of snoring

Sleep Med Rev, 14 (2) (2010), pp. 131-144

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[114] H.W. Duchna

### [Sleep-related breathing disorders--a second edition of the international classification of sleep disorders (ICSD-2) of the American academy of sleep medicine (AASM)]

Pneumologie, 60 (9) (2006), pp. 568-575

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[115] N. Ben-Israel, A. Tarasiuk, Y. Zigel

### Obstructive apnea hypopnea index estimation by analysis of nocturnal snoring signals in adults

Sleep, 35 (9) (2012), pp. 1299-1305C

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[116] H. Luo, L. Zhang, L. Zhou, X. Lin, Z. Zhang, M. Wang

### Design of real-time system based on machine learning for snoring and OSA detection

Paper presented at: ICASSP 2022 - 2022 IEEE international conference on acoustics, speech and signal processing (ICASSP); 23-27 may 2022 (2022)

[Google Scholar ↗](#)

[117] M.B. Norman, S.M. Pithers, A.Y. Teng, K.A. Waters, C.E. Sullivan

### Validation of the sonomat against PSG and quantitative measurement of partial upper airway obstruction in children with sleep-disordered breathing

Sleep, 40 (3) (2017)

[Google Scholar ↗](#)

[118] A. Amaddeo, M. Fernandez-Bolanos, J. Olmo Arroyo, S. Khirani, G. Baffet, B. Fauroux

### Validation of a suprasternal pressure sensor for sleep apnea classification in children

J Clin Sleep Med, 12 (12) (2016), pp. 1641-1647

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)


[119] R. Westreich, A. Gozlan-Talmor, S. Geva-Robinson, *et al.*

### The presence of snoring as well as its intensity is underreported by women

J Clin Sleep Med, 15 (3) (2019), pp. 471-476

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [120] J. Tong, L. Jugé, P.G. Burke, *et al.*  
Respiratory-related displacement of the trachea in obstructive sleep apnea  
J Appl Physiol, 127 (5) (1985), pp. 1307-1316  
2019  
[Google Scholar ↗](#)
- [121] S. Hayashi, M. Tamaoka, T. Tateishi, Y. Murota, I. Handa, Y. Miyazaki  
A new feature with the potential to detect the severity of obstructive sleep apnoea  
via snoring sound analysis  
Int J Environ Res Public Health, 17 (8) (2020)  
[Google Scholar ↗](#)
- [122] H. Nakano, K. Hirayama, Y. Sadamitsu, *et al.*  
Monitoring sound to quantify snoring and sleep apnea severity using a  
smartphone: proof of concept  
J Clin Sleep Med, 10 (1) (2014), pp. 73-78  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [123] S.G. Kim, S.W. Cho, J.W. Kim  
Definition of the snoring episode index based on the analyses of snoring  
parameters and the apnea hypopnea index  
Sci Rep, 12 (1) (2022), p. 6761  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [124] L.A. Lee, J.F. Yu, Y.L. Lo, *et al.*  
Energy types of snoring sounds in patients with obstructive sleep apnea  
syndrome: a preliminary observation  
PLoS One, 7 (12) (2012), Article e53481  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [125] C. Varon, J. Morales, J. Lázaro, *et al.*  
A comparative study of ECG-derived respiration in ambulatory monitoring using  
the single-lead ECG  
Sci Rep, 10 (1) (2020), p. 5704  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [126] S.K. Longmore, G.Y. Lui, G. Naik, P.P. Breen, B. Jalaludin, G.D. Gargiulo  
A comparison of reflective photoplethysmography for detection of heart rate, blood  
oxygen saturation, and respiration rate at various anatomical locations  
Sensors, 19 (8) (2019)  
[Google Scholar ↗](#)

- [127] J. Allen  
Photoplethysmography and its application in clinical physiological measurement  
Physiol Meas, 28 (3) (2007), pp. R1-R39  
[Google Scholar ↗](#)
- [128] J.-P. Barral, A. Croibier  
2 - circulatory physiology  
J.-P. Barral, A. Croibier (Eds.), Visceral vascular manipulations, Churchill Livingstone, Oxford (2011), pp. 27-45, [10.1016/B978-0-7020-4351-2.00002-8 ↗](#)  
 [View PDF](#) [View article](#) [Google Scholar ↗](#)
- [129] C.K. Sun, C.C. Liu, W.M. Liu, H.T. Wu, R.M. Huang, A.B. Liu  
Compatibility of pulse-pulse intervals with R-R intervals in assessing cardiac autonomic function and its relation to risks of atherosclerosis  
Ci Ji Yi Xue Za Zhi, 32 (1) (2020), pp. 41-46  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [130] F. Shaffer, J.P. Ginsberg  
An overview of heart rate variability metrics and norms  
Front Public Health, 5 (2017), p. 258  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [131] A. Alqaraawi, A. Alwosheel, A. Alasaad  
Heart rate variability estimation in photoplethysmography signals using Bayesian learning approach  
Healthc Technol Lett, 3 (2) (2016), pp. 136-142  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [132] Y.S. Hsu, T.Y. Chen, D. Wu, C.M. Lin, J.N. Juang, W.T. Liu  
Screening of obstructive sleep apnea in patients who snore using a patch-type device with electrocardiogram and 3-axis accelerometer  
J Clin Sleep Med, 16 (7) (2020), pp. 1149-1160  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [133] C.P. O'Donnell, L. Allan, P. Atkinson, A.R. Schwartz  
The effect of upper airway obstruction and arousal on peripheral arterial tonometry in obstructive sleep apnea  
Am J Respir Crit Care Med, 166 (7) (2002), pp. 965-971  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [134] R. Schnall, A. Shlitner, J. Sheffy, R. Kedar, P. Lavie  
Periodic, profound peripheral vasoconstriction—a new marker of obstructive sleep apnea

Sleep, 22 (1999), pp. 939-946

[View in Scopus](#) [Google Scholar](#)

[135] H. Qin, B.T. Keenan, D.R. Mazzotti, *et al.*

Heart rate variability during wakefulness as a marker of obstructive sleep apnea severity

Sleep, 44 (5) (2021)

[Google Scholar](#)

[136] D. Linz, M. Baumert, L. Desteghe, *et al.*

Nightly sleep apnea severity in patients with atrial fibrillation: potential applications of long-term sleep apnea monitoring

Ijc Heart Vasc, 24 (2019)

[Google Scholar](#)

[137] K.M. Chang

Portable obstructive sleep apnea screening system using overnight ECG and a PDA-based wireless transmission system

Telemed J e Health, 15 (4) (2009), pp. 353-361

[Crossref](#) [View in Scopus](#) [Google Scholar](#)

[138] J.B. Martinot, F. Senny, S. Denison, *et al.*

Mandibular movements identify respiratory effort in pediatric obstructive sleep apnea

J Clin Sleep Med, 11 (5) (2015), pp. 567-574

[Crossref](#) [View in Scopus](#) [Google Scholar](#)

[139] J.L. Pepin, N.N. Le-Dong, V. Cuthbert, *et al.*

Mandibular movements are a reliable noninvasive alternative to esophageal pressure for measuring respiratory effort in patients with sleep apnea syndrome

Nat Sci Sleep, 14 (2022), pp. 635-644

[Crossref](#) [View in Scopus](#) [Google Scholar](#)

[140] J.B. Martinot, N.N. Le-Dong, V. Cuthbert, *et al.*

Respiratory mandibular movement signals reliably identify obstructive hypopnea events during sleep


Front Neurol, 10 (2019), p. 828

[View in Scopus](#) [Google Scholar](#)


[141] I. Sadek, T.T.S. Heng, E. Seet, B. Abdulrazak

A new approach for detecting sleep apnea using a contactless bed sensor: comparison study

J Med Internet Res, 22 (9) (2020), Article e18297

- [Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [142] I. Sadek, J. Biswas, B. Abdulrazak  
Ballistocardiogram signal processing: a review  
Health Inf Sci Syst, 7 (1) (2019), p. 10  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [143] N. Hasanzadeh, M.M. Ahmadi, H. Mohammadzade  
Blood pressure estimation using photoplethysmogram signal and its morphological features  
IEEE Sensor J, 20 (8) (2020), pp. 4300-4310  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [144] E. Yuda, M. Shibata, Y. Ogata, *et al.*  
Pulse rate variability: a new biomarker, not a surrogate for heart rate variability  
J Physiol Anthropol, 39 (1) (2020), p. 21  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [145] A.H. Khandoker, C.K. Karmakar, M. Palaniswami  
Comparison of pulse rate variability with heart rate variability during obstructive sleep apnea  
Med Eng Phys, 33 (2) (2011), pp. 204-209  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [146] D. Chabot, M. Bayer, A. de Roos  
Instantaneous heart rates and other techniques introducing errors in the calculation of heart rate  
Can J Zool, 69 (1991), pp. 1117-1120  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [147] A. Azarbarzin, M. Ostrowski, P. Hanly, M. Younes  
Relationship between arousal intensity and heart rate response to arousal  
Sleep, 37 (4) (2014), pp. 645-653  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [148] S.D. Pittman, N.T. Ayas, M.M. MacDonald, A. Malhotra, R.B. Fogel, D.P. White  
Using a wrist-worn device based on peripheral arterial tonometry to diagnose obstructive sleep apnea: in-laboratory and ambulatory validation  
Sleep, 27 (5) (2004), pp. 923-933  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [149] R.P. Schnall, J. Sheffy, T. Penzel  
Peripheral arterial tonometry–PAT technology  
Sleep Med Rev (2022), [10.1016/j.smrv.2021.101566](https://doi.org/10.1016/j.smrv.2021.101566);61:101566 [↗](#)

[Google Scholar ↗](#)

- [150] T. Penzel, K. Kesper, T. Ploch, H.F. Becker, C. Vogelmeier  
Ambulatory recording of sleep apnea using peripheral arterial tonometry  
Conf Proc IEEE Eng Med Biol Soc, 2004 (2004), pp. 3856-3859  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [151] R.M. Kwasnicki, G.W.V. Cross, L. Geoghegan, *et al.*  
A lightweight sensing platform for monitoring sleep quality and posture: a simulated validation study  
Eur J Med Res, 23 (1) (2018), p. 28  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [152] A.H. Yüzer, H. Sümbül, K. Polat  
A novel wearable real-time sleep apnea detection system based on the acceleration sensor  
IRBM, 41 (1) (2020), pp. 39-47  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [153] W. Kukwa, T. Lis, J. Laba, R.B. Mitchell, M. Mlynczak  
Sleep position detection with a wireless audio-motion sensor-A validation study  
Diagnostics, 12 (5) (2022)  
[Google Scholar ↗](#)
- [154] C. Kalkbrenner, M. Eichenlaub, S. Rudiger, C. Kropf-Sanchen, W. Rottbauer, R. Brucher  
Apnea and heart rate detection from tracheal body sounds for the diagnosis of sleep-related breathing disorders  
Med Biol Eng Comput, 56 (4) (2018), pp. 671-681  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [155] S. Isono, A. Tanaka, T. Nishino  
Lateral position decreases collapsibility of the passive pharynx in patients with obstructive sleep apnea  
Anesthesiology, 97 (4) (2002), pp. 780-785  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [156] R. Cao, I. Azimi, F. Sarhaddi, *et al.*  
Accuracy assessment of oura ring nocturnal heart rate and heart rate variability in comparison with electrocardiography in time and frequency domains: comprehensive analysis  
J Med Internet Res, 24 (1) (2022), Article e27487  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [157] M. Glos, A. Sabil, K.S. Jelavic, *et al.*

- Tracheal sound analysis for detection of sleep disordered breathing  
Somnologie, 23 (2) (2019), pp. 80-85  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [158] J. Hayano, E. Watanabe, Y. Saito, *et al.*  
Screening for obstructive sleep apnea by cyclic variation of heart rate  
Circ Arrhythm Electrophysiol, 4 (1) (2011), pp. 64-72  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [159] J. Hayano, H. Yamamoto, I. Nonaka, *et al.*  
Quantitative detection of sleep apnea with wearable watch device  
PLoS One, 15 (11) (2020), Article e0237279  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [160] J. Rosa, A. Peres, L. Gasperin Junior, D. Martinez, V. Fontanella  
Diagnostic accuracy of oximetry for obstructive sleep apnea: a study on older adults in a home setting  
Clinics, 76 (2021), Article e3056  
 [View PDF](#) [View article](#) [Google Scholar ↗](#)
- [161] A. Kulkas, P. Tiihonen, K. Eskola, P. Julkunen, E. Mervaala, J. Toyras  
Novel parameters for evaluating severity of sleep disordered breathing and for supporting diagnosis of sleep apnea-hypopnea syndrome  
J Med Eng Technol, 37 (2) (2013), pp. 135-143  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [162] A. Azarbarzin, S.A. Sands, K.L. Stone, *et al.*  
The hypoxic burden of sleep apnoea predicts cardiovascular disease-related mortality: the Osteoporotic Fractures in Men Study and the Sleep Heart Health Study  
Eur Heart J, 40 (14) (2019), pp. 1149-1157  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [163] F. Chen, K. Chen, C. Zhang, *et al.*  
Evaluating the clinical value of the hypoxia burden index in patients with obstructive sleep apnea  
Postgrad Med, 130 (4) (2018), pp. 436-441  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [164] A. Wellman, A.S. Jordan, A. Malhotra, *et al.*  
Ventilatory control and airway anatomy in obstructive sleep apnea  
Am J Respir Crit Care Med, 170 (11) (2004), pp. 1225-1232  
[View in Scopus ↗](#) [Google Scholar ↗](#)

- [165] D.J. Eckert, D.P. White, A.S. Jordan, A. Malhotra, A. Wellman  
Defining phenotypic causes of obstructive sleep apnea. Identification of novel therapeutic targets  
Am J Respir Crit Care Med, 188 (8) (2013), pp. 996-1004  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [166] D.J. Eckert, R.L. Owens, G.B. Kehlmann, *et al.*  
Eszopiclone increases the respiratory arousal threshold and lowers the apnoea/hypopnoea index in obstructive sleep apnoea patients with a low arousal threshold  
Clin Sci (Lond), 120 (12) (2011), pp. 505-514  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [167] S.A. Sands, B.A. Edwards, P.I. Terrill, *et al.*  
Identifying obstructive sleep apnoea patients responsive to supplemental oxygen therapy  
Eur Respir J, 52 (3) (2018)  
[Google Scholar ↗](#)
- [168] B.A. Edwards, C. Andara, S. Landry, *et al.*  
Upper-airway collapsibility and loop gain predict the response to oral appliance therapy in patients with obstructive sleep apnea  
Am J Respir Crit Care Med, 194 (11) (2016), pp. 1413-1422  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [169] J.C. Carberry, J. Amatoury, D.J. Eckert, *et al.*  
Personalized Management Approach for OSA  
Chest, 153 (3) (2018 Mar), pp. 744-755, [10.1016/j.chest.2017.06.011 ↗](https://doi.org/10.1016/j.chest.2017.06.011)  
Epub 2017 Jun 16. PMID: 28629917  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [170] A. Wellman, D.J. Eckert, A.S. Jordan, *et al.*  
A method for measuring and modeling the physiological traits causing obstructive sleep apnea  
2011  
J Appl Physiol, 110 (6) (1985), pp. 1627-1637  
[Google Scholar ↗](#)
- [171] C.D. Nguyen, J. Amatoury, J.C. Carberry, D.J. Eckert  
An automated and reliable method for breath detection during variable mask pressures in awake and sleeping humans  
2011

PLoS One, 12 (6) (2017 Jun 13), Article e0179030, [10.1371/journal.pone.0179030](https://doi.org/10.1371/journal.pone.0179030) ↗

PMID: 28609480; PMCID: PMC5469467

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[172] A. Azarbarzin, S.A. Sands, L. Taranto-Montemurro, *et al.*

Estimation of pharyngeal collapsibility during sleep by peak inspiratory airflow

Sleep, 40 (1) (2017)

[Google Scholar](#) ↗

[173] A.M. Osman, B.K. Tong, S.A. Landry, *et al.*

An assessment of a simple clinical technique to estimate pharyngeal collapsibility in people with obstructive sleep apnea

Sleep, 43 (10) (2020)

[Google Scholar](#) ↗

[174] A.M. Osman, J.C. Carberry, P.G.R. Burke, B. Toson, R.R. Grunstein, D.J. Eckert

Upper airway collapsibility measured using a simple wakefulness test closely relates to the pharyngeal critical closing pressure during sleep in obstructive sleep apnea

Sleep, 42 (7) (2019)

[Google Scholar](#) ↗

[175] S.A. Sands, B.A. Edwards, P.I. Terrill, *et al.*

Phenotyping pharyngeal pathophysiology using polysomnography in patients with obstructive sleep apnea

Am J Respir Crit Care Med, 197 (9) (2018), pp. 1187-1197

[Crossref](#) ↗ [View in Scopus](#) ↗ [Google Scholar](#) ↗

[176] P.I. Terrill, B.A. Edwards, S. Nemati, *et al.*

Quantifying the ventilatory control contribution to sleep apnoea using polysomnography

Eur Respir J, 45 (2) (2015), pp. 408-418

[Crossref](#) ↗ [View in Scopus](#) ↗ [Google Scholar](#) ↗

[177] J. Amatory, A.S. Jordan, B. Toson, C. Nguyen, A. Wellman, D.J. Eckert

New insights into the timing and potential mechanisms of respiratory-induced cortical arousals in obstructive sleep apnea

Sleep, 41 (11) (2018), Article zsy160

[Google Scholar](#) ↗

[178] S.A. Sands, P.I. Terrill, B.A. Edwards, *et al.*

Quantifying the arousal threshold using polysomnography in obstructive sleep apnea

Sleep, 41 (1) (2018)

[Google Scholar ↗](#)

- [179] A. Wellman, A. Malhotra, A.S. Jordan, K.E. Stevenson, S. Gautam, D.P. White  
Effect of oxygen in obstructive sleep apnea: role of loop gain

Respir Physiol Neurobiol, 162 (2) (2008), pp. 144-151

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [180] B.A. Edwards, S.A. Sands, D.J. Eckert, *et al.*  
Acetazolamide improves loop gain but not the other physiological traits causing obstructive sleep apnoea

J Physiol, 590 (5) (2012), pp. 1199-1211

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [181] A.S. Jordan, D.P. White, Y.L. Lo, *et al.*  
Airway dilator muscle activity and lung volume during stable breathing in obstructive sleep apnea

Sleep, 32 (3) (2009), pp. 361-368

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [182] Y.L. Lo, A.S. Jordan, A. Malhotra, *et al.*  
Influence of wakefulness on pharyngeal airway muscle activity

Thorax, 62 (9) (2007), pp. 799-805

[Crossref ↗](#) [Google Scholar ↗](#)

- [183] A.S. Jordan, A. Wellman, R.C. Heinzer, *et al.*  
Mechanisms used to restore ventilation after partial upper airway collapse during sleep in humans

Thorax, 62 (10) (2007), p. 861

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [184] A.J. McWhorter, J.A. Rowley, D.W. Eisele, P.L. Smith, A.R. Schwartz  
The effect of tensor veli palatini stimulation on upper airway patency

Arch Otolaryngol Head Neck Surg, 125 (9) (1999), pp. 937-940

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

- [185] J. Hayano, F. Yasuma, E. Watanabe, *et al.*  
Blunted cyclic variation of heart rate predicts mortality risk in post-myocardial infarction, end-stage renal disease, and chronic heart failure patients

Europace, 19 (8) (2017), pp. 1392-1400

[View in Scopus ↗](#) [Google Scholar ↗](#)

- [186] S. Yatsu, T. Kasai, R. Naito, *et al.*

## Utility of cyclic variation of heart rate score as a screening tool for sleep-disordered breathing in patients with heart failure

J Clin Sleep Med, 17 (11) (2021), pp. 2187-2196

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[187] C.-Y. Tsai, Y.-S. Liu, A. Majumdar, *et al.*

## Association between cyclic variation in the heart rate index and biomarkers of neurodegenerative diseases in obstructive sleep apnea syndrome: a pilot study

J Clin Neurosci, 98 (2022), pp. 37-44

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[188] H. Basheer, S. Sharma, M. Patel

## Can we use the oxygen desaturation index alone to reliably diagnose obstructive sleep apnoea in obese patients?

Eur Respir J, 48 (suppl 60) (2016), Article PA2315

[Google Scholar ↗](#)

[189] C.M. Tsai, C.H. Kang, M.C. Su, *et al.*

## Usefulness of desaturation index for the assessment of obstructive sleep apnea syndrome in children

Int J Pediatr Otorhinolaryngol, 77 (8) (2013), pp. 1286-1290

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[190] S. Kainulainen, J. Toyras, A. Oksenberg, *et al.*

## Severity of desaturations reflects OSA-related daytime sleepiness better than AHI

J Clin Sleep Med, 15 (8) (2019), pp. 1135-1142

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[191] S. Kainulainen, B. Duce, H. Korkalainen, *et al.*

## Severe desaturations increase psychomotor vigilance task-based median reaction time and number of lapses in obstructive sleep apnoea patients

Eur Respir J, 55 (4) (2020)

[Google Scholar ↗](#)

[192] E. Bondia Real, J. Signes-Costa, J. Gavara, *et al.*

## Hypoxic burden and angiogenic factors in patients with myocardial infarction and obstructive sleep apnea


Eur Respir J, 54 (suppl 63) (2019), Article PA897

[Crossref ↗](#) [Google Scholar ↗](#)

[193] C. Ma, Y. Zhang, J. Liu, G. Sun

## A novel parameter is better than the AHI to assess nocturnal hypoxaemia and excessive daytime sleepiness in obstructive sleep apnoea

- Sci Rep, 11 (1) (2021), p. 4702  
[View in Scopus](#) [Google Scholar](#)
- [194] E.I. Huang, S.-Y. Huang, Y.-C. Lin, *et al.*  
Negative impact of the hypopnea index or duration increase after a non-frame work surgery in patients with very severe obstructive sleep apnea  
Sci Rep, 12 (1) (2022), p. 2251  
[View in Scopus](#) [Google Scholar](#)
- [195] D. Yumino, T.D. Bradley  
Central sleep apnea and Cheyne-Stokes respiration  
Proc Am Thorac Soc, 5 (2) (2008), pp. 226-236  
[Crossref](#) [View in Scopus](#) [Google Scholar](#)
- [196] J.E. Orr, S.A. Sands, B.A. Edwards, *et al.*  
Measuring loop gain via home sleep testing in patients with obstructive sleep apnea  
Am J Respir Crit Care Med, 197 (10) (2018), pp. 1353-1355  
[Crossref](#) [View in Scopus](#) [Google Scholar](#)
- [197] C.N. Schmickl, J.E. Orr, P. Kim, *et al.*  
Point-of-care prediction model of loop gain in patients with obstructive sleep apnea: development and validation  
BMC Pulm Med, 22 (1) (2022), p. 158  
[View in Scopus](#) [Google Scholar](#)
- [198] P.J. Arnal, V. Thorey, E. Debellemanni, *et al.*  
The Dreem Headband compared to polysomnography for electroencephalographic signal acquisition and sleep staging  
Sleep, 43 (11) (2020)  
[Google Scholar](#)
- [199] S. Kwon, H.S. Kim, K. Kwon, *et al.*  
At-home wireless sleep monitoring patches for the clinical assessment of sleep quality and sleep apnea  
Sci Adv, 9 (21) (2023), Article eadg9671  
[View in Scopus](#) [Google Scholar](#)
- [200] F. Ehrlich, J. Bender, H. Malberg, M. Goldammer  
Automatic sleep arousal detection using heart rate from a single-lead electrocardiogram  
Paper presented at: 2022 computing in cardiology (CinC); 4-7 sept. 2022 (2022)  
[Google Scholar](#)

- [201] C. Karmakar, A. Khandoker, T. Penzel, C. Schöbel, M. Palaniswami  
Detection of respiratory arousals using photoplethysmography (PPG) signal in sleep apnea patients  
IEEE J Biomed Health Inform, 18 (3) (2014), pp. 1065-1073  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [202] M. Radha, P. Fonseca, A. Moreau, *et al.*  
A deep transfer learning approach for wearable sleep stage classification with photoplethysmography  
NPJ Digit Med, 4 (1) (2021), p. 135  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [203] P.J. Strollo Jr., R.J. Soose, J.T. Maurer, *et al.*  
Upper-airway stimulation for obstructive sleep apnea  
N Engl J Med, 370 (2) (2014), pp. 139-149  
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [204] J. Sun, X. Hu, S. Peng, C.K. Peng, Y. Ma  
Automatic classification of excitation location of snoring sounds  
J Clin Sleep Med, 17 (5) (2021), pp. 1031-1038  
[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [205] Y. Liu, Y. Feng, Y. Li, W. Xu, X. Wang, D. Han  
Automatic classification of the obstruction site in obstructive sleep apnea based on snoring sounds  
Am J Otolaryngol, 43 (6) (2022), Article 103584  
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

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2025, Ieee Transactions on Electrical and Electronic Engineering

## [A Compact and Wireless Wearable System for Sleep Apnea Detection](#)

2025, IEEE Sensors Journal

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