ECG-Derived Respiratory Power Index to Evaluate and Track Pediatric Sleep Apnea

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***Abstract*—** Pediatric obstructive sleep apnea (OSA) is characterized by apneic events, typically quantified using the apnea-hypopnea index (AHI). Due to limitations of the standard diagnostic test, overnight polysomnography, alternative methods are studied. This study assesses the Respiratory Power Index (RPI) in pediatric OSA, a surrogate AHI measure obtained from electrocardiogram-derived respiration signals. An exploratory analysis was conducted on the non-randomized group (654 children, grouped by OSA severity) from the Childhood Adenotonsillectomy Trial dataset to evaluate RPI changes with disease severity. Additionally, the RPI potential to monitor treatment response from baseline to follow-up group (332 children) was investigated. RPI effectively distinguished OSA severity groups and correlated positively with OSA severity indices. Moreover, RPI differentiated between children at baseline and follow-up, with changes attributable to alterations in OSA-related variables that includes both obstructive and central apneic events. These findings underscore the utility of RPI in pediatric OSA, enabling differentiation of severity groups and monitoring treatment response.

Keywords*—ECG-derived respiration, Respiratory Power Index, Pediatric Obstructive Sleep Apnea.*

I. Introduction

Pediatric obstructive sleep apnea (OSA) is a prevalent respiratory sleep disorder characterized by airflow disruptions known as apneas and hypopneas. The documented cardiovascular and neurocognitive implications of this condition underscore the importance of early detection and treatment [1]. However, the standard diagnostic test, overnight polysomnography (PSG), is complex, time-consuming, and particularly uncomfortable for children [1]. Consequently, there has been a growing interest for simplified alternatives to diagnose and study pediatric OSA consequences [2].

Several biological signals are collected during PSG and manually scored to extract the apnea-hypopnea index (AHI), providing an OSA diagnosis [1]. Using electrocardiogram (ECG) recording, the ECG-derived respiration (EDR) signal allows monitoring respiratory activity without acquisition of respiratory signals [3]. In a previous work, a surrogate measure of AHI called the respiratory power index (RPI) was derived through a joint analysis of EDR signals. However, this methodology was evaluated exclusively in adults [3], and its application in pediatric populations remains unexplored.

This study aims to adapt RPI extraction to the intrinsic properties of cardiac pediatric behavior due to OSA, and to evaluate, for the first time, its efficacy to differentiate OSA severity groups and monitoring post-treatment improvement.

II. Materials and Methods

A. Database

The Childhood Adenotonsillectomy Trial (CHAT) was a randomized controlled trial to assess the efficacy of pediatric OSA treatment in children aged 5 to 10 [4]. Our study comprised 986 children from CHAT, with 654 in the non-randomized group and 332 having two PSGs (baseline and follow-up). The first group was devoted to performing an exploratory analysis, stratified by OSA severity (No-OSA, AHI<1 event/hour (e/h); mild OSA, 1≤AHI<5 e/h; moderate OSA, 5≤AHI<10 e/h; and severe OSA, AHI>10 e/h). The second group aimed to assess RPI changes from baseline to follow-up. Table 1 provides demographic and clinical data of the included populations.

B. Respiratory Power Index extraction

RPI extraction involves integrating EDR signals [3]. It estimates individual respiratory power by combining multiple EDR signals and averaging their power spectral densities, emphasizing respiratory components. Later, non-respiratory components are reduced by estimating the instantaneous respiratory frequency, to finally estimate respiratory power. By the use of two adaptive thresholds based on power distribution ('selection' and 'minimum' levels,), an event qualifies as apneic if its power falls below the selection level during longer than a specified minimum duration and reaches the minimum level at least once [3].

To adapt RPI extraction to pediatric patients, considering the variance in the duration of apneic events between children and adults [1], we empirically shortened the minimum duration of an event to be classified as respiratory to eight seconds. Later, respiratory power was computed by integrating the power spectral densities of three EDR signals: R-peak amplitude, QRS complex, and respiratory sinus arrhythmia [5]. Figure 1 illustrates an example of a 5-min segment where a respiratory event is detected. RPI is computed as the number of events detected per hours of sleep.

C. Statistical Analysis

RPI differences among four OSA severity groups were assessed using the Kruskal-Wallis test. Differences between

TABLE I. Demographic and Clinical Data.

	Non-randomized	*Baseline*	*Follow-up*
#Subjects	654	332	332
Age (years)	6.90 [2.40]	6.00 [2.50]	7.00 [2.00]
Males (*n*)	313	160	160
BMI (Kg/m^2)	17.28 [4.64]	17.06 [6.90]	17.68 [7.49]
AHI (e/h)	1.45 [2.02]	5.51 [6.19]	2.10 [3.62]
AI (e/h)	0.81 [1.14]	2.25 [2.52]	1.00 [1.62]
HI (e/h)	0.52 [1.01]	3.22 [4.31]	0.90 [2.15]
OAHI (e/h)	0.77 [1.15]	4.55 [6.06]	1.23 [2.66]
OAI (e/h)	0.13 [0.42]	1.08 [1.98]	0.20 [0.62]
OHI (e/h)	0.52 [1.01]	3.22 [4.31]	0.90 [2.15]

Data are shown as median [interquartile range] or n; BMI: body mass index; AHI: apnea hyponea index; AI: apnea index; HI: hypopnea index; OAHI: obstructive AHI; OAI: obstructive AI; OHI: obstructive HI.

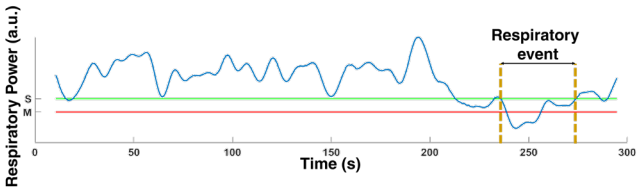

Fig. 1. Example of a 5-min segment where a respiratory event is detected. The blue line represents the estimated respiratory power from combinatorial analysis of EDR signals, and the event is detected based on the green line (selection level, S) and red line (minimum level, M).

baseline and follow-up were analyzed using Wilcoxon signed-rank test. Significance was defined as p-value < 0.05 after Bonferroni correction To complement our analysis, we computed a Spearman's partial correlation analysis, controlling for age, between RPI and various OSA-related variables (AHI; apnea index, AI; hypopnea index, HI; obstructive AHI; obstructive AI; obstructive HI). Similarly, partial correlation analysis was conducted between the change in these OSA-related variables and RPI in the second approach. Finally, causal mediation analysis (CMA) [6] was applied to investigate if treatment effects on RPI were mediated by changes in the OSA-related variables.

III. RESULTS & DISCUSSION

Figure 2 depicts boxplot distributions for both the exploratory analysis and treatment effects. Firstly, RPI differentiated OSA severity groups (p-value < 0.05 after correction), with RPI values increasing as OSA worsened. The partial correlation analysis supported this finding, revealing significant positive correlations between RPI and all severity indices, particularly when considering both obstructive and central apneic events (stronger correlations for AHI, AI, and HI compared to OAHI, OAI, and OHI, respectively). The differences between groups and the fact that RPI increased with severity, suggest that RPI may serve as a surrogate AHI measure also in pediatric OSA, as in adults [3].

RPI also distinguished between children at baseline and follow-up, with changes in most OSA-related variables, except OAHI and OAI, showing significant positive correlations with RPI changes. To verify that these changes were specifically linked to alterations in OSA severity, we conducted CMA. The analysis revealed that treatment impacted RPI, showing statistically significant causal mediation effects with OSA-related variables, except for those exclusively associated with obstructive events. Along with the CMA findings, the lower RPI in the follow-up group reflects that RPI effectively tracks the improvement experienced by children post-treatment, influenced by changes in severity. Despite central events are not accompanied by inspiratory effort, these events alter blood pressure and heart rate [7]. Thus, it seems that isolated obstructive events do not sufficiently influence RPI to monitor OSA, underscoring the importance of both obstructive and central apneic events in the RPI information.

IV. CONCLUSION

Usefulness of RPI in pediatric OSA, distinguishing between severity groups and tracking the progress of children post-treatment, has been confirmed. Its enhanced validity is observed when accounting for both obstructive and central apneic events, suggesting its potential as a biomarker for pediatric OSA post-treatment improvement.

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REFERENCES

[1] C. L. Marcus *et al.*, "Diagnosis and management of childhood obstructive sleep apnea syndrome," *Pediatrics*, vol. 130, no. 3, pp. e714–e755, 2012.

[2] H.-L. Tan, L. Kheirandish-Gozal, and D. Gozal, "Pediatric Home Sleep Apnea Testing: Slowly Getting There!," *Chest*, vol. 148, no. 6, pp. 1382–1395, Dec. 2015.

[3] M. M. Lyons *et al.*, "Screening for Obstructive Sleep Apnea in Commercial Drivers Using EKG-Derived Respiratory Power Index," *J. Clin. Sleep Med.*, vol. 15, no. 01, pp. 23–32, Jan. 2019.

[4] S. Redline *et al.*, "The Childhood Adenotonsillectomy Trial (CHAT): Rationale, Design, and Challenges of a Randomized Controlled Trial Evaluating a Standard Surgical Procedure in a Pediatric Population," *Sleep*, vol. 34, no. 11, pp. 1509–1517, 2011.

[5] C. Varon *et al.*, "A Comparative Study of ECG-derived Respiration in Ambulatory Monitoring using the Single-lead ECG," *Sci. Rep.*, vol. 10, no. 1, p. 5704, Mar. 2020.

[6] K. Imai, L. Keele, and D. Tingley, "A General Approach to Causal Mediation Analysis," *Psychol. Methods*, vol. 15, no. 4, pp. 309–334, 2010.

[7] D. M. O'Driscoll *et al.*, "Central apnoeas have significant effects on blood pressure and heart rate in children," *J. Sleep Res.*, vol. 18, no. 4, pp. 415–421, Dec. 2009.

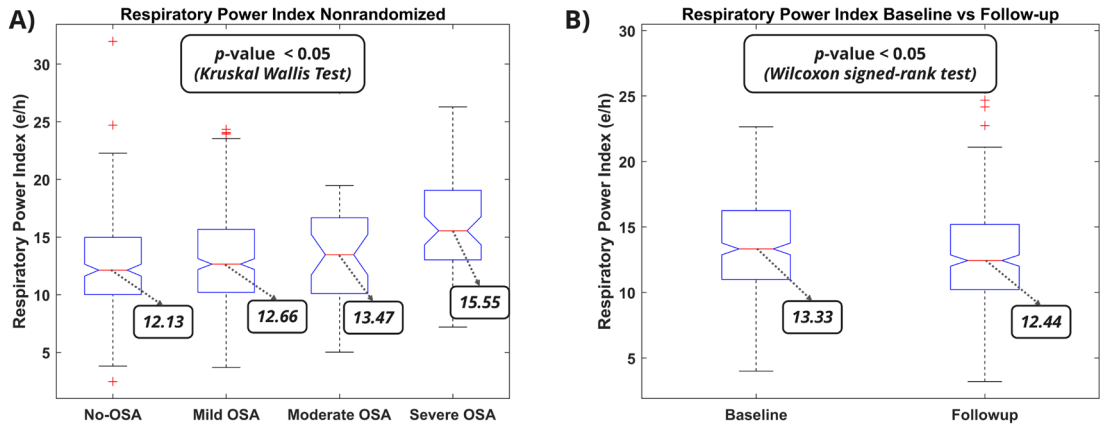

Fig. 2. Boxplot distribution of the RPI in the different analyses. The values of the median for each group, as well as the p-value resulting from each statistical comparison, have been depicted on the figure. A) Boxplot distribution of the RPI computed for each OSA severity group in the exploratory analysis. B) Boxplot distribution of the RPI computed for the same children at baseline and follow-up.