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Secondhand Smoke exposure and risk of Obstructive Sleep Apnea in Children☆

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ABSTRACT

Objectives: Obstructive sleep apnea (OSA) has a prevalence of 4% in children. Few studies have explored the role of secondhand smoke (SHS) on OSA severity and have shown contradicting results. Most studies have focused on the effect of SHS on snoring. This study explored the association of SHS exposure and OSA severity in children aged 3–18 years.

Methods: This is a retrospective single center IRB-approved study. Electronic Medical Records (EMR) were queried between 1/24/2015 and 1/24/2018 to obtain data on SHS exposure with standard questionnaires from perioperative database. SHS was analyzed as a binary variable and OSA was measured using obstructive apnea hypopnea index (OAHI) from polysomnography (PSG) as a continuous variable. Analyses were done on all children and in those with severe OSA (OAHI ≥ 10/h) as a subgroup.

Results: EMR query yielded 101,884 children of whom 3776 had PSG. Limiting baseline PSG in 3–18-year-old and reliable information on SHS yielded 167 analyzable children of whom 70 had severe OSA. Children exposed to SHS had significantly more public insurance than non-exposed ($p < 0.0001$). Among children with severe OSA, median OAHI was significantly higher in SHS exposed compared to non-exposed (29.0vs.19.5,$p = 0.04$), but not across all children. In multivariable analysis SHS exposure increased OAHI by 48% in severe OSA subgroup (95%CI: 8%–102%; $p = 0.01$) when adjusted for race, body mass index, and adjusted household income.

Conclusion: Children aged 3–18 years with severe OSA who were exposed to SHS were found to have 1.48 increase in odds of OAHI than those without SHS exposure. Results could be limited by retrospective nature of study and EMR tools.

1. Introduction

Obstructive sleep apnea (OSA) is the most severe pediatric sleep disorder that can contribute to significant morbidity and mortality [1,2]. Although literature supports upper airway architecture and neuromuscular tone as the main contributing factors for OSA, researchers have begun to focus on the role of environmental factors, like exposure to secondhand tobacco smoke (SHS), as an additional contributor to the etiology and severity of OSA. The associations between early exposure of children to SHS and respiratory comorbidity (infections, increased asthma attacks) are well documented [3]. Approximately 40 million non-smoking children in the U.S. are exposed to the toxins of SHS [3].

To date, despite the critically important role of sleep health to current and future medical and psychiatric well-being, there is remarkably limited data on the link between SHS exposure and OSA in children. The existing literature is limited to the study of habitual snoring since it is easier to study and has a higher prevalence (10%),

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compared to OSA, which has a comparatively low prevalence (4%) and also requires an overnight polysomnography (PSG). The relationship between OSA and SHS exposure has been proposed, although evidence to support such a relationship is very limited. Indeed, there are only few papers to date that have been published and the results were conflicting [4–6]. Although a recent systematic review [7] concluded that SHS is significantly associated with sleep disordered breathing, only one of 18 studies in the review was on OSA and SHS [5].

There is, therefore, a critical need to determine how exposure to SHS relates to OSA occurrence and severity. Without such knowledge, a conceptual framework for the subsequent development of prevention related to OSA will remain unlikely. The aim of this retrospective study was to explore the relationship of SHS exposure assessed by questionnaire and the polysomnographic severity of OSA in children aged 3–18 years. We focused on severe OSA as a clinically important subgroup at increased risk for significant morbidity and mortality.

2. Material and methods

This is a retrospective single center database review approved by the IRB with a waiver of consent. This manuscript is reported based on STROBE guidelines. Electronic Medical Records (EMR) were queried for anesthesia cases performed between 1/24/2015 and 1/24/2018 at our hospital.

This study was done as a secondary analysis of perioperative data collected to explore the relationship of SHS exposure and perioperative respiratory adverse events. Children were identified based on their encounter with anesthesia and data were obtained from multiple clinical EMRs such as EPIC, Chartmaxx, and Compurecord. Inclusion criteria included children aged 3–18 years old and who had valid questionnaire based SHS exposure assessment. Children who had SHS exposure assessment one year after the PSG were excluded from the analysis. The data fields were downloaded on an excel chart. PSG outcomes, SHS exposure assessment, as well as demographic characteristics including age, gender, race, ethnicity, body mass index (BMI), procedure performed, type of medical insurance, and household zip code were queried. Household income was further estimated based on the median household income by zip code based on information from U.S. Census Bureau, 2013–2017 American Community Survey 5-Year Estimates factfinder census.

2.1. SHS exposure

Exposure to SHS was documented via a questionnaire that was administered during patient intake in outpatient and inpatient encounters and recorded in the EMR. Patient screening for tobacco use and SHS exposure is performed routinely as part of the Meaningful Use program for Electronic Health Record implementation [8]. The smoking status was analyzed based on the following categories: current every day smoker, current some day smoker, former smoker, heavy tobacco smoker, light tobacco smoker, never assessed, and never smoker. Children were classified as exposed to SHS or not exposed to SHS based on these responses. Children who were classified as exposed to SHS (in other words passive smoke exposure in children who have never smoked) belonged to families who were positive to one or more of the following categories: current every day smoker, current some day smoker, former smoker, heavy tobacco smoker, and light tobacco smoker. Children who were classified as not exposed to SHS belonged to families who reported “never smoker”. Data reliability on SHS status was maintained by including only those children where the details of smoke exposure was additionally typed in a free text box.

2.2. Polysomnography (PSG)

PSG was performed in our hospital sleep laboratory accredited by the American Academy of Sleep Medicine. PSG results were scored by sleep medicine boarded physicians. Initial potential subject lists were queried from multiple sources of the EMR. Subsequently, the EMR was queried for sleep study results on these children. Then, manual chart review collected PSG results. All PSG's were done preoperatively. The following data were collected on PSG: days from PSG to SHS documentation, total recording time, total sleep time, sleep efficiency, sleep latency, obstructive apnea/hypopnea, central apnea/hypopnea, mixed apnea/hypopnea, saturation, peak end-tidal carbon di-oxide ($EtCO_2$), and other variables during rapid eye movement (REM), non REM sleep or both. Severity of OSA was classified based on OAHI. OAHI < 1.5/h was normal; 1.5–5.0/h was mild OSA; 5.1–9.9/h was moderate OSA; and OAHI ≥ 10/h was classified as severe OSA. We chose to study 3 to 18-year-old children most of whom are operated as day surgery compared to < 3-year-old who are managed with inpatient monitoring [9].

2.3. Statistical analysis

All analyses described were completed for the full sample and a subset of those with severe (OAHI ≥ 10/h) OSA (n = 70). Baseline and demographic characteristics were summarized by standard descriptive summaries, e.g., means and standard deviations (SDs) or medians and interquartile ranges (IQRs) for continuous variables, and frequencies and percentages for categorical variables. Two sample t-tests or the Wilcoxon rank-sum test were used to compare differences in continuous variables between children with SHS and without SHS, while Chi-square or Fisher's exact test, as appropriate, were used for categorical variables. SHS was analyzed as a binary variable and OSA was measured using OAHI as a continuous variable. Distributions were checked for normality by examining histograms and tests of kurtosis and skew. Due to extreme skewness, the OAHI index was log-transformed for linear regressions. Simple linear regressions were used to assess the bivariate relationships between SHS, as well as other demographic characteristics, with log-OAHI. Multivariable linear regression was further conducted to control for confounders of the association between SHS and OAHI index. Race was considered an *a priori* confounder and therefore included in the model. Backward elimination, with $\alpha_B = 0.2$, was then used for other covariate selection. Candidate variables included age at PSG study, BMI at PSG study, gender, ethnicity, procedure type (i.e. airway and non-airway), insurance type (i.e., private and public) and estimated household income. Separate models were fitted for all 167 children in the sample and for those with severe (OAHI ≥ 10/h) OSA (n = 70). A p value of < 0.05 was considered significant. The statistical analysis was done with SAS software version 9.4 (SAS institute Inc., Cary, NC).

3. Results

The query yielded data on 101,884 children undergoing anesthesia over the three years, of whom 3776 also underwent a PSG. After going through all the exclusions, only 167 children were allocated for the final analysis of which 90 (53.9%) were exposed to SHS and 77 (46.1%) were not exposed to SHS. Most exclusions occurred due to inconsistency in data collection on smoking related variables and non-baseline PSG's (Fig. 1). Out of the 167 children with PSG, 16 had no OSA, 46 had mild, 35 moderate, and 70 severe OSA. A subgroup analysis was performed on children with severe OSA where 38 (54.3%) children were exposed to SHS and 32 (45.7%) were not exposed to SHS. There were 3 children whose parents had quit smoking and were included in the analysis as exposed group.

3.1. Demographics and PSG

Patient demographic characteristics are summarized in Table 1. The mean age from all children was 6 years (SD = 4.3). There were 13 children (7.8%) aged ≥10 years old and did not differ with SHS

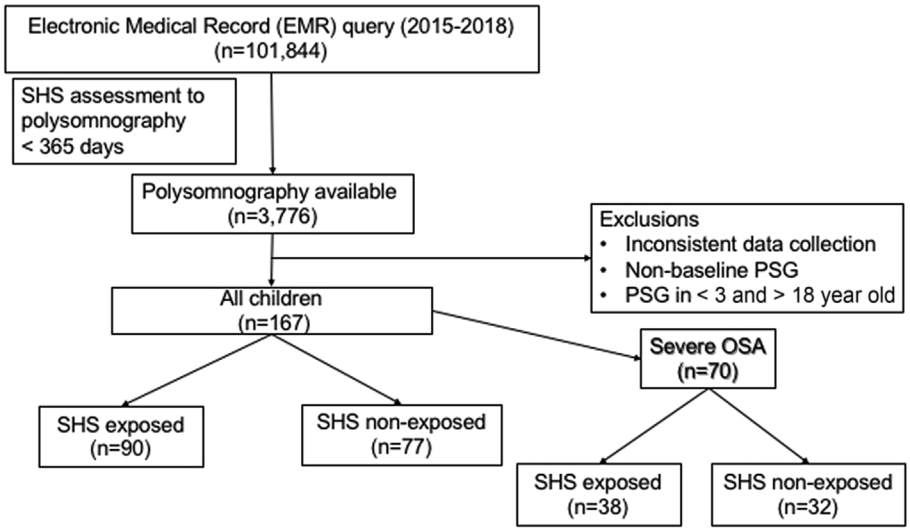

Fig. 1. STROBE flow diagram.

exposed and non-exposed groups. Children exposed to SHS had significantly more public insurance than non-exposed ($p < 0.0001$). (See Table 1). Table 2 shows the PSG related variables. In children with severe OSA, OAHI was significantly higher in SHS exposed compared to SHS non-exposed group ($p = 0.04$).

3.2. Regression analysis

Table 3 shows the unadjusted simple linear regression analyses individually predicting log OAHI from SHS, age, BMI, gender, ethnicity, race, type of procedure (airway vs. non-airway), insurance (private vs. public), and estimated household income. In the full sample, procedure type is the only predictor with a significant association with OAHI ($p < 0.001$), and in the severe OSA group, SHS is the only predictor with a significant association ($p = 0.03$).

In the multivariable analysis for the full sample, SHS exposure was not related to log-OAHI after adjusting for race and procedure type. In the multivariable model of the subset of children with severe OSA, SHS exposure ($p = 0.01$) was an independent predictor of OAHI after adjusting for BMI, race, and estimated household income (Table 4).

4. Discussion

Children aged 3–18 years with severe OSA who were exposed to SHS were found to have 1.48 increase in odds of OAHI than those without SHS exposure, in multivariable analysis. Pediatric OSA is a disorder with significant comorbidity that causes poor sleep quality [10,11], and evidence in the literature supports that treatment of OSA improves sleep quality [12]. OSA in children is strongly associated with a range of perioperative respiratory complications [13,14], long-term behavioral (neurocognitive dysfunction, hyperactivity, inattentive behaviors) [15–17] and medical (obesity, increased blood pressure, diabetes, changes in heart and vascular geometry) [18,19] problems, and death [1,2]. SHS is known to cause sleep deficiency in both adolescents [20] and adults [21]. To date, despite the critically important role of smoking and sleep health, there is remarkably limited data on the link between SHS exposure and OSA. The existing literature is summarized in a recent systematic review [7]. This literature is limited to the study of habitual snoring since it is easier to study and has a higher prevalence (10%), compared to OSA, which has a comparatively low prevalence (4%) and also requires PSG.

Very few published studies have explored the relationship of

Table 1
Demographic characteristics comparing children with versus without secondhand smoke exposure.

N	All children	All OSA children (n = 167)			Severe OSA (n = 70)		
		SHS exposed	SHS non-exposed	p value	SHS exposed	SHS non-exposed	p value
	167	90 (53.9%)	77 (46.1%)		38 (54.3%)	32 (45.7%)	
Age (years)	6 (4.3)	5.9 (2.4)	6.2 (2.6)	0.41	6.1 (2.2)	6.1 (2.8)	0.90
Body Mass Index[a]	18.4 (5.6)	18.8 (6.0)	18.0 (5.1)	0.38	19.4 (6.4)	18.6 (6.3)	0.61
Gender - Male	83 (49.7%)	45 (50%)	38 (49.4%)	0.93	18 (47.4%)	14 (43.8%)	0.76
Ethnicity - Hispanic[b]	16 (9.8%)	8 (9.2%)	8 (10.4%)	0.80	4 (10.8%)	2 (6.3%)	0.68
Race				0.78			0.56
African American	63 (37.7%)	33 (36.7%)	30 (39.0%)		13 (34.2%)	15 (46.9%)	
White	74 (44.3%)	42 (46.7%)	32 (41.6%)		18 (47.4%)	12 (37.5%)	
Others/refused	30 (18.0%)	15 (16.7%)	15 (19.5%)		7 (18.4%)	5 (15.6%)	
Procedure - airway	150 (89.8%)	83 (92.2%)	67 (87.0%)	0.27	38 (100%)	31 (96.9%)	0.46
Insurance - private	66 (39.5%)	21 (23.3%)	45 (58.4%)	< .0001	8 (21.1)	22 (68.8%)	< .0001
Estimated median household income (1,000)[c]	60.5 (27.1)	59.4 (26.0)	61.7 (28.3)	0.59	57.8 (24.9)	66.49 (30.9)	0.19

Data presented as mean (SD) or n (%).
OSA = Obstructive Sleep Apnea; SHS: Secondhand Smoke.
[a] 4 children missing body mass index value at sleep study.
[b] 3 children refused the question for ethnicity.
[c] 1 child missing zip code information.

Table 2
Comparing variables from polysomnography by children with versus without secondhand smoke exposure.

N	All children	All OSA children (n = 167)			Severe OSA children (n = 70)		
		SHS exposed	SHS non-exposed	p value	SHS exposed	SHS non-exposed	p value
	167	90 (53.9%)	77 (46.1%)		38 (54.3%)	32 (45.7%)	
Days from PSG to SHS assessment	124.2 (76.8)	125.1 (83.1)	123.1 (69.2)	0.87	101.9 (67.6)	96.2 (65.4)	0.72
Severity of OSA				0.99	N/A		
None (AHI 0–1.5)	16 (9.6%)	9 (10%)	7 (9.1%)				
Mild (AHI 1.6–5.0)	46 (27.5%)	25 (27.8%)	21 (27.3%)				
Moderate (AHI 5.1–9.9)	35 (21.0%)	18 (20.0%)	17 (22.1%)				
Severe (AHI ≥ 10)	70 (41.9%)	38 (42.2%)	32 (41.6%)				
Total recording time	515.8 (40.0)	515.3 (39.2)	516.2 (40.9)	0.89	507.1 (32.4)	513.9 (38.1)	0.42
Total sleep time	430.2 (54.7)	433.7 (52.5)	426.1 (57.2)	0.38	424.3 (35.7)	434.8 (50.4)	0.33
Sleep efficiency	83.4 (8.5)	84.1 (7.9)	82.6 (9.2)	0.25	83.8 (6.8)	84.6 (6.8)	0.66
Peak $EtCO_2$ during total sleep	52.0 (4.2)	52.6 (4.3)	51.3 (4.0)	0.05	54.2 (5.0)	53.3 (4.1)	0.41
Sleep latency	38.6 (30.6)	41.3 (33.4)	35.0 (27.0)	0.19	42.6 (37.3)	29.3 (19.2)	0.06
Apnea Hypopnea Index (Median (IQR))	8.1 (3.1–19.3)	8.2 (3.1–25.1)	7.8 (3.3–16.2)	0.62	29 (18.9–39.1)	19.5 (13.5–31)	0.04
Minimum SpO_2 during total sleep	85.9 (7.6)	85.8 (8.0)	86.1 (7.1)	0.77	81.4 (9.5)	82.2 (8.2)	0.70

Data presented as mean (SD) or n (%).
AHI = Apnea Hypopnea Index; EtCO2: End tidal carbon dioxide; IQR = Interquartile Range.
OSA = Obstructive Sleep Apnea; PSG = Polysomnography; SHS: Secondhand Smoke; SpO2 = Oxygen saturation.

pediatric SHS exposure and OSA, and the results were contradictory. The first study by Weinstock et al. assessed the relationship between SHS and OSA [4]. The major strength of that study was focusing on the potential influence of race on OSA severity and the results showed that both SHS exposure and African American race increased OSA severity by 20% as measured with apnea hypopnea index (AHI). Another strength of that study was that the data were collected from pediatric centers around the U.S. The study was performed as a secondary analysis of a larger study and showed promising results. This study was limited to children younger than 10 years and our study provides further evidence of the relationship between OSA severity and SHS exposure in children with severe OSA up to 18 years of age. The second study by Tamayan et al. explored if demographic or clinical factors predict OSA severity in a 3-17-year-old cohort from Australia [6]. They found that paternal smoking was associated with a 53% increased risk of moderate to severe OSA measured by PSG. The moderate/severe OSA group was defined as an AHI > 5 and comprised 75 children. Socioeconomic status was not a risk factor. Unlike our study, the data collection on smoking related variables is unclear and smoking was not the primary independent variable. The third study to explore the relation of SHS with OSA by Kahn et al. assessed the effect of prenatal smoking on infant respiratory behavior during sleep [5]. This was a well conducted multicenter study with PSG performed on infants. The authors showed that only prenatal maternal smoking, and not postnatal exposure, was associated with OSA. Compared to maternal smoking, neither prenatal nor postnatal paternal smoking increased the risk for OSA. Although no explanation was offered for this varied finding on maternal and paternal smoking in this study [5], postnatal efforts to limit SHS exposure may prevent adverse events. This study, however, focused only on infants, when OSA is infrequent and did not control for any confounders [22]. In two of the above studies [4,5], SHS exposure was assessed using self-report questionnaires. Although self-report questionnaires have reliability [23], there are concerns about false reporting without the use of a specific biomarker like cotinine, a metabolite of nicotine [24]. Our retrospective study is also based on self-report questionnaire and we emphasize the importance of prospective biomarker based studies.

The U.S. high school students reporting having used electronic cigarettes jumped from 2% in 2011 to 16% in 2015 [25]. With the perception of low risk with the use of e-cigarettes, the use is expected to increase. Our study did not specifically look at e-cigarette exposure. The EMR documentation may be sporadic, non-standardized, and may not easily distinguish between e-cigarettes and combustible cigarette. A family may answer “no” to tobacco use, but may actually be using e-cigarettes with nicotine. Although our study did not specifically categorize e-cigarettes from combustible cigarettes, we certainly expect to see more children exposed to SHS with e-cigarettes and increased teenage e-cigarette smokers. Furthermore, e-cigarette smoking further increases the risk of future combustible cigarettes [25]. Hence, the harmful effects of SHS exposure including that on the severity of OSA is critical information to help develop interventions for two public health problems: OSA and SHS.

Table 3
Unadjusted Analysis for Log-transformed obstructive apnea hypopnea index.

	All children (n = 167)		Children with severe OSA (n = 70)	
	Parameter Estimate (SE)	p value	Parameter Estimate (SE)	p value
Secondhand smoke exposure (yes vs. no)	0.10 (0.22)	0.6601	0.30 (0.14)	0.0296
Age	0.003 (0.04)	0.9443	0.01 (0.03)	0.6327
Body mass index	0.01 (0.02)	0.4577	0.02 (0.01)	0.0839
Gender (Male vs. Female)	0.03 (0.22)	0.8828	0.11 (0.14)	0.4513
Ethnicity (Hispanic vs. non-Hispanic)	−0.05 (0.37)	0.8843	−0.33 (0.25)	0.1972
Race (White vs. African American)	−0.30 (0.24)	0.2159	−0.05 (0.16)	0.7710
(Other/Refused vs. African American)	0.19 (0.31)	0.5380	0.01 (0.20)	0.9580
Procedure (Airway vs. non-Airway)	1.89 (0.33)	< .0001	0.42 (0.59)[a]	0.4796
Insurance (Private vs. Public)	−0.14 (0.22)	0.5186	−0.21 (0.14)	0.1295
Estimated median household income (1,000)	−0.001 (0.004)	0.8032	0.0005 (0.003)	0.8525

[a] Only 1 out of 70 children was non-airway procedure.

Table 4
Multivariable Analysis for Log-transformed obstructive apnea hypopnea index[a].

	All children (n = 167)		Children with severe OSA (n = 70)	
	Parameter Estimate (SE)	p value	Parameter Estimate (SE)	p value
Intercept	0.47 (0.35)	0.2054	2.46 (0.32)	< .0001
Secondhand Smoke (SHS) exposed (yes vs. no)[b]	0.001 (0.21)	0.9961	0.39 (0.16)	0.0148
Exp (SHS) & 95% CI	1.00 (0.66, 1.51)		1.48 (1.08, 2.02)	
Body mass index (BMI)			0.02 (0.01)	0.1104
Exp (BMI) & 95% CI			1.02 (1.00, 1.04)	
Race[b] (Other/Refused vs. African American)	0.10 (0.30)	0.7332	−0.20 (0.23)	0.3851
Exp (Other/Refused vs. African American) & 95%CI	1.11 (0.61, 1.99)		0.82 (0.52, 1.29)	
(White vs. African American)	−0.30 (0.23)	0.1937	−0.30 (0.24)	0.2118
Exp (White vs. African American) & 95%CI	0.74 (047, 1.16)		0.74 (0.46, 1.19)	
Procedure (Airway vs. non-Airway)	1.83 (0.34)	< .0001		
Exp (procedure) & 95% CI	6.23 (3.20, 12.14)			
Estimated median household income (per 1000 increase)			0.006 (0.004)	0.1269
Exp (1000 increase in estimated median household income) & 95%CI			1.006 (0.99, 1 .01)	

[a] Backward elimination was used for variable selection, with F statistics significant at SLSTAY ≤ 0.2.
[b] Secondhand smoke exposure and race type were forced into the model regardless of backward elimination method.

4.1. Limitations

The main limitation of the study was the inconsistency of data collection related to SHS exposure which significantly limited the sample size available that may result in selection bias. Although the smoking variables existed as best practice evidence on the EMR, inconsistency resulted due to variable interpretation of who the smoking variables should be applied to (child vs. family). This problem with data collection has been described by other investigators [26]. Hence, we included data only when the smoking variable information was documented by typing in a free text box on EMR. This study was a good learning opportunity and helped us create standardized definitions from the American Cancer Society, the Center for Disease Control, and the PhenXToolkit, which is an FDA data collection repository, and various other publications to refine our data collection on smoking related variables. Another limitation is that the smoking related variables were collected by self-reported questionnaires with no objective verification with biomarkers. Although data suggests that questionnaires reliably estimate smoke exposure [23], other data question this [24]. Nevertheless, only children whose EMR had a completed free-text description of the smoking status (who smoked and where) were included, to maximize the reliability of data collection. Although the procedure status was not an independent predictor for severity of OSA, all our children had an airway procedure except for one in the severe OSA group. This has to be interpreted with caution since most children who have severe OSA undergo airway procedure as their primary treatment. Another limitation is a possible selection bias where most children in our sample had OSA. Given that tonsillectomy is the typical first line treatment for OSA in children, children who undergo anesthesia as well as PSG are much more likely to have been found to have higher OAHI index on their PSG than a representative sample of children from the general population not selected based on history of surgery. Lastly, we did not control for known or unknown confounders like asthma, obesity, allergies, pets, urban/rural, diet, parental OSA, etc. [27]. In spite of the limitations of the study methodology, we would like to emphasize the learning process of data collection related to smoking by creating standardization amongst front line health care workers.

5. Conclusions

Based on questionnaire assessment measuring SHS exposure and severity of OSA on PSG, children aged 3–18 years with severe OSA who were exposed to SHS were found to have 1.48 increase in odds of OAHI than those without SHS exposure, in multivariable analysis. This does not mean causation and further research is needed to further ascertain this result. Results however are limited by the retrospective nature of the study, EMR tools, and a weak association in the subgroup but not the full sample analysis. This study created a learning opportunity to review the data collection on smoking and to create standardized infrastructure that can help refine measures and improve our understanding of risk factors for OSA.

Author declaration

All authors have seen and approved the final manuscript as submitted.

Clinical trial number

Not Applicable.

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Declaration of competing interest

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Abbreviations

OAHI	Obstructive Apnea Hypopnea Index
EMR	Electronic Medical Record
$EtCO_2$	End tidal carbon dioxide
FDA	Food and Drug Administration
IQR	Interquartile Range
OSA	Obstructive Sleep Apnea
PSG	Polysomnography
SHS	Second Hand Smoke
SpO_2	Oxygen saturation

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijporl.2019.109807.

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