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1. Introduction

Childhood obstructive sleep apnea (OSA) is characterized by recurring events of partial (hypopnea) or complete upper airway obstruction (apnea) associated with oxygen desaturation and arousals/awakenings during sleep. This can disrupt normal sleep architecture and breathing [1]. Untreated OSA in childhood is associated with detrimental effects on cardiovascular functions, development, growth [2], behaviour, and neurocognitive function [3]. Therefore, diagnosis and management of this condition is a public health priority.

The gold standard for diagnosing OSA in children is a comprehensive sleep study, a polysomnography (PSG), performed in a sleep lab [4]. It measures sleep stages as well as breathing parameters during sleep, which allow to calculate the number of apnoeas and hypopneas per hour of sleep (AHI).

Conducting a PSG is expensive and time consuming, and hence only available to a small fraction of the overwhelming number of children in need of accurate OSA assessment. Thus, the majority of clinical settings treating children with obstructive sleep disordered breathing are unable to add PSG to the standard diagnostic procedures [5], although, studies have shown that a clinical diagnosis correlates poorly with the presence or severity of OSA as confirmed by PSG [6,7].

Respiratory polygraphy (PG) is a simplified version of the PSG, offering cardiorespiratory monitoring without measuring brain activity and, thus, no sleep staging or arousals are reported. In adults, PG is widely used for diagnosing OSA, but the validity of PG in children is still controversial [4]. Of the few studies previously conducted, the majority have found PG to underestimate the AHI compared to PSG to various extents [8–11] and others did not confirm this underestimation [12].

According to the American Academy of Sleep Medicine (AASM), PSG is recommended over PG for diagnosing OSA in children due to insufficient data on the validity of PG. However, they also recommend that the resources at hand should be taken into consideration. The International Pediatric Otolaryngology group and the European Respiratory Society (ERS) Task Force recommend the use of either PSG or in-lab PG when objective testing is indicated [13,14].

In Europe, there is a tendency to perform an in-lab respiratory PG (PG lab) instead of PSG [8].

However, three crucial challenges of using PG in children are 1) obtaining PGs of sufficient quality for scoring, 2) assessing accurate total sleep time for calculation of the AHI without measuring sleep stages, and 3) scoring hypopneas causing arousals without desaturation [4].

The aims of this study were to address the second and third challenges of PG in children, by comparing the performance and diagnostic value of PG home and PG lab versus gold standard PSG for baseline and follow-up evaluation. Additionally, we aimed to validate the PG at home to in-lab PG.

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2. Materials and methods

Children aged 2–10 years were enrolled from two Departments of Otolaryngology and three practitioners of otolaryngology in Central Denmark Region, during 2019 and 2020. All children included had tonsillotomy, with or without adenoidectomy, performed. The eligibility for surgery was based on "treatment as usual", a clinical evaluation of tonsil size and adenoids combined with parent reported disordered breathing during sleep, and daytime symptoms indicative of childhood OSA. Such daytime symptoms could be daytime sleepiness, poor school performance, behavioural problems, failure to thrive and nocturnal enuresis.

An unattended type III home sleep apnea test, also called respiratory home polygraphy (PG home) was performed at home and a gold standard polysomnography (PSG) was performed in a sleep lab before surgery. Both recordings were repeated three months after surgery.

2.1. Sleep studies

All PSGs were performed in accordance with the AASM recommendations [15], by trained personnel mounting the device and attending the recording throughout one night.

In order to test the PG performance without night-to-night variation, an in-lab PG (PG lab) was derived from the PSG. The PG lab was, hereby, made from the same recording as the PSG by deleting the electroencephalogram (EEG), electrooculograms (EOG), and chin electromyogram (EMG), video and thermistor leads. Thus, PG lab recordings with leads equivalent to the PG home recordings were created. The NoxA1 (Nox Medical®) was used for PSG recording and the Nox T3 (Nox Medical®) was used for PG home recording. This enabled using the same software and sensors for all three sleep recording modalities (Table 1).

Home PG recording was conducted by parents after careful instruction on mounting the Nox T3. Instructions were given by a nurse or the investigating physician (TKL), at one of four inclusion sites. The instruction procedure was conducted in accordance with a previous study on optimizing PG home signal quality [16]. In case of failed recordings, a repeated recording was performed when possible.

The PSG with PG lab was scheduled as close as possible to execution of the PG home.

2.2. Scoring

All sleep recordings were pseudonymized after end of follow-up in 2021, removing all identifiable data, and subsequently scored in random order. This enabled blinding the scorer to the results of the other sleep studies of each patient.

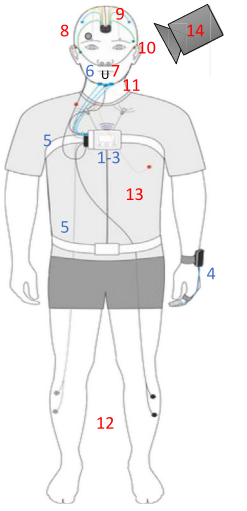
Two trained sleep scorers each scored a random half of the EEGs. Initially, five EEGs were scored independently by both scorers and subsequently reviewed and compared, epoch by epoch, to optimize scoring consistency. All respiratory scoring of PSGs and PGs was conducted manually by only one of the scorers. Sleep stages and arousals were scored using EEG, EOG, and chin EMG. Nasal cannula, inductance plethysmography of thorax and abdominal respiratory movements, oximetry, and electrocardiogram were used to score the respiratory parameters, as recommended by the AASM. All recordings were scored using the Noxturnal version 6.1 interface (Nox Medical®).

2.2.1. Defining respiratory events

A desaturation was defined as a 3% drop in SpO_2. An apnoea was defined as an airflow amplitude decrease of ≥90% compared to baseline for a minimum duration of two breaths. Central apnoeas were only scored when associated with a desaturation or an arousal or when lasting ≥20 s. Hypopneas were scored when airflow amplitude decreased ≥30%, for a minimum of two breaths associated with a desaturation and/or an arousal. In PG home and PG lab, information about arousals was not available. Therefore, only

Table 1
Devices and Sensors used for Sleep Recording.

Devices and Sensors used for Sleep Recording:	PSG	PG lab	PG home
Noxturnal 6.1 Software	X	X	X
1. Recording device	Nox A1	Nox A1	Nox T3
2. Position/movement sensor	X	X	X
3. Microphone on chest for detecting snoring	X	X	X
4. Pulse-oximetry:	X	X	X
Nonin® 3150 Wristox2 Bluetooth Wrist Pulse Oximeter, using pediatric sized or medium sized finger probes as appropriate (WristOx2 Soft Sensor)			
5. Thorax and abdomen belts: calibrated respiratory inductive plethysmograph (RIP)	X	X	X
6. Nasal cannula	X	X	X
7. Thermistor (Child airflow sensor-Key connector)	X		
8.Transcutaneous CO2 monitoring (Sentec)	X		
9. Electroencephalogram with frontal, central, and occipital leads (FP1, FP2, C3, C4, O1, O2, A1, and A2)	X		
10. Electrooculogram	X		
11. Chin electromyogram	X		
12. Leg electromyogram (one on each leg)	X		
13. Electrocardiogram	X		
14. Real time video recording	X		



PSG: Polysomnography. PG lab: Respiratory polygraphy performed in a sleep lab. PG home: Respiratory polygraphy performed at home. Illustration from Nox Medical instruction material.

hypopneas associated with a desaturation and central apnoeas associated with either a desaturation or lasting ≥20 s, were scored. The obstructive apnoea-hypopnea index (oAHI) was defined as the total number of obstructive apnoeas, mixed apnoeas and hypopneas divided by total sleep time in hours. In accordance with the ERS Task Force statement of 2016, childhood OSA was defined by a PSG oAHI≥2 or oAI≥1 [14]. There is consensus that children with an oAHI≥5 should be treated regardless of clinical symptoms and children with mild OSA (oAHI<5) should only be treated if displaying symptoms of sleep disordered breathing [14]. Therefore, analyses of PG performance were made for a diagnostic cut-off of oAHI≥2 or oAI≥1 and a treatment cut-off of oAHI≥5.

2.2.2. Assessing total sleep time

Total sleep time (TST) for PSG was defined by the total time spent in either REM sleep or non-REM sleep, defined by EEG. For PG lab and PG home an estimated total sleep time was defined. This was estimated by the investigator based on signals indicating that the patient was asleep (i.e., decrease in body movement in actigraphy and belt artefact indicating a resting state and breathing becoming more regular indicating sleep). These times were adjusted according to parent or nurse reported sleep times, when appropriate (i.e., when reported sleep times agreed with signals, or reported times of wake periods during the night).

2.2.3. Data quality

The minimum criterion for an acceptable recording was 4 h of artefact free signals on all leads. During periods of nasal cannula artifacts, using respiratory inductive plethysmograph (RIP) flow scoring as a substitute, was only acceptable when preceded by a valid airflow signal. Recordings missing airflow or thermistor signal entirely or with less than 4 h of artefact free recording of essential leads; belts, airflow, and oximetry, were excluded and categorized as fails.

2.3. Statistics

Based on an intraclass correlation coefficient (ICC) of 0.843 for PG home, reported by Alonso-Alvarez et al. [12], a sample size of 12 children would be required to obtain a 0.02 probability of a type I error and a 0.10 probability of at type II error. Due to the clinical inclusion criteria of the present study, the distribution of OSA severity was unknown before PSG scoring, which was conducted at end follow-up. Therefore, we aimed to include approximately 50 children to ensure confidence within the simple snorers, mild and moderate-to-severe OSA severity groups.

The mean respiratory events indexes for PG home, PG lab and PSG were compared using a paired *t*-test or Wilcoxon signed rank test depending on data distribution. Assumptions of variance homogeneity and difference homogeneity were assessed using a Bland-Altman plot and normality of the difference by a Q-Q plot. Limits-of-agreement of 95% between PG and PSG were computed using a paired-normal model. Sensitivity, specificity, negative predictive value, and positive predictive values were calculated. Receiver operating characteristic (ROC) curves comparing PG home and PG lab to PSG were computed and intraclass correlation coefficients were calculated. Estimates are presented with 95% confidence intervals.

Statistical analysis was performed using Stata version 16 (Statacorp, Texas, USA).

2.4. Ethical considerations

The study was approved by the Institutional Review Board, and the Scientific Ethical Committee of Central Denmark Region. Requirements on privacy and informed consent from patients and parents were met. The study was registered at the Danish Data Protection Agency and was performed in accordance with the Declaration of Helsinki. An independent senior consultant reviewed the respiratory part of the PSG to rule out central sleep apnea as a differential diagnosis before surgery and screened the post-operative PSG for residual OSA, so patients in obvious need of intervention did not need to wait for the meticulous blinded scoring to be completed.

3. Results

3.1. Study population and data

The clinical characteristics of the included patients are summarized in Table 2.

At baseline, 49 PSG, 49 PG lab, and 36 PG home recordings of acceptable quality were obtained. Three patients discontinued the study before follow-up. At follow-up, 49 PSG, 49 PG lab and 34 PG home recordings of acceptable quality were obtained. A flow chart of obtained records is enclosed as appendix 1. The main cause of recording failure was poor quality of the nasal cannula lead (4/103 (4%) in lab recordings and 7/85 (8%) PG home recordings). Signal quality in oximetry lead was significantly lower in both baseline and follow-up PG home compared to PSG and PG lab ($p < 0.01$). No significant difference was found in belt leads. Additionally, 7 baseline and 11 follow-up PG home recordings were accidently deleted before scoring, at two ENT clinics, during an IT system upgrade.

Table 2
Baseline characteristics of cohort N = 53.

	Mean	n	%/± SD	(min-max)
Age, years	5.0		±1.76	(2.6–10.3)
Female sex, n (%)		26	49%	
Male sex, n (%)		27	51%	
Caucasian, n (%)		49	92%	
Baseline oAHI by PSG (n/h)	7.7		±10.0	(0.3–47.0)
Comorbidity, n (%)				
Overweight, n (%)		4	8%	
Obese, n (%)		3	6%	
Asthma		3	5%	
Allergies		3	5%	
Recruitment site				
Hospital n (%)		33	62%	
Practice n (%)		20	38%	
	Median		CI	
Time between				
Baseline PG home − PSG, days	6		(4–9)	(1–53)
Follow-up PG home − PSG, days	4		(2–6)	(1–709)
Surgery − PSG, days	108		(102–117)	(73–219)
Surgery − PG home, days	105		(98–111)	(45–844)

oAHI = obstructive apnea hypopnea index. SD = standard deviation. **CI = 95% confidence interval.** BMI z-score: body mass index standard adjusted for child age and sex defined by the World Health Organization: normal weight ±1 SD, overweight >+1SD, obese >+2SD.

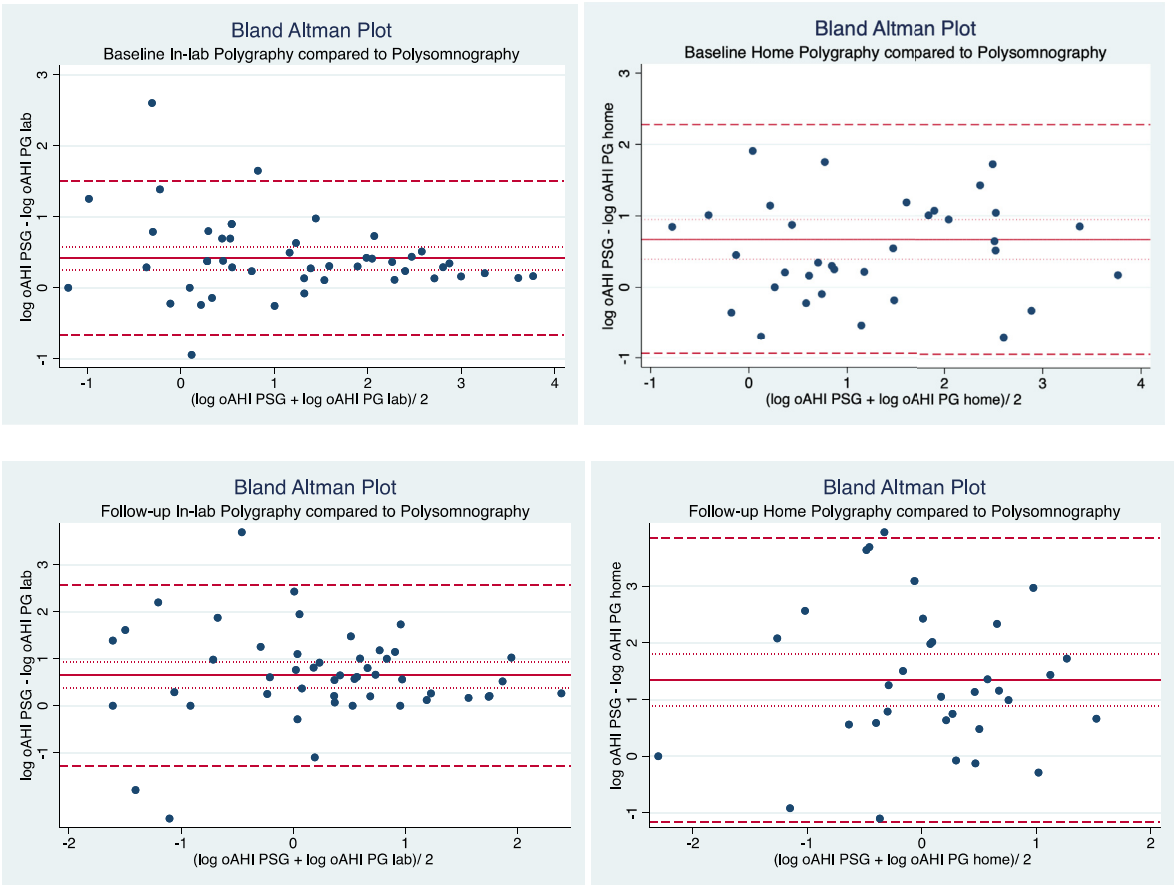


Fig. 1. Bland Altman Plots of PG oAHI versus PSG oAHI
Bland Altman plots on a multiplicative scale showing underestimation of the polygraphy (PG) obstructive apnoea-hypopnea index (oAHI) compared to polysomnography (PSG) oAHI.

3.2. PG compared to PSG

3.2.1. Bland Altman analysis

When plotting the absolute differences against the average, we found the mean difference to increase with the average. On a multiplicative scale we found for the relative differences, the assumptions of variance homogeneity and mean difference homogeneity were satisfied in baseline and follow-up Bland Altman plots (Fig. 1). A systematic underestimation of the PG oAHI compared to PSG was revealed. The relative difference between oAHI PSG and oAHI PG lab was 1.52 CI (1.29–1.79) and the relative difference between oAHI PSG and oAHI PG home was 1.67 CI (1.31–2.13) at baseline.

3.2.2. Respiratory indexes

The AHI and oAHI were significantly lower on PG compared to PSG, but the obstructive apnea (oAI) indexes were not significantly different at baseline. The hypopnea indexes (HI) for PG and PSG were significantly different with p values < 0.001, but no significant difference was found between HI for PG home and PG lab (baseline p = 0.4162, follow-up p = 0.0563). The respiratory indexes and paired comparison of the differences are listed in Table 3.

The total sleep times used to calculate the respiratory indexes were significantly longer in the PG lab recordings compared to the same night PSG recordings. The difference between the PG lab and PG home estimated TST duration was not significant (Table 3).

3.2.3. Performance of PG

Sensitivity, specificity, positive predictive and negative predictive values for the diagnostic cut off oAHI ≥2 or aAI≥1 and treatment cut-off oAHI≥5 are listed in Table 4.

Receiver operating characteristic (ROC) curves giving a graphic representation of sensitivity and specificity of PG oAHI tested against gold standard PSG are shown in Fig. 2.

Based on the difference ratios between PG and PSG, the corresponding treatment cut-off would be between 3.0 and 3.3 for PG home and PG lab. Using the oAHI≥3 cut-off for PG yielded a sensitivity of 87% (60%–98%) and specificity of 90% (67%–99%) for PG home, and sensitivity of 90% (68.3%–99%) and specificity of 83% (64%–94%) for PG lab. The AUCs for the oAHI≥3 treatment cut-off AUCs were 0.92 for PG home and 0.95 for PG lab. Thus, sensitivity increased with a trade-off decrease in specificity while the AUC remained the same.

The baseline OSA severity determined by PSG and the ability of PG to correctly classify OSA severities, is depicted in a bar chart in Fig. 3.

3.2.4. Intraclass correlation

The baseline oAHI intraclass correlation coefficient (ICC) was 0.86 (CI 0.77–0.92) for PG lab and 0.70 (CI 0.60–0.85) for PG home, compared to PSG oAHI. The ICC between PG lab and PG home oAHI was 0.80 (CI 0.73–0.90). The follow-up oAHI intraclass correlation coefficient (ICC) was 0.68 (CI 0.50–0.81) for PG lab and 0.22 (CI

Table 3
Respiratory indexes from Polysomnography and Respiratory Polygraphy.

	Respiratory Index	Mean	± SE	CI	Different from PSG p-value	Different from PG lab p-value
Baseline	**oAHI (events/hour)**					
	PSG	7.77	±1.29	5.17–10.37		
	PG lab	5.76	±1.11	3.53–7.99	<0.001*	
	PG Home	5.58	±1.33	2.89–8.27	<0.001 *	0.148
	AHI (events/hour)					
	PSG	8.60	±1.32	5.93–11.26		
	PG lab	6.78	±1.23	4.30–9.25	0.002*	
	PG Home	6.01	±1.34	3.29–8.72	<0.001*	0.148
	oAI (events/hour)					
	PSG	2.49	±0.60	1.28–3.69		
	PG lab	2.42	±0.59	1.24–3.60	0.621	
	PG Home	1.68	±0.36	0.94–2.41	0.540	0.948
	oA (n)					
	PSG	21.00	5.16	10.63–31.37		
	PG lab	23.08	5.43	12.18–33.97	0.257	
	PG Home	13.80	3.01	7.72–19.88	0.197	0.173
	TST* (minutes)					
	PSG	8.83	±0.15	8.53 9.14		
	PG lab	9.18	±0.15	8.87 9.49	<0.001*	
	PG Home	8.88	±0.35	8.18 9.58	0.2019	0.448
Follow-up	**oAHI (events/hour)**					
	PSG	3.20	±0.43	2.34–4.06		
	PG lab	1.64	±0.25	1.13–2.15	<0.001*	
	PG Home	0.86	±0.15	0.56–1.16	<0.001*	0.021*
	AHI (events/hour)					
	PSG	3.80	±0.45	2.88 4.71		
	PG lab	2.36	±0.30	1.74 2.96	<0.001*	
	PG Home	1.11	±0.18	0.75 1.47	<0.001*	0.003*
	oAI (events/hour)					
	PSG	0.81	±0.15	0.51–1.10		
	PG lab	0.61	±0.12	0.39–0.83	0.064	
	PG Home	0.27	±0.06	0.15–0.39	0.004*	0.017*
	oA (n)					
	PSG	5.57	0.99	3.58–7.56		
	PG lab	5.48	0.97	3.53–7.43	0.980	
	PG Home	3.57	1.04	1.47–5.67	0.020*	0.003*
	TST* (minutes)					
	PSG	9.07	±0.14	8.79 9.34		
	PG lab	9.42	±0.15	9.11 9.73	0.006*	
	PG Home	8.89	±0.44	8.00 9.78	0.343	0.831

Baseline: PSG n = 49, PG lab n = 49, PG Home n = 36. Follow-up: PSG n = 49, PG lab n = 49, PG Home n = 34. *Total sleep time (TST) in PSG = estimated TST in PG recordings due to no sleep stages available. oAHI: Obstructive apnea-hypopnea index (events/h). AHI: Apnea-hypopnea index (events/h). oAI: Obstructive apnea index. PSG: Polysomnography. PG lab: Respiratory polygraphy performed in a sleep lab. PG home: Respiratory polygraphy performed at home.

Table 4
Accuracy of Home Polygraphy (PG home) and Polygraphy in sleep lab (PG lab) vs Polysomnography (PSG).

Cut off		oAHI≥ 2 or oAI≥1		oAHI≥5	
Baseline					
PG Lab	Sensitivity	77%	(60%–89%)	85%	(62%–97%)
	Specificity	100%	(77%–100%)	100%	(88%–100%)
	PPV	100%	(87%–100%)	100%	(81%–100%)
	NPV	64%	(41%–83%)	91%	(75%–98%)
PG home	Sensitivity	77%	(57%–91%)	60%	(33%–82%)
	Specificity	88%	(47%–100%)	100%	(82%–100%)
	PPV	95%	(76%–100%)	100%	(66%–100%)
	NPV	54%	(25%–81%)	76%	(55%–91%)
Follow-up					
PG Lab	Sensitivity	41%	(22%–60%)	40%	(12%–74%)
	Specificity	96%	(77%–100%)	100%	(91%–100%)
	PPV	92%	(62%–100%)	100%	(40%–100%)
	NPV	57%	(40%–73%)	87%	(73%–95%)
PG home	Sensitivity	10%	(1%–32%)	0%	–
	Specificity	100%	(75%–100%)	100%	–
	PPV	100%	(16%–100%)	100%	–
	NPV	42%	(25%–61%)	0%	–

Baseline N: PSG = 49, PG lab = 49, PG Home = 36.
Follow-up N: PSG = 49, PG lab = 49, PG Home = 34.
oAHI: obstructive apnea-hypopnea index. oAI obstructive apnea index.
Data are given with 95% confidence intervals. - All follow-up PG home oAHI values were below 5, but PSG identified 6/34 children with oAHI≥5.

0.04–0.81) for PG home compared to PSG oAHI. The follow-up oAHI ICC between PG lab and PG home was 0.53 (CI 0.41–0.76).

4. Discussion

4.1. PG compared to PSG

In the present study, we found that baseline PG performed with excellent accuracy compared to PSG with an area under the curve (AUC) of 0.95 for PG lab and 0.92 for PG home at an oAHI≥5 cut-off, and an AUC of 0.82 for PG lab and 0.88 for PG home at an oAHI≥2/oAI≥1 cut-off.

However, due to the different preconditions of PG and PSG for scoring hypopneas and estimating TST, a systematic underestimation of oAHI by PG was identified.

Addressing the first issue of the three major challenges in pediatric PG compared to PSG, we found that obtaining PG of sufficient quality for scoring succeeded in 82% of cases for PG home compared to 95% for PG lab and PSG. This shows that obtaining PG home of acceptable quality is feasible and suggests that PG lab should be considered if PG home fails for baseline assessment. Instructing parents in setting an alarm for regularly checking correct placement of probes during the night, especially the nasal cannula, may further optimize PG home signal quality. In this

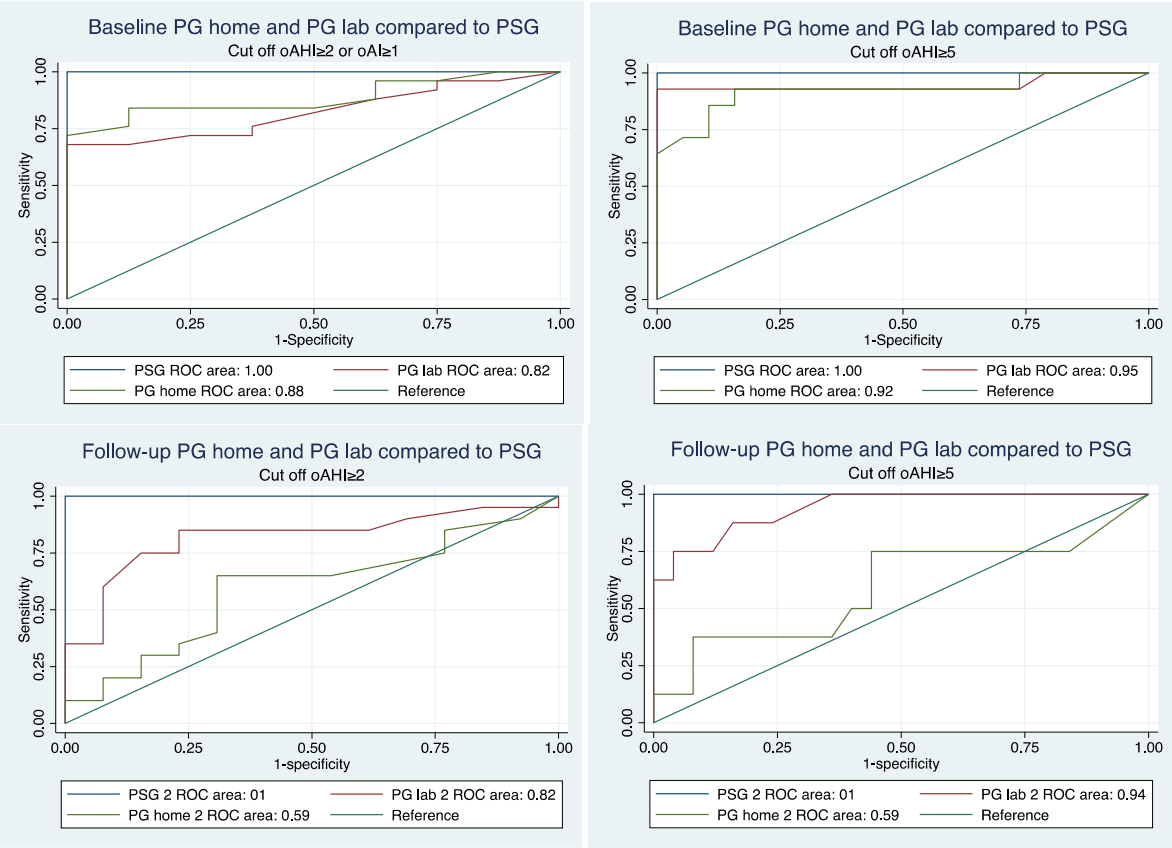


Fig. 2. Receiver Operating Characteristic Curves of Baseline PG lab and PG home against Gold Standard PSG
Receiver Operating Characteristic (ROC) curves with reported area under the curve for oAHI of PG home and PG lab compared to gold standard PSG, at cut-offs oAHI≥2/h or oAI≥1 (right) and oAHI≥5 (left). The graphs represent children who completed both PG lab and PG home recordings.

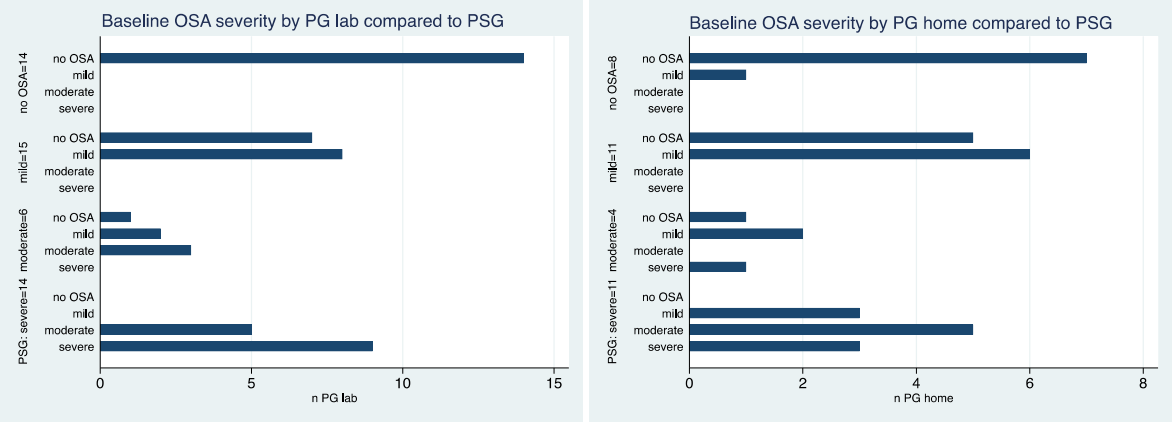


Fig. 3. Baseline OSA severity classification by polysomnography and home respiratory polygraphy
A) Classification of obstructive sleep apnea (OSA) severity by in-lab polygraphy (PG lab) within the polysomnography (PSG) defined severity groups (vertical numbers on the left). B) Classification of OSA severity by home polygraphy (PG home) within the PSG defined severity groups (vertical numbers on the left).

context, it is important to mention that a learning curve in regards of obtaining satisfactory signal quality is to be expected when implementing pediatric PG home [16].

Regarding the second issue of accurately estimating TST, we found that PG lab TST duration was significantly longer than PSG TST. Thus, when hypnogram and video recording are not available the sleep time estimated on PG was significantly longer than for the same night PSG recording. The scoring rules for obstructive apnoeas do not differ between PG and PSG and, interestingly, the apnoea index did not differ significantly between PG and PSG

despite a significant difference in the TST used for calculating the indexes (Table 3). This is consistent with previous findings [8,17] and indicates that the systematic underestimation of PG lab oAHI was a result of missed hypopneas causing arousals without desaturation and the difference in TST was of less significance.

This leads to the third issue of scoring hypopneas associated with arousals without oxygen desaturation (missed hypopneas). We found that both PG lab and PG home systematically underestimated the number of hypopneas and thus underestimated the oAHI and AHI. Despite this, we found acceptable baseline sensitivity of 77% for both PG lab and PG home and high specificity of 100% for PG lab and 88% for PG home (cut-off oAHI≥2/oAI≥1), which shows that PG is a useful tool for detecting OSA. Lowering cut-off to AHI≥1 as done in some studies, would have increased sensitivity, however, we used the first OSA definition stated by the ERS Task Force [14]. ROC analysis provides an overall estimate of the discriminatory power of a continuous test. The baseline AUCs were good for predefined cut-off oAHI≥2/h (baseline PG home AUC = 0.88, PG lab 0.82) and excellent for the cut-off oAHI≥5/h (baseline PG home AUC = 0.92, PG lab 0.95). Using analyses for detecting cut-offs generating optimal AUC values would have further increased the reported AUCs of the study however, the reported AUCs are rooted in the clinically relevant cut-offs corresponding to the ERS Task Force recommendations. Tan et al. conducted a comparison of PSG and PG lab by a similar method as used in this study [8]. They reported sensitivity of 82.5%, specificity of 90% and an AUC of 0.86 for AHI≥1. For AHI≥5 they found sensitivity of 62.5%, specificity of 100% and an AUC of 0.81. Scalzitti et al. included both PG lab and PG home and reported sensitivity of 81% and specificity of 60% for PG lab, and sensitivity of 70% and specificity of 43% for PG home for an AHI>1 [9]. Alonso-Alvarez et al. reported an AUC of 95.5% (90.6%–100%) for PG lab and 93.5% (86.8%–100%) for PG home at an oAHI≥3 cut-off [12]. Although these studies utilized different respiratory cut-off indexes, it seems that the accuracy measured in the present study, is in agreement with previous findings. Tan et al. and Scalzitti et al. also found a statistically significant difference between PG AHI and PSG AHI, however, Alonso-Alvarez did not find a significant difference in oAHI [8,9,12]. Alonso-Alvarez et al. compared the total counts of respiratory events instead of indexes despite mean TST was longer for PG home than for PSG, and they used a general linear model for repeat measurement for statistical comparison instead of a paired model [12], which may have contributed to the different findings.

Baseline intraclass correlation coefficients showed good agreement between PG lab and PSG (ICC = 0.86) and moderate agreement for PG home (ICC = 0.70). The lower ICC for PG home are most likely attributed to night-to-night variability, and to measurement error due to lower oximetry quality in PG home resulting in more sequences with invalid data. The median time between PSG and PG home was 6 for at baseline and 4 at follow-up. Performing the two recordings without any time lapse may have decreased any night-to-night variability, however this was not practically feasible to achieve in all patients. The difference between PG home and PG lab are likely attributed to night-to- night variability, and perhaps the decreased PG home quality of oximetry leads, to some extent.

When using the childhood OSA severity classifications, the underestimated PG oAHI caused slightly less than half the children with mild OSA, to be misclassified as “no OSA” by PG., but all children without OSA, except for one PG home, were correctly classified. Thus, PG classification of mild and moderate OSA was somewhat challenged due to oAHI underestimation (Fig. 3). To compensate for this, a lower cut-off oAHI should be considered in PG. The log transformed Bland-Altman plot satisfied the assumptions of variance homogeneity and mean difference homogeneity which allowed us to make inference on the relative differences. The ratio for PSG oAHI and PG lab oAHI derived from the same night indicates that a factor between the values 1.5 (CI: 1.3–1.8) at baseline and a factor of 1.9 (CI: 1.4–2.5) at follow-up could be used when interpreting the PG oAHI. Previous studies have reported improved agreement between PG and PSG when lowering the cut-off for the corresponding PG AHI [12,17].

Nevertheless, a full night PSG may be required in children with mild or no OSA on PG, when the clinical picture is highly suggestive of OSA.

4.2. Clinical value of PG

The role of PSG is to avoid unnecessary or ineffective surgery in children with primarily nonobstructive events and to confirm the presence of obstructive events that would benefit from surgery. The present study shows that PG indicating OSA has a positive predictive value of 95%–100%. Caregiver reports of snoring, witnessed apnoeas, or other nocturnal symptoms are unreliable if the caregiver does not directly observe the child while sleeping or observes the child in only the early evening. Objective information obtained from PG may help detect OSA, that may otherwise have been overlooked and could be improved after surgical treatment.

As for clinical settings where PSG is not feasible, implementing PG lab or PG home for baseline assessment in children is recommended as a second choice.

For providing a baseline oAHI to assess the risk of complications in patients with severe OSA, the PG lab correctly assessed all severe OSA patients at baseline, whilst PG home assessed 3/4 correctly.

As the 90th percentile for AHI in healthy children in this age group ranges up to 3.2, there is a considerable overlap with the AHI of mild OSA [14]. Accordingly, the AHI cannot stand alone in the decision on when to start treatment in children with mild OSA. These children are only recommended treatment if clinical symptoms or OSA related morbidities are present [14]. Therefore, in mild OSA, the clinical consequences of a false positive PG result are less pronounced than of a false negative result. Conversely, there is consensus that children with oAHI≥5 should be treated for OSA irrespective of the presence of morbidity. As for the oAHI≥5 treatment cut-off, PG lab misclassified 3 children (1 as no OSA and 2 as mild OSA) and PG home misclassified 6 children (5 as mild OSA and 1 child in the moderate category as not having OSA). Thus, there is a slight risk of an otherwise asymptomatic child being falsely classified below the treatment cut-off. Importantly, no children were incorrectly diagnosed as moderate/severe by PG and therefore no children would have received unnecessary surgery based on oAHI exceeding the treatment cut-off of 5.

The AASM does not recommend PG for screening in asymptomatic populations in adults [18]. At follow-up we found very high specificity, which was accompanied with a critical decrease in sensitivity. This may be explained by the lower levels of follow-up oAHIs being more sensitive to measurement error. The low follow-up sensitivity of both PG home and PG lab in this study, suggests that screening in asymptomatic populations is not justified in children either. In children with suspicion of persistent OSA, a

positive PG result will support the decision for further OSA treatment, whereas a negative result should be verified by PSG to rule out any persistent OSA. As for obtaining an oAHI for comparison of baseline and follow-up outcome to assess treatment effect, that would require both recordings to be of the same modality (PG or PSG).

Using the oAHI≥3 treatment cut-off increased sensitivity while maintaining the same AUC. This increases the utility of PG but may be accompanied with a slightly increased risk of overestimating OSA severity. Utilizing this ratio for narrowing the gap between PG oAHI and PSG oAHI due to missed hypopneas should be further investigated in a different and large sample with a wide range of oAHI.

4.3. Strengths and limitations

Unfortunately, a total of 18 PG home recordings were accidently deleted before assessment, but the number of included patients well surpassed the calculated sample size despite this.

The prospective study design with blinded respiratory scoring by one investigator was a strength. This prevented inter-rater bias which may be substantial even among experienced scorers [19]. Using the same sensors on the same night provided optimal conditions for comparing PSG to PG lab by eliminating night-to-night variability and any bias derived from using different sensors. By adding PG home using same sensors and software, we also provided valid insight on PG performance in a realistic clinical setting. By comparing PG home to PG lab, we found no significant difference in oAHI indicating that the baseline night-to-night variability was of less consequence than the significant PG to PSG oAHI difference measured on the same night.

Utilizing thermistor in PSG resulted in less artifacts due to lost airflow signal and added an extra dimension for distinguishing hypopneas from obstructive apnoeas, which was not included in the PG. This may have caused underestimation of the obstructive apnoea index for PG, as some apparent obstructive apnoeas would have been identified as hypopneas by the thermistor, but the difference was not significant.

The 4 h of artefact free recording as minimum criterion is an arbitrary criterion, as no evidence based recommendations for minimum recording time and quality currently exists. The proportion of different sleep stages or sleep in different positions could have differed in the comparison of PG to PSG and in the comparison of baseline and follow-up recordings. This could have affected oAHI as more events are often seen in REM and in supine position. Although this could be problematic in short recordings, the mean recording/sleep time were of reasonable duration (Table 3).

The study population consisted of children with low comorbidity and high clinical suspicion of OSA. Thus, inference can only be made for this patient group. Selection bias in terms of social class and caregiver self-efficacy towards performing an unattended PG home and supporting a child through a PSG in a sleep lab, may have occurred. Including all referred children instead of confining to children eligible for surgery, could have added information on PG performance in identifying children who do not have clear clinical OSA manifestations.

5. Conclusions

We found PG to be a feasible and valid tool for diagnosing OSA in children aged 2–10 years without underlying medical conditions and with clinical suspicion of OSA. Despite systematically underestimating oAHI due to missed hypopneas, baseline PG performed with acceptable sensitivity and excellent specificity compared to gold standard PSG. Meticulous attention to signal quality and a lower oAHI cut-off in PG should be considered to further optimize PG performance.

A PG lab should be performed in case PG home fails. In case of discrepancies between PG results and clinical suspicion, a PSG should be performed. Routine follow-up PG in asymptomatic children is not recommended due to low sensitivity. However, PG lab may be used for identifying persistent OSA in symptomatic children after treatment.

CRediT authorship contribution statement

Tina Kissow Lildal: Conceptualization, Methodology, Formal analysis, Investigation, Writing – original draft, Visualization, Funding acquisition, Project administration. **An Boudewyns:** Conceptualization, Writing – review & editing, Visualization, Supervision. **Konstantinos Kamperis:** Conceptualization, Resources, Formal analysis, Writing – review & editing, Project administration. **Søren Rittig:** Conceptualization, Resources, Visualization, Writing – review & editing, Supervision, Project administration. **Jannik B. Bertelsen:** Writing – review & editing, Supervision. **Marit Otto:** Conceptualization, Resources, Writing – review & editing. **Ole Nørregaard:** Conceptualization, Resources, Methodology. **Therese Ovesen:** Conceptualization, Resources, Visualization, Writing – review & editing, Supervision, Project administration.

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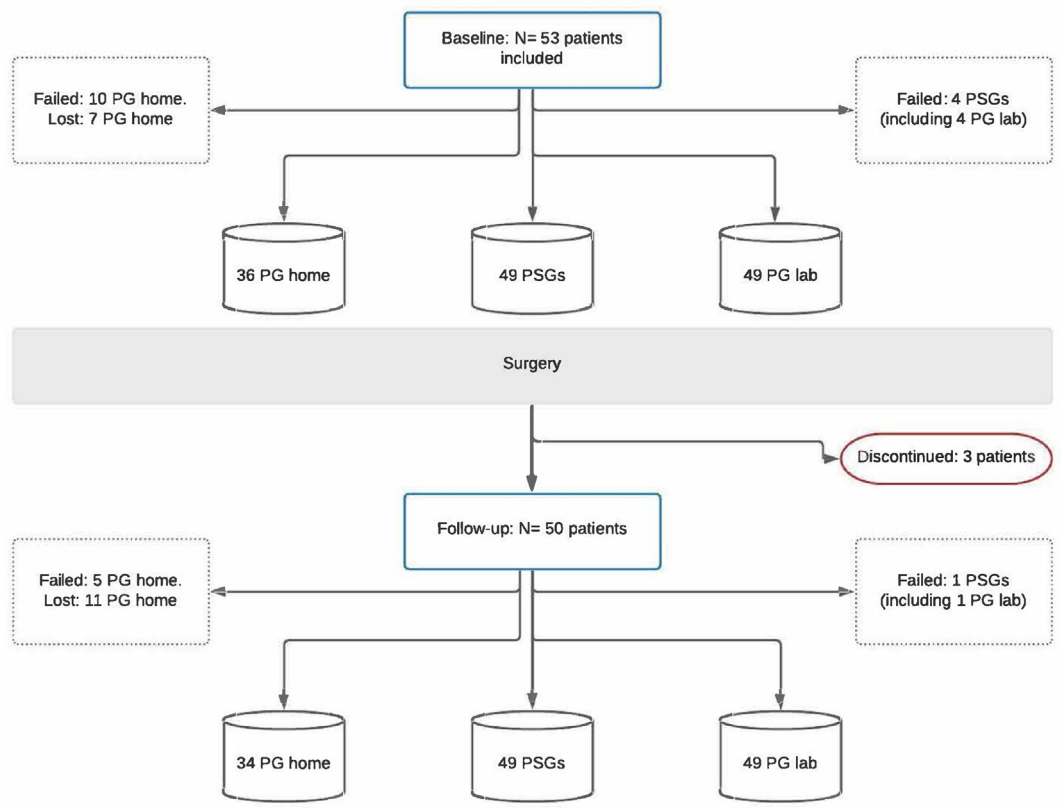
Appendix 1. Flow Chart of included children and recordings



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