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SCIENTIFIC INVESTIGATIONS

Correlation Between Oxygen Saturation and Pulse Tracing Patterns on Overnight Oximetry With Normal Desaturation Index Is an Independent Predictor of Obstructive Sleep Apnea

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Study Objectives: Overnight pulse oximetry (OPO) is commonly used as a screening test for obstructive sleep apnea. Heart rate variability (HRV) correlates well with apnea-hypopnea index during polysomnography (PSG). We hypothesized that visual correlation of episodic increase in HRV with minimal oxygen desaturations on normal OPO (oxygen desaturation index less than 5 events/h) is predictive of OSA.

Methods: A retrospective analysis of patients undergoing OPO and PSG in 1 year was performed. We included only OPO performed on room air and interpreted as normal. Visual correlation between simultaneous increase in HRV and minimal oxygen desaturation was independently assessed by three raters, resulting in the consensus agreement. The primary outcome was presence of OSA on the subsequent PSG.

Results: Of 936 patients with OPO and PSG, 109 patients had normal overnight oximetry study on room air. Of these, 65 (60%) were females, median (interquartile range) age was 54 years (44, 67), body mass index was 29 kg/m^2 (25, 32), and the median oxygen desaturation index was 1.8 events/h (1, 2.7). Consensus agreement identified 54 patients with visual correlation between pulse and minimal oxygen desaturations. Thirty-two patients (29%) were found to have OSA on PSG, of which 24 (75%) could have been accurately predicted by the consensus agreement (odds ratio 4.70, 95% confidence interval 1.87–11.8, $P < .001$). When adjusted for pertinent clinical and demographic variables, consensus agreement was independently associated with diagnosis of OSA on subsequent PSG (odds ratio 5.6, 95% confidence interval 1.76–20.9, $P = .003$).

Conclusions: Visual correlation between episodic increase in HRV and minimal oxygen desaturations on OPO is an independent predictor of OSA, and promising marker for clinical use.

Keywords: pulse oximetry, heart rate variability, oxygen desaturation index, obstructive sleep apnea

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BRIEF SUMMARY

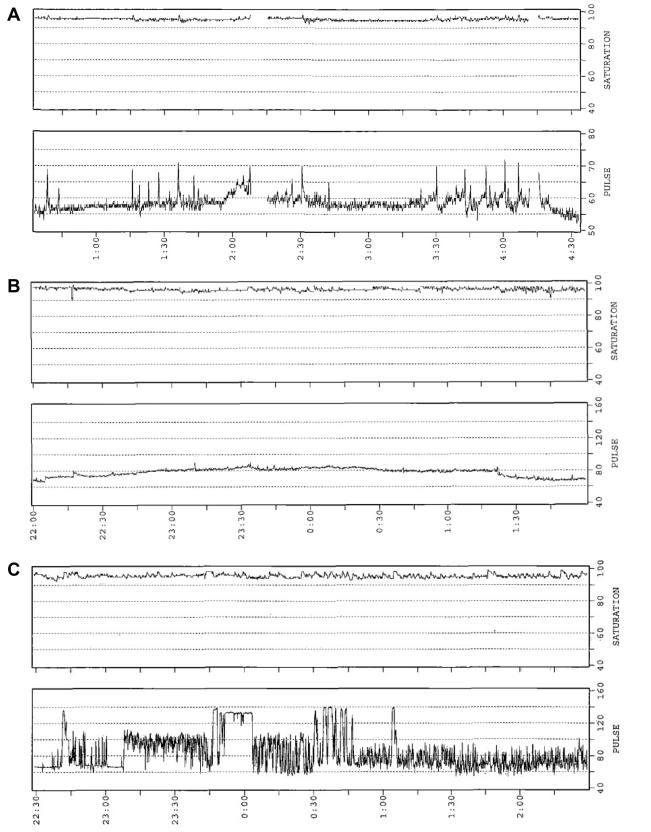
Current Knowledge/Study Rationale: The correlation of heart rate variability with sleep-disordered breathing during polysomnography has been well established. In order to assess whether this established finding could be translated to clinical practice, we studied visual correlation of heart rate variability with pulse oximetry in a cohort of patients with normal overnight pulse oximetry studies.

Study Impact: The patients with episodic heart rate variability occurring simultaneously with minimal oxygen desaturations despite normal oximetry results have five times higher odds of receiving a diagnosis of obstructive sleep apnea on a subsequent polysomnogram, when adjusted for pertinent demographic and clinical characteristics. Assessment of pulse tracing on overnight oximetry offers an additional predictive utility when screening for obstructive sleep apnea with overnight pulse oximetry.

INTRODUCTION

Although polysomnography (PSG) is the gold standard for diagnosis of obstructive sleep apnea (OSA),[1] overnight pulse oximetry (OPO) is commonly used as a screening test.[2] This test is widely available, inexpensive, and simple enough to be used by the patients with minimal instructions,[3] although not without limitations.[4,5] Based on the cutoff for "significant" oxygen desaturations (most commonly 4% or 3%), oxygen desaturation index (ODI) results can be used in clinical decision making. However, there is no universal agreement as to what level of ODI is considered abnormal.[3] In addition to the numeric report of the measured parameters, OPO provides a graphic display of the continuous tracings of oxygen saturation and heart rate. Therefore, both numeric report and the visual pattern are readily available to the interpreting physician when screening for sleep-disordered breathing (SDB). Although the visual pattern of oximetry may be very helpful in ruling in suspected SDB, in the case of flat tracing it may also suggest that the patient did not have continuous sleep, thus leading to falsely low numeric ODI.[6] On the contrary, visual pattern of the pulse tracing is largely underutilized in routine clinical interpretation of OPO,[4] although a solid body of experimental evidence confirms that heart rate variability (HRV) correlates well with SDB as measured by apnea-hypopnea index (AHI) during PSG.[7,8] As the pulse tracing is routinely displayed

Figure 1— Example of positive and negative visual correlations between oximetry tracing and heart rate variability and uninterpretable study due to atrial fibrillation.



(A) Positive visual correlation: 69-year old man with ODI of 1.1 events/h and subsequent AHI of 26.5 events/h. **(B)** Negative visual correlation: 58-year-old woman with ODI of 3.3 events/h and subsequent AHI of 8.0 events/h. **(C)** Uninterpretable study: 56-year-old man with atrial fibrillation, ODI of 1.5 events/h and subsequent AHI of 3.2 events/h. AHI = apnea-hypopnea index, ODI = oxygen desaturation index.

directly below the oxygen tracing, simultaneous perturbations in both tracings may provide additionally useful information relative to visual interpretation of oximetry graph alone. Therefore, we hypothesized that the visual correlation of minor oxygen tracing perturbations (desaturations) occurring simultaneously with episodic increases in HRV on OPO reported as normal (ODI < 5 events/h) would be predictive of OSA on subsequent PSG.

METHODS

The study was approved by the Mayo Clinic Institutional Review Board (ID 17-001873) as a minimal-risk study. We retrospectively included all adult patients undergoing OPO followed by PSG, in a 1-year period (July 1, 2014 to June 30, 2015) at a single academic medical center. Subsequently, we excluded patients with OPO interpreted as abnormal (ODI ≥ 5 events/h), performed on supplemental oxygen or on positive airway pressure therapy. Therefore, the final cohort included only patients with OPO studies done on room air and interpreted as normal (ODI < 5 events/h) by a sleep specialist. The main predictor variable was visual correlation between simultaneous increases in HRV and minimal oxygen desaturations (**Figure 1**). All included OPO studies were independently and blindly assessed for the main predictor variable by three raters: a sleep specialist, a clinical sleep fellow, and a research fellow. Prior to the blinded review of OPO studies, all three reviewers established the qualitative rules for identification of positive correlation between heart rate tracing and oxygen saturation tracing. In order for the study to be considered positive, both heart rate and oxygen tracing would show simultaneous episodic abnormalities manifested as concordant decline from the baseline tracings where heart rate vertical tracings converge toward the oximetry tracing and vice versa. **Figure 1** demonstrates examples of positive, negative, and uninterpretable OPO studies for visual correlation between HRV and desaturations. The primary independent review of each reviewer resulted in assignment of patients with positive visual correlation versus those with no visual correlation. In the next phase of review, assignments of all three reviewers were taken into consideration in order to reach the consensus agreement. If all three assignments of a patient were either positive or negative, the assignment was confirmed as such and considered as a consensus agreement. In the remaining cases, where one reviewer had an assignment different from that of two other reviewers, the consensus agreement was achieved by taking into account the assignment of two reviewers versus that of a single reviewer. For example, if one reviewer assigned 1, while two other reviewers assigned 0 for the same patient, consensus agreement was no correlation.

Other predictive variables included demographics, comorbidities, medications, and additional parameters reported on OPO, including ODI and heart rate (HR). The primary outcome was presence of OSA on the subsequent PSG, defined as AHI ≥ 5 events/h. Overnight pulse oximetry studies were performed with NONIN 2500 pulse oximeter and PROFOX software. PSG was scored based on The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications, Version 2.1.[9]

Categorical data were reported as frequencies and percentages, whereas continuous data were reported as medians and interquartile range (IQR). We used nonparametric tests assuming non-normalcy, Fisher exact and Wilcoxon rank-sum tests, for categorical and continuous variables, respectively. The interrater agreement with the consensus was analyzed with the kappa statistic. All variables from the univariate analysis, which were deemed either clinically pertinent or with a resulting alpha ≤ 0.1, were initially included into stepwise multivariate logistic regression analysis to assess for the independent effects. The second and final step in the multivariate logistic regression analysis included only variables with a resulting alpha ≤ 0.1. The clinical significance was assumed at alpha ≤ 0.05. We also calculated sensitivity, specificity, positive and negative likelihood ratios, and overall accuracy of binary classification by consensus agreement relative to the gold standard diagnosis of OSA on PSG. The statistical analysis

Table 1—Baseline characteristics in patients with and without OSA on PSG.

Baseline Characteristic	All	OSA	No OSA	*P*
Age (years)	54 (44, 68)	60 (52, 71)	50 (40, 67)	.009
Female, n (%)	44 (40)	13 (20)	19 (43)	.011
BMI (kg/m^2)	28.6 (25.2, 32.5)	29.1 (26.8, 31.9)	28.2 (23.9, 32.6)	.52
Tobacco use, n (%)	8 (7)	2 (6)	6 (8)	1.0
Hypertension, n (%)	52 (48)	21 (66)	31 (40)	.021
Cardiac disease, n (%)	30 (28)	14 (44)	16 (21)	.019
Atrial fibrillation, n (%)	20 (18)	8 (25)	12 (16)	.28
COPD, n (%)	23 (21)	8 (25)	15 (19)	.61
Neurovascular disease, n (%)	7 (6)	2 (6)	5 (6)	1.0
HR controlling medications, n (%)	39 (36)	19 (59)	20 (26)	.002
ODI (events/h)	1.8 (1, 2.7)	2.6 (1.7, 3.3)	1.5 (0.8, 2.5)	< .001
Consensus agreement, n (%)	54 (50)	24 (75)	30 (39)	< .001

Values are presented as median (interquartile range) or n (%). BMI = body mass index, COPD = chronic obstructive pulmonary disease, HR = heart rate, ODI = oxygen desaturation index, OSA = obstructive sleep apnea, PSG = polysomnography.

was performed with JMP Pro software (SAS Inc., Cary, North Carolina, United States).

RESULTS

There were 936 patients who had undergone overnight oximetry followed by PSG at our institution in a 1-year period (July 1, 2014 to June 30, 2015). Of these, 127 patients had overnight oximetry study on room air interpreted as normal (ODI < 5 events/h). The final cohort comprised 109 patients after excluding 18 patients who had normal OPO either on positive airway pressure therapy or supplemental oxygen. Of the 109 patients, 65 (60%) were females, median age was 54 years (44, 67), median body mass index (BMI) was 29 kg/m^2 (25, 32), and the median ODI was 1.8 events/h (1, 2.7). None of the baseline characteristics available at the time of the interpretation of OPO correlated significantly with the resulting ODI.

Based on the consensus agreement of 3 raters, 54 patients (approximately 50%) had episodic increase in HR variability simultaneous with minor perturbations on oxygen tracing (minimal desaturations). The kappa interrater agreement with the consensus was highest for the sleep specialist (82%), followed by the clinical sleep fellow (73%), and finally the research fellow (64%).

Thirty-two patients (29%) in the cohort with normal OPO studies (ODI < 5 events/h) were found to have OSA on subsequent PSG with the median AHI of 11.7 events/h (8.1, 21.4). Increasing age, male sex, comorbid history of hypertension, cardiac disease, and use of heart rate-controlling medications (beta blockers, calcium channel blockers, or antiarrhythmic agents), were significantly associated with the diagnosis of OSA on subsequent PSG (**Table 1**). The strongest univariate associations with OSA were noted with the consensus agreement and ODI. Using consensus agreement, 24 patients (75% of those in whom OSA was diagnosed) could have been accurately predicted by the visual correlation between increase in HRV and oxygen tracing perturbations on normal overnight oximetry (odds ratio [OR] 4.70, 95% confidence interval [CI] 1.87–11.8, $P < .001$). The performance measures of the consensus agreement of OPO and diagnosis of OSA on PSG are shown in **Table 2**. The patients with OSA had median ODI of 2.6 events/h (IQR 1.72, 3.28), whereas those without OSA had median ODI of 1.5 events/h (0.75, 2.5), which although statistically significant may not necessarily be a clinically relevant difference. The median AHI in patients with visual correlation between HRV and desaturations was 3.6 (0.5, 9.5) and in those with no correlation was 1.4 (0.4, 2.8). Among patients with positive visual correlation on normal overnight oximetry, 18% demonstrated moderate or severe OSA on subsequent PSG (AHI ≥ 15 events/h). In a sensitivity analysis, we excluded 20 patients with history of atrial fibrillation. Relative to the whole cohort, the resulting OR increased to 6.5 (95% CI 2.14–19.6) confirming better predictive power of the consensus agreement as patients with atrial fibrillation were more likely to have "uninterpretable" heart tracings for the episodic correlation with oxygen desaturations (**Figure 1C**).

Table 2—Binary classification of consensus agreement and OSA on PSG.

Measure	Value	95% CI
Sensitivity (%)	75	57–89
Specificity (%)	61	49–72
Positive likelihood ratio	1.92	1.36–2.71
Negative likelihood ratio	0.41	0.22–0.77
Accuracy (%)	65	55–74

CI = confidence interval, OSA = obstructive sleep apnea, PSG = polysomnography.

The patients with OSA, compared to those without OSA, had numerically lower median pulse rate and HRV based on the standard deviation of the heart rate on oximetry: 65 (58, 73) versus 68 (62, 78), and 4.85 (3.5, 6.2) versus 5.8 (4.6, 7), respectively, but these were not statistically different. However, OSA patients were significantly more likely to be using heart rate

Table 3—Two-step multivariate analysis for OSA on PSG.

Step 1	OR (95% CI)	*P*
Age	1.04 (0.99–1.08)	.07
Female	0.17 (0.05–0.59)	.002
BMI	1.04 (0.94–1.16)	.40
Cardiac disease	0.93 (0.26–3.37)	.91
Hypertension	1.18 (0.35–4.02)	.78
HR controlling meds	6.03 (1.47–24.71)	.008
ODI	1.81 (1.09–3.02)	.014
Consensus correlation	5.65 (1.65–19.37)	.003
Step 2	**OR (95% CI)**	***P***
Age	1.03 (0.99–1.08)	.07
Female	0.20 (0.06–0.63)	.0033
HR controlling meds	6.69 (1.91–23.5)	.0015
ODI	1.88 (1.18–3.11)	.0084
Consensus correlation	5.41 (1.63–18.05)	.0032

BMI = body mass index, CI = confidence interval, HR = heart rate, ODI = oxygen desaturation index, OR = odds ratio, OSA = obstructive sleep apnea, PSG = polysomnography.

controlling medications compared with those without OSA (OR 4.16, 95% CI 1.74–9.94, $P < .002$).

When adjusted in a two-step multivariate regression analysis (**Table 3**), consensus agreement was an independent predictor of OSA on subsequent PSG (OR 5.41, 95% CI 1.63–18.05, $P = .0032$). The predictive power of visually rated episodic HRV was independent of the fact that patients with OSA used heart rate-controlling medications more frequently.

DISCUSSION

Our study has shown that in the cohort of patients with normal OPO (ODI < 5 events/h), subjective visual correlation of episodic increases in HRV with the minimal oxygen desaturations is an independent predictor of OSA on subsequent PSG, even when adjusted by clinically pertinent demographic characteristics, comorbidities and medications. This is the first study to our knowledge that evaluated utility of episodic visual increase in HRV on normal overnight oximetry to assist in clinical prediction of OSA.

Numerous studies performed in the past 20 years have shown that during the PSG recording, HRV correlates well with OSA, as well as with respiratory effort arousals.[7,8] The HRV index directly correlated with the AHI, allowing prediction not only of the presence of the OSA but also of its severity.[10] This association of SDB with HRV is rooted in the autonomic nervous system physiology. Intermittent sleep-related hypoxemia and respiratory arousals may lead to sympathetic hyperactivity and rise in pulse and blood pressure, followed by a short-term increase in vagal activity and decrease in heart rate.[11] Given direct cardiac and hemodynamic effects of intermittent hypoxemia and respiratory arousals, it is not surprising that this phenomenon, sometimes termed sympathovagal balance, has been studied frequently in patients with cardiovascular disease.[12–14] However, several studies suggested that not all pathophysiological interactions can be explained by sympathovagal pathway alone.[8,15]

From the clinical standpoint, OPO is a frequently used test for evaluation of hypoxemia and as a screening method for SDB. To date, there is no validated or consensus-based professional standard for interpretation of OPO, and as a consequence there has been a significant interrater variability in its interpretation.[4] In a recent study, this variability persisted despite the use of a standardized worksheet, although the worksheet for interpretation was deemed useful by the study participants.[5] Although the oximetry saturation graph and pattern have been shown to be used consistently in the interpretations of OPO, only a small number of the physicians were shown to use a heart rate tracing pattern.[4,16] It is likely that the actual use of HRV in the community practice setting is even lower than in the academic setting. To our knowledge and after independent reviews of Medline, we did not find published references on the use of visual correlation of episodic HRV occurring simultaneously with minimal oxygen desaturations in adults with normal OPO. In 1992, investigators from Stanford University experimentally validated the use of the MESAM ambulatory device with four recording channels, which included a single EKG lead, a snoring monitor, a pulse oximeter, and a position sensor.[17] In this study, HRV was measured based on the R-R intervals from the EKG lead and alone had modest predictive accuracy (sensitivity 58% and specificity 32%). Also, simultaneous PSG recording was used, as in all other studies on the topic,[7,10,18–21] However, the translation of the findings into the routine clinical practice arena is lacking. Therefore, we performed our study with the main hypothesis and practical aim that the well-established finding of direct correlation of HRV with SDB could be clinically utilized by the sleep medicine practitioners and other providers who routinely interpret OPO. Indeed, we have shown that in patients with episodic HRV occurring simultaneously with minimal oxygen desaturations despite relatively normal oximetry results, odds of having diagnosis of OSA on the subsequent PSG were five times higher. Almost one in five of such patients had a diagnosis of moderate or severe OSA, despite having normal oxygen parameters on OPO. Although the patients selected by the consensus agreement or those with OSA on PSG had higher ODI than their respective counterparts, these differences were small, within the normal range of ODI, and as such would not have discerning power. Among all parameters routinely available to a physician during interpretation of normal OPO (ODI < 5 events/h), only visualized correlation in episodic HRV, apart from male sex, had a practical, strong predictive power relative to the diagnosis of OSA. Nevertheless, the visualized correlation in episodic HRV has not been routinely used as such in the clinical practice. As everyone in our cohort of patients with normal OPO subsequently underwent PSG, it is likely that these patients had high clinical risk of SDB, so our results can only be generalized to the similar patient populations. We believe that future studies and automatization of the subjectively visualized data may lead to standardization and increased utilization of this parameter in the diagnosis of SDB. We base this assumption, at least in part, on the fact that some existing diagnostic home

sleep apnea tests (HSAT) predominantly rely on both oxygen and pulse signals. In addition, the indication for HSAT is in part based on the pretest probability of OSA, which frequently factors in the results of OPO. Therefore, increased predictive ability of OPO interpretation that uses episodic HRV may allow performance of HSAT where it currently would not be indicated. This is only hypothetical and future studies could address this.

Our study has several important limitations. The subjectivity or interrater variability in visual rating of OPO was reported previously and also observed in our study. Although we did not employ specific training as e part of the study protocol, we noted that the previous experience in OPO interpretation was likely an important factor. Specifically, the sleep specialist showed very good agreement with the consensus, compared to the clinical fellow's good agreement, and finally the research fellow who had lower agreement rate (and no clinical sleep medicine training). This suggests that there may be a learning curve; although even at the level of the sleep specialist some variability would likely still exist. This calls for the conception of an automated correlation between oxygen and pulse waveforms by pulse oximeter machines. Such an automated algorithm would need to be derived and validated before widespread use.

There are limitations inherent in the retrospective nature of our study. Although OPO was performed prior to PSG, the data were abstracted simultaneously, causing possible unblinding. However, one of the investigators obtained the whole cohort data, while three other investigators reviewed oximetry results blinded to other results, including the PSG.

Another limitation would be interpretability of the pulse tracing in cases of atrial fibrillation or other cardiac rhythm disorders. We did not exclude such patients because we aimed to be all-inclusive to better reflect real-world practice. A sensitivity analysis confirmed that our conservative decision to include patients with history of atrial fibrillation only directed results toward the null hypothesis, thus decreasing predictable power of the main predictor variable.

Last, but equally important, the visual correlation of perturbations between oxygen saturation and heart tracing in our study was only nominal. It is possible that some quantifiable methods may provide better predictive power with respect to the severity of the underling OSA.

CONCLUSIONS

In summary, episodic increase in HRV occurring simultaneously with minimal oxygen desaturations on otherwise normal overnight oximetry is a strong independent predictor of OSA on subsequent PSG, despite the more frequent use of heart rate-controlling medications among patients with OSA. Future studies should derive and validate automated scalable algorithms for the prediction of OSA using both oximetry and pulse tracings.

ABBREVIATIONS

AHI, apnea-hypopnea index
HRV, heart rate variability
HSAT, home sleep apnea test
IQR, interquartile range
ODI, oxygen desaturation index
OPO, overnight pulse oximetry
OSA, obstructive sleep apnea
PSG, polysomnography
SDB, sleep-disordered breathing

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DISCLOSURE STATEMENT

All authors have seen and approved the manuscript. The authors report no conflicts of interest.