

Research Article

Comparison of Demographical and Sleep Architecture between OSA Patients with Various Severities and Developing a Simple Prediction Model

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Abstract

Background: Due to changes in dietary habits and demographic structure, obstructive sleep apnea with obesity and aging as risk factors has become an important public health issue. The aim of this study is to investigate the relationships between demographics features as well as sleep characteristics of patients and severity of OSA and using easily available measurements to develop a simple model for rapidly identify OSA patients.

Methods: A retrospective review of the polysomnography records of patients who refer to the Sleep Center of Shuang-Ho Hospital, New Taipei City, Taiwan with a diagnosis of suspected sleep apnea between March 2015 and March 2019. Patients under 20 and over 80 years old were excluded. The demographic and anthropometric data of the patients were recorded, and comparing those parameters between patients with different severity of OSA.

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Results: According to AHI assessed by PSG, 6800 patients were divided into four groups as follows: AHI<5: 1223 common snoring (17.99%), 5≤AHI<15: 1341 mild OSA (19.72%), 15≤AHI<30: 1357 moderate OSA (19.96%) and AHI≥30: 2879 severe OSA (42.34%) patients. Bodyweight, waistline, and BMI, as well as heart rate, snoring index, and oxygen desaturation-induced arousal, were increased with OSA severity, while the proportion of females was decreased (all $p<0.001$). In sleep architecture, patients with more severe OSA showed extended duration of Wake after Sleep Onset (WASO), poor sleep efficiency, and also expanded N1 proportion and reduced N2, N3, and REM. The simple prediction model considered only three factors including weight, age, and oxygen saturation showed great accuracy in identifying OSA patients with the cut-off value AHI>5 (AUC 0.901). Predicted AHI value deduced by linear regression showed substantial correlation with PSG-assessed AHI (Pearson correlation coefficient, 0.73), and considerable ability in explanatory (Ratio variance = 53%).

Conclusion: This study revealed the demographic and clinical differences in patients with various OSA severities and using easily available measurements including weight, age, and mean of oxygen saturation during sleep, we can precisely discriminate OSA patients from the common snoring population.

Keywords: Demographical difference; Obstructive Sleep Apnea (OSA); Sleep architecture; Simple prediction model

Introduction

Because of the changes in dietary habits and demographic structure, patients with sleep disorders were substantially increased in recent years and becoming an important public health issue in modern society [1,2]. According to an epidemiological investigation, the prevalence of sleep disorder was estimated at 4-24% in males and 2-9% in females within American populations [3]. Among patients with sleep disorders, Obstructive Sleep Apnea (OSA) is a general type characterized as repeated episodes of apnea during sleep, with complete or incomplete upper airway obstruction that often causes blood oxygen desaturation and usually ends with brief awakenings from sleep [4]. In addition, one of the obvious symptoms of OSA patients was loud and irregular snoring during sleep [5]. Due to the difference in cranial structure, the prevalence of OSA within Asian populations was expected higher than Euro-Americans [6,7]. The prevalence of OSA showed in Asian studies was greatly varied, ranging from 3.2% to 27% [6-9].

Previous studies revealed the risk factors of OSA, among them, obesity was the main factor [10,11]. The prevalence of OSA was documented as high as 40% in male obese subjects and 81% in severely obese individuals [12,13]. The possible mechanism that obesity effect on OSA may be mediated by reduced ventilator stability result from airway surrounding adipose tissue deposition [10]. In clinical manifestation, sleep duration and quality were decreased associated with the increased body weight and adiposity [14,15]. On the other hand, in spite of the evidence was less direct,

OSA may also reciprocally contributing to obesity [16]. Aging was another significant risk factor for OSA [17]. Previous studies showed the prevalence of OSA and snoring were increased with age [18,19]. It probably due to the reduced pharyngeal muscle strength or shifted circadian rhythm [20,21]. A study found a significant reduction in the amount of slow-wave sleep and increases in stage 1 and 2 non-rapid eye movement sleep in the elderly [17]. Other potential risk factors including smoking and alcohol consumption, nasal congestion, as well as estrogen depletion in menopause, and ethnicity were also documented and discussed [22,23]. Besides, air pollution was also documented as a risk factor for OSA [24].

Growing evidence revealed the associations of OSA with high prevalence in mortality and comorbidity [1,25,26]. Adverse medical conditions associated with OSA including hypertension, stroke, congestive heart failure, coronary artery disease, cardiovascular mortality and neurocognitive dysfunction [23]. OSA reciprocal contributing to obesity through sympathetic activation, insulin resistance, elevated angiotensin II and aldosterone levels, oxidative and inflammatory stress, and perhaps by effects on renal function [27]. In addition, with the long-term intermittent hypoxia, OSA adversely affects multiple organs and systems, aggravates cardiovascular diseases, respiratory disease and metabolic dysfunction [28-31]. Therefore, the prevalence of OSA in those patients with critical illness was observed higher than normal, for example, the prevalence of sleep apnea among patients with essential hypertension is over 25% [32,33]. In clinical application, snoring and sleep apnea were taken as potential determinants of cardiovascular and cerebrovascular risk [32]. Moreover, OSA and companion nocturnal hypoxemia were also shown to increase the risk of diabetes and cancer, as well as stroke [34,35]. Not only influence physiological function, but OSA also influences psychological status and cognition impairment, previous studies documented that OSA elevates the risk of dementia [36,37], deteriorate depression [38], thus diminish the academic and work performance [39,40] and quality of life [22]. Sleepiness and fatigue caused by poor sleep quality may also increase the occurrence of car accidents [22,41].

OSA was of great importance in view of individual and public health and Polysomnography (PSG) was the golden standard for screening OSA for decades [42]. However, due to the increased awareness of OSA but the insufficient PSG inspection manpower and space, waiting times for hospital-based PSG monitoring are continuously rising [43-45]. There is an urgent need for a prediction model of OSA severity, which is helpful for OSA screening and treatment strategy formulation. In this study, we retrospectively review the PSG record of patients who were admitted to the Sleep Center of Shuang-Ho Hospital between March 2015 and March 2019. Not merely comparing demographic and clinical features between groups with various OSA severities, we plan to explore the risk factor of OSA and built a predictive model.

Materials and Methods

Database

The Institutional Review Board of Taipei Medical University approved analyses of our clinical sleep laboratory database without requiring additional consent. The sleep center of Shuang-Ho hospital uses the Remlogic and Noxturnal recording system and follows Taiwan Society of Sleep Medicine (TSSM) practice standards, including

regular scoring evaluation of technologists compared to the medical director. All scoring technologists are certified. Subjects inclusion criteria were: Age ≥ 20 years and < 80 years old, and underwent clinical Polysomnography (PSG) in our sleep center between March 2015 and March 2019, without regard to clinical reason.

Data processing and statistics

We retrospectively review the PSG records of 7131 individuals. Subjects without complete records and out of range in age of inclusion criteria were first excluded. A total of 6800 OSA patients were included in the final analyses. Based on the guidance of the American Academy of Sleep Medicine (AASM), patients were designated into one of the following four groups according to AHI assessed by PSG, $AHI < 5$: Common snorer, $5 \leq AHI < 15$: Mild OSA, $15 \leq AHI < 30$: Moderate OSA and $AHI \geq 30$: Severe OSA patients [46]. Comparisons of demographic features including height, weight, BMI, age, neck circumference and waistline are made between various severities, as well as the clinical characteristics obtain by PSG, including total sleep time, sleep efficiency, Wake after Sleep Onset (WASO) period, AHI, RDI, mean and minimum of oxygen saturation, desaturation index, heart rate, snoring and respiratory-related limb movement indexes. The distribution normality of each parameter was tested by the Shapiro-Wilks test, and then the Student's t-test or Mann-Whitney U test was employed according to the data type. Fisher exact test was applied to examine the difference in gender composition between every two populations. We also analyze the sleep architecture and the proportion of sleep posture. Because the sleep stage and posture were presented in proportion, the Kruskal-Wallis test was used for sleep architecture and posture analysis.

Construction and validation processes for prediction models

For distinguishing OSA patients from healthy ones, multivariate logistic regression analysis was used to predict the probability of having OSA from demographic and clinical variables with a step-wise selection procedure. With various cut-off values, we shift AHI into dichotomous data and randomized choice 50% of data of both categories, and then integrated as one dataset for initial model construction. Although the accuracy of the initial prediction model was stunning ($AUC=0.9497$), considering the clinical applicability, we try to develop a simple one. After applied Principal Component Analysis (PCA) for dimensionality reduction to explore the association between variables, a simple prediction model that included only three variables, including weight, age, and oxygen saturation was developed. The remaining data were used to validate both models. Sensitivity and specificity were calculated, as well as the ROC curve was plotted to evaluate the area under the curve as an index of accuracy. The probability threshold used for the classification of OSA was 0.50. Persons correlation test was used to identify variables that made an important contribution to the variability of AHI. Stepwise multiple linear regressions were used to yield an equation for predict AHI. And we also transfer both predict AHI and PSG-assessed AHI into nominal variables, Persons correlation test, and chi-square test was used to check the similarity of the two variables respectively.

All results were obtained using R software.

Results

Demographic features

Among 6800 patients included in this study, the distribution of patients with various severity was described as follow: Common snorer ($AHI < 5$): 1223 (17.99%), mild OSA ($5 \leq AHI < 15$): 1341 (19.72%), moderate OSA ($15 \leq AHI < 30$): 1357 (19.96%), and severe OSA ($AHI \geq 30$): 2879 (42.34%) patients according to the criteria issued by AASM. Body height, weight, BMI, neck circumference and waistline were significant increased with the increasing OSA severity revealed by the Kruskal-Wallis test as well as the modified Bonferroni post-hoc test (all $p < 0.001$). For example, the mean body weight was increased from 62.67 ± 11.83 kg in common snorer to 83.62 ± 16.63 kg in patients with severe OSA. Besides, the proportion of female subjects were 57.4%, 35.2%, 27.6%, and 15.4%, significantly decreased with the increasing OSA severity (Fisher exact test: $p < 0.001$ between every two groups). While common snorer was younger compare to those patients with OSA regardless of the severity (Table 1).

Clinical characteristics

The total sleep time was a bit longer in the patient with OSA comparing to the common snorer, and within the patient with OSA,

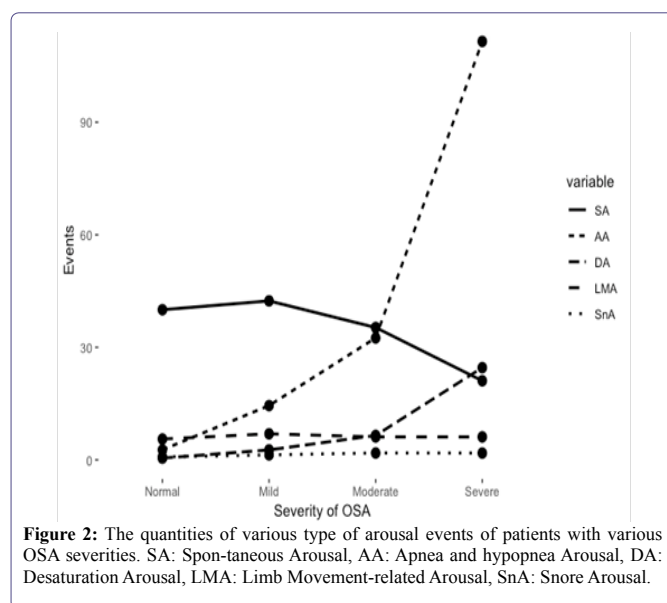
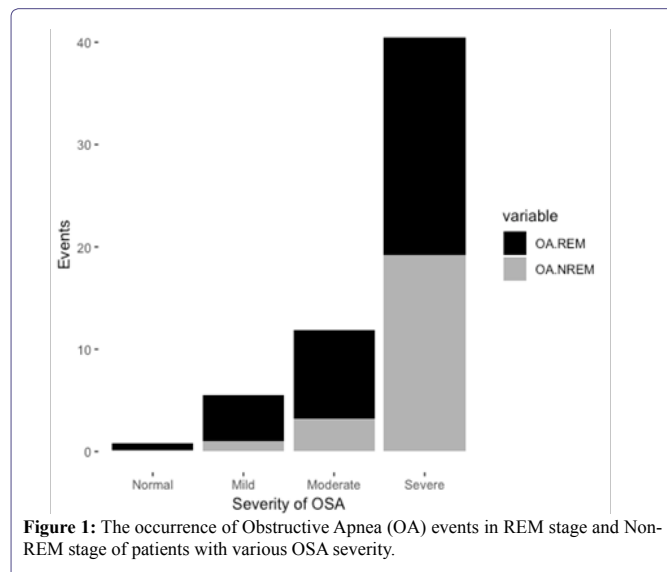
sleep efficiency was reduced and the duration of Wake After Sleep Onset (WASO) was elongated as severity rose (Table 2). Sleep architecture analysis revealed the proportion of N2 and N3 was reduced with growing severity; we also observed a significant reduction in the proportion of Rapid Eye Movement (REM) stage in severe OSA patients. Moreover, it mainly compensates by an elevated proportion of the N1 stage. The occurrence of Obstructive Apnea (OA) events in the REM stage and Non-REM stage were both increased as increasing severity, however, the composition ratio (OA event in REM/OA event in N-REM) shifted, it drops from 4.19 in common snorer to 1.11 in patients with severe OSA (Figure 1). The mean of oxygen saturation during sleep decreased with increasing severity and Oxygen Desaturation Index (ODI) which defined as the number of times per hour of sleep that the blood's oxygen level drop by criteria of at least 4% was highly correlated with AHI (Pearson correlation coefficient: 0.9907, $p < 0.001$). We also notice that the number of apnea, hypopnea as well as desaturation arousal was significantly expanded, while spontaneous arousal slightly declines (Figure 2). Other clinical manifestations including the mean of heart rate, snoring index and frequency of limb movement during sleep were also shown the growing trend as severity increased (Table 2).

	Normal	Mild	Moderate	Severe	p value
N	1223	1341	1357	2879	
Height (cm)	163.26 ± 8.87^a	165.73 ± 9.17^b	166.42 ± 8.87^c	168.14 ± 7.97^d	< 0.001
Weight (kg)	62.67 ± 11.83^a	69.64 ± 12.8^b	74.96 ± 14.08^c	83.62 ± 16.63^d	< 0.001
BMI	23.4 ± 3.32^a	25.33 ± 4.77^b	27.02 ± 4.44^c	29.49 ± 5.06^d	< 0.001
Age (yr)	44.64 ± 13.66^a	49.44 ± 13.53^b	50.61 ± 12.9^b	50.23 ± 12.65^b	< 0.001
Neck circumference (cm)	34.96 ± 6.35^a	37.16 ± 11.99^b	38.27 ± 5.42^c	40.85 ± 10.09^d	< 0.001
Waistline (cm)	79.91 ± 11.73^a	87.02 ± 11.34^b	92.51 ± 25.21^c	99.31 ± 12.36^d	< 0.001
Proportion of female	57.40%	35.20%	27.63%	15.39%	< 0.001

Table 1: Demographic features within groups with various OSA severities. The comparisons of height, weight, BMI, age, neck circumference and waistline were conducted by Kruskal-Wallis test and superscript indicate the statistic outcome of pairwise test with Bonferroni correction. Comparisons of proportion of female were conducted by Fisher exact test.

		Normal	Mild	Moderate	Severe	p value
	Sleep time (hr)	5.62 ± 0.6^a	5.7 ± 0.49^b	5.68 ± 0.5^b	5.7 ± 0.54^b	< 0.001
	Sleep efficiency	0.76 ± 0.18^a	0.78 ± 0.15^b	0.77 ± 0.16^c	0.75 ± 0.17^c	< 0.001
	WASO	58.28 ± 53.2^a	56.19 ± 45.77^b	61.66 ± 48.94^c	67.68 ± 50.68^d	< 0.001
Sleep stage	REM	12.9 ± 7.3^a	13.3 ± 6.87^a	13.06 ± 7.23^a	11.06 ± 7.12^b	< 0.001
	N1	9.88 ± 8.08^a	11.59 ± 9.11^b	13.85 ± 10.51^c	20.22 ± 15.03^d	< 0.001
	N2	71.54 ± 12.4^a	70.72 ± 11.58^a	69.47 ± 12.19^b	66.61 ± 15.21^c	< 0.001
	N3	5.67 ± 8.9^a	4.39 ± 7.41^b	3.62 ± 6.92^c	2.11 ± 4.98^d	< 0.001
Apnea events	Apnea in REM	0.67 ± 2.55^a	4.49 ± 8.77^b	8.66 ± 13.02^c	21.31 ± 23.3^d	< 0.001
	Apnea in NREM	0.16 ± 0.38^a	1.02 ± 1.71^a	3.25 ± 4.6^b	19.18 ± 21.8^c	< 0.001
Oxygen saturations	SaO2	97.25 ± 1.1^a	96.41 ± 1.28^b	95.77 ± 1.47^c	94.13 ± 2.75^d	< 0.001
	Oxygen Desaturation	1.77 ± 1.56^a	9.13 ± 3.43^b	20.82 ± 5.47^c	56.74 ± 21.21^d	< 0.001
	Desaturation Arousal	0.1 ± 0.21^a	0.57 ± 0.55^b	1.41 ± 1.08^c	5.4 ± 4.81^d	< 0.001
Sleep posture	Ratio of supine	32.19 ± 16.28^a	32.7 ± 16.37^a	32.42 ± 16.13^a	33.51 ± 15.65^a	0.075
Other parameters	Heart rate	64.3 ± 8.63^a	65.12 ± 9.05^b	65.99 ± 8.74^c	69.25 ± 9.88^d	< 0.001
	Snoring index	100.6 ± 157.08^a	196.56 ± 208.83^b	261.58 ± 226.56^c	288.57 ± 203.19^d	< 0.001
	RRLM	0.13 ± 0.25^a	0.79 ± 0.9^b	1.79 ± 1.87^c	6.44 ± 7.35^d	< 0.001

Table 2: Clinical characteristics of patients with various OSA severities. WASO: Wake after Sleep Onset, REM: Rapid Eye Movement stage, N1: Non-REM stage 1, N2: Non-REM stage 2, N3: Non-REM stage 3, RRLM: Respiratory-Related Limb Movements.



Logistic prediction models for discriminating OSA from common snorer

Nine variables including Body height, weight, age, gender, mean heart rate and oxygen saturation during sleep, snoring index, the proportion of supine posture, and total sleep time were significant for our prediction model with cut-off values of $AHI < 5$. The accuracy presented by the area under ROC (Receiver Operating Characteristic) curve was 0.95; indicate the great ability in discriminating OSA patients from the common snorer. In addition, all nine variables were also significant for models with other cut-off values ($AHI < 15$ and $AHI < 30$). The accuracy of these two models was 0.92 and 0.89 respectively; imply these variables can also apply properly in discriminating patients of various severities. Considering the convenience of data acquisition and future clinical applicability, we explore the interrelationship of variables in a dimensionality

reduction manner, Principal Component Analysis (PCA) revealed that component 1 was mainly associated with weight, with the similar effect of neck circumference, waistline, snoring index and mean heart rate during sleep and reverse effect of mean oxygen saturation during sleep. While component 2 was more related to the age as well as sleep characteristics and architecture. After several tests, we develop a simple model considering only three variables, which were weight, age and mean oxygen saturation during sleep with the area under the ROC curve of 0.90.

Linear model for predict AHI

We found a direct relationship between AHI and the 14 variables that entered into the simple linear regression model as independent variables. By stepwise selection procedure, all variables except total sleep time were included equation and showed the adjusted R-square of 0.53. The correlation test showed a strong association between predict value and PSG-assessed AHI (Pearson correlation coefficient: 0.7275, $p < 0.001$). We shift both predicted value and PSG-assessed AHI into ordinal variables for evaluating the prediction accuracy. The contingency table presented that our linear model is more accurate in predicting severities in moderate and severe patients, with the accuracy of 0.80 and 0.79, respectively (Table 3).

	$AHI < 5$	$5 < AHI < 15$	$15 < AHI < 30$	$AHI > 30$
Predict < 5	5	0.96	0.25	0.06
$5 < \text{Predict} < 15$	6.67	4.15	1.96	0.56
$15 < \text{Predict} < 30$	5.24	10.64	8.86	8.15
Predict > 30	1.07	3.96	8.9	33.57

Table 3: Proportion of corresponding categories of two ordinal variables (Model predict value and PSG-assessed AHI).

Discussion

Demographical features of subjects were changed as the severity of OSA increased. In agree with previous studies, our study suggested that obesity was the main risk factor for OSA [11,47]. Four indexes, including weight, BMI, neck circumference, and waistline which were all proposed related to obesity, were increased with the severity of OSA. Among these four indicators, BMI was the most commonly used in describing obesity and has been concluded correlated with AHI [48-50]. In a pediatric study, morbidly obese children showed a decrease in sleep efficiency and percentage of time in Rapid Eye Movement (REM) [51]. Because compressed upper airway due to the adipose deposition was considered as one main factor contributing to OSA, neck circumference and its derivatives were used as a surrogate measure of upper airway obesity [52,53]. Neck to Height Ratio (NHR) was another index used to predict AHI for diminishing the gender effect [53]. A recent study discovered Neck-to-Abdominal Fat percentage (NAF %) also associated with OSA severity [54]. However, an investigation showed waistline was more powerful in predict OSA severity [55]. Besides, the association between obesity and OSA can be illustrated from the other aspect. The effect of obesity on OSA was reversible; weight management has been evaluated as a treatment for OSA [47]. Several studies showed the weight loss benefit in improving OSA, as well as reduce daytime sleepiness, as well as metabolic syndrome, and quality of life improvements [56,57]. Unlike the relationship between obesity and OSA, the effect of age in

OSA was ambiguous; the prevalence of OSA was increased while severity was reduced [18,19,49,58]. We suspect that it may due to the combination of conflict effect brought by aging; previous studies showed elder people has reduced upper airway muscle strength but they are less obese [49,59,60].

The clinical characteristics advance trend with OSA severity could be discussed from the following three dimensions: Frequent awake during sleep and its cause, shift in sleep architecture and occurrence timing of apnea, and other related physiological distortions. First, in OSA patients, sleep efficiency significantly decreased with increasing OSA severity, while Wake after Sleep Onset (WASO) and the number of arousal events were increased. Furthermore, the number of oxygen desaturation arousal was increased with growing severity besides apnea and hypopnea arousal events. Intermittent hypoxia was one of the remarkable characteristics of OSA patients [61]. Our data revealed that hypoxemia was not only increased in the occurrence frequency but also the duration. Oxygen desaturation index has shown its high degree of correlation with AHI and presented high sensitivity and specificity in the diagnosis of OSA [62,63]. A recent study showed the correlation of sleepiness evaluated by Epworth Sleepiness Scale (ESS) with Oxygen De-saturation Index (ODI) was stronger than that with AHI, therefore authors suggested that ODI is as valuable as AHI in diagnosing and grading the OSA [64]. ODI is also associated with the degree of obesity [65]. Moreover, one study showed the association between obesity and sleep duration in older adults [15]. Although the regulatory mechanisms between obesity, oxygen desaturation, and awake still waited for exploration, the evidence clearly showed the associations.

Second, we observed a significant change in sleep architecture between subjects with various OSA severities. As severity increased, the proportion of N2, N3, as well as REM stage, were reduced (REM only significant in severe OSA patients), but compensated with elongated N1 stage. It was agreed with the study focusing on comparing sleep architecture between primary snorer, OSA and normal populations [66], as well as the pediatric study [67,68]. Other studies were using the Markov chain model and transition matrix to demonstrate the alternation in sleep stage transition dynamics, they showed less stability of sleep stage transition in OSA patients [69,70]. The less stability implies more frequent sleep-wake transitions, also known as sleep fragmentation which plays a major role in the pathogenesis of most of the consequences of OSA (i.e. Neuropsychiatric, respiratory and cardiovascular) and may contribute to the progression of OSA severity [71]. Excessive fatigue or sleepiness, waking up tired, falling asleep during the day, trouble in paying attention, snoring and insomnia were confirmed related to decreased N3 sleep [72]. Another key role of sleep was memory consolidation, the decreased N2 and N3, as well as REM stage, may harm spatial memory [73]. Besides, this is the first study that observed a shift in the ratio of REM/Non-REM apnea events. The occurrence timing of apnea events was generally considered mainly in REM, in which the strength of upper airway muscle was lowest compared to other sleep stages [74]. However, the magnification in the occurrence of apnea in Non-REM was outweighing to the increased Non-REM duration and proportion, made the ratio of the probability of apnea occurrence between in REM and Non-REM was nearly equal in severe OSA patients, while the ratio was larger than 4 times in common snorer and mild OSA patients. It could result from both obesity-related neurological and physical distortion. Investigations

about the effect of weight loss showed a decreased stage 1 Non-REM as well as elevated sleep efficiency [75,76]. Thus, weight loss was one of the recommended treatments for OSA patients, and effective in improve sleep quality as well as benefit to reduce the risk of several critical illnesses [47,57].

Finally, physiological distortion also including snoring index and heart-related illness. Consistent with another study, the snoring index was increased with OSA severity [77]. Individuals with OSA or overweight were presented a narrow airway than the normal population, thus more likely to snore during sleep [78,79]. Obesity is also associated with increased upper airway collapsibility and reduced upper airway muscle protective force [80], that contributing on snore. However, data showed that one-third in male and one-half in female moderate to severe OSA patients were considered snore-free, those patients often ignored by the physician (unpublished data). On the other hand, the Heart Rate (HR) pattern was also influenced by OSA in both short and long terms. In short term, cumulative carbon dioxide speed the HR when an apnea event occurred, and in long term, intermittent hypoxia activates the sympathetic nervous system, thus accelerate the HR and expanded Heart Rate Variability (HRV) [81,82]. HRV was showed associated with severity in pediatric OSA patients [83].

Due to the awareness of sleep disorders has increased but the amplification in the number of beds providing for sleep inspection was not synchronized, waiting times for hospital-based PSG monitoring are successively increased [43-45]. In practice, a prolonged waiting time of hospital-based PSG can delay treatment and deter patients from complying with the sleep study and subsequent treatment [84]. It is obvious of the urgent needs in portable sleep screening devices and simple prediction model, which can save time and cost in individual, hospital and department of insurance than traditional hospital-based PSG inspection [85]. It also provides objective references to medical care personnel for prioritizing the order in which patients with suspected OSA were referred for PSG. By means of the development and certification of the portable device during the decades, the mean oxygen saturation could be measured by Photoplethysmography (PPG) as well as the posture during and limb movement scenario could be detected by the three-dimensional accelerometer, both were relatively easily available comparing to Electroencephalography (EEG), Electrooculogram (EOG) and other sleep-related parameters [86,87]. In 2007, AASM also agree to diagnosis by certificated type 3 portable sleep monitoring devices [88]. Generally believed that the home sleep test was closer to the real scenario. Through patch-free, wireless portable sleep monitoring devices, our team has demonstrated the overestimation of hospital-based PSG (article in press).

Respond to the needs, the development of OSA prediction models was growing vigorously since the last century. Australia and USA researchers proposed that craniofacial photographic analysis provides detailed anatomical data useful in the prediction of OSA [89,90]. One research team in the USA using physiological traits and showed good sensitivity and specificity for predicting OSA [91]. Another USA research team indicated that age, sex, BMI, and medical history are superior to the symptom variables [92]. Mexico re-searchers evaluated the diagnostic value of the morphometric model and the Sleep Apnea Clinical Score [93] and United Kingdom scholars assessed the contribution of the STOP-BANG questionnaire [94]. In recent years, researchers focusing on integrated multidisciplinary

variables, Korean team using clinical, anthropometric and cephalometric variables to predicting OSA [95], while Iran researchers using anthropometric and Mallampati indices [96] and Hong-Kong researchers using McGill scores together with overnight oximetry [97]. Here, we developed two models, one with 9 variables including clinical and anthropometric measurements, showed great accuracy and another one, surprisingly, simply consider only three variable including weight, age, and oxygen saturation which are available easily by the wearable device and showed high discrimination ability in identifying OSA patient. However, Singapore researchers found that the prediction model for OSA derived from a foreign population exhibits markedly different diagnostic characteristics from one that is developed locally, thus they underlined the importance of local clinical prediction models [98]. USA research team comparing four prediction models which developed in different cities but all using only clinical variables, the accuracy was varied and not sufficient to discriminate between patients with or without OSA [99]. Thus, the next step will be targeting to evaluate the accuracy between different clinical environments, for example, cross-departments or hospitals. Not only discriminating OSA patients from common snorer, models also predict the OSA severity. A research team in Finland predicts AHI by nasal resistance and change in mandibular position on lying down besides BMI [100]. Spanish researchers using anthropometric, clinical, and epidemiological parameters predict AHI, and the ratio variance in the number of respiratory events explained by the model was 33% [101]. Our linear model presented higher ability in explanation and categorization, especially for moderate and severe OSA patients.

Conclusion

In view of the association of many critical illnesses and risk of cognition impairment, OSA is becoming a global public health concern. We here retrospective reviewed the PSG records over the past 4 years of sleep center in Shuang-Ho hospital (New Taipei City) and found the severity of OSA was mainly associated with the degree of obesity. Clinical characteristics including sleep efficiency, sleep architecture, oxygen saturation and heart rate were all fluctuated with a tendency following by the increase of OSA severities. Considering the reality and convenience of data measurement, and future clinical applicability, we develop a simple prediction model that considered merely three easy available variables. It showed high discriminative ability and could apply in the extensive clinical field in saving medical cost and time, programming precisely treatment strategy in time as well as contributing to prioritizing the suspected OSA patients to refer for PSG.

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Footnote

We present the foregoing article in accordance with the STROBE reporting checklist. The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Data Availability Statement

The datasets generated during the current study are not publicly available due to involving the privacy of research participants but are available from the corresponding author on reasonable request.

Ethical Statement

The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Joint Institutional Review Board of Taipei Medical University (TMU-JIRB: N201911007) and individual consent for this retrospective analysis was waived.

Clinical Trial Registration Number

The Joint Institutional Review Board of Taipei Medical University approved analyses of clinical sleep laboratory database without requiring additional consent (TMU-JIRB: N201911007).

Author's Contribution

Wen-Te Liu conceived of the presented idea. Cheng-Yu Tsai and Shang-Yang Lin developed the theory and performed the computations. All authors discussed the results and contributed to the final manuscript. All authors certify that this material or similar material has not been submitted to or published in any other publication yet.

Conception and design: Wen-Te Liu

Administrative support: Shang-Yang Lin

Provision of study materials or patients: Wen-Te Liu, Shang-Yang Lin

Collection and assembly of data: All authors

Data analysis and interpretation: Shang-Yang Lin, Cheng-Yu Tsai

Manuscript writing: All authors

Final approval of manuscript: All authors

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Conflicts of Interest

All authors declare that there is no conflict of interest regarding the publication of this article.

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