Comparison of EEG Signal Characteristics Between Polysomnography and Self Applied Somnography Setup in a Pediatric Cohort

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ABSTRACT We aimed to investigate differences in the electroencephalography (EEG) signal characteristics recorded with a type II polysomnography (PSG) setup including the American Academy of Sleep Medicine recommended EEG montage and Self Applied Somnography (SAS) setup. The PSG and SAS monitoring were simultaneously performed in a pediatric cohort (n = 111) with Nox A1 equipment (Nox Medical, Reykjavik, Iceland). The PSG channels F4-M1 and F3-M2 were compared to the SAS channels AF4-E3E4 and AF3-E3E4. The analyses were conducted separately in each sleep stage. The amplitude levels were compared by investigating envelope curves of each epoch and the frequency content by investigating the power spectrums obtained with Welch’s method. The EEG spectral morphology was similar between SAS and PSG. However, the SAS had consistently lower median amplitudes in all sleep stages compared to the PSG. In Stage N3 (slow-wave sleep), the lower and upper envelope curves had 42.4–47.4% lower median absolute amplitudes. Similarly, the SAS channels had consistently less power in the whole analysed frequency range of 0.3–35 Hz. In conclusion, the results illustrate that the SAS signals have similar EEG spectral morphology but consistently lower amplitudes and less power across the whole EEG frequency range compared to PSG signals. To achieve scoring corresponding to PSG, either the raw SAS signals should be digitally preprocessed or the amplitude threshold for identifying N3 should be lowered from 75 µV to e.g. 45 µV when using SAS instead of PSG. Further clinical validation studies are required to demonstrate scoring reliability using modified scoring rules.

INDEX TERMS Electroencephalography, pediatric sleep disorders, polysomnography, self-applied somnography, sleep staging, spectral analysis.

I. INTRODUCTION

Sleep disorders form a growing global health problem and cause significant economical costs [1]. One of the most
common sleep disorders, obstructive sleep apnea (OSA), is globally affecting over 900 million adults [2] and up to 6% of children [3]. The gold standard in diagnosing sleep disorders, both in adult and pediatric populations, is polysomnography (PSG) conducted at a sleep laboratory. PSG records electroencephalography (EEG), electrooculography (EOG), electromyography (EMG), and cardiorespiratory signals among others. From these, the EEG, EOG, and chin EMG signals are the basis of sleep staging used to provide information on the sleep architecture and the total sleep time [4].

Sleep staging can be considered the cornerstone of sleep medicine. In OSA diagnostics, sleep staging provides invaluable information on the sleep architecture and sleep quality and helps differentiate between various sleep disorders. Accurate sleep staging is especially crucial as the most often used diagnostic parameters (e.g. apnea-hypopnea index, periodic limb movement index, and arousal index) rely on the determined total sleep time. In children, the importance of accurate determination of sleep stages is even greater due to lower parameter thresholds and more strict diagnostic criteria [5], [6]. The main limiting issue with PSG is that it suffers from high costs and limited availability. Type I PSG requires the patient to sleep in an unfamiliar environment which can negatively impact sleep quality [7] and continuous monitoring by trained staff is necessary [8]. Type II PSGs, where the electrodes are placed at a sleep laboratory and the patient sleeps at home, still require approximately a 1-hour setup by sleep laboratory staff [8]. This can be impractical for the patient and the electrodes are prone to be detached [9]. To diagnose OSA in adults, simpler type III polygraphy can be used [4]; however, these cannot identify sleep stages due to the absence of the EEG, EOG, and chin EMG recording montage. Furthermore, type III polygraphy is not accepted internationally for diagnosis of pediatric OSA [4], [10].

To overcome these issues, easily applicable electrode sets and wearable EEG devices have been developed [11]–[13]. For example, sleep staging based on a screen-printed ambulatory electrode set [14] has reached a good correspondence to type I diagnostic PSG [15], [16]. Various consumer devices, such as wristbands, smartwatches, and rings for sleep tracking also exist. However, these often suffer from low concordance compared to PSG especially during disordered sleep [17]. Furthermore, there have also been promising approaches attempting sleep staging with machine learning using only a single channel EEG, electrocardiogram, or photopletysmogram signals to enable a simpler measurement setup compared to PSG [18], [19]. However, sleep staging from surrogate signals to EEG is still not as accurate as EEG-based analysis [19], [20], and cannot yet be adapted to the current clinical protocol. Therefore, EEG is required when the accuracy of sleep staging is crucial.

One potential solution is the recently introduced Self Applied Somnography (SAS) setup (Nox Medical, Reykjavik, Iceland), a simplified EEG montage, developed for self-applicable use in a non-hospital environment (Figure 1).

Compared to traditional EEG montage, it introduces fixed-position frontal montage including ten electrodes and EOG-based referencing without recording the mastoid channels. SAS does not include chin EMG but introduces an EMG derived from the eye electrodes (not yet clinically validated). As the sleep stages are determined by visual inspection of the EEG signals performed by trained sleep technologists or physicians, measurement setups should be comparable to each other to reach a sufficient level of similarity. This is especially crucial for identifying deep sleep with the requirement of fixed peak-to-peak levels of the delta oscillations. The possibility to use the predefined scoring rules of the American Academy of Sleep Medicine (AASM) [4] should be carefully addressed.

In this study, we aim to investigate the differences in the EEG signal characteristics obtained via simultaneous type II PSG and SAS recording in a pediatric cohort. We particularly focus on the differences in amplitude levels and frequency-domain characteristics within different sleep stages when PSG-EEG and SAS-EEG signals are recorded simultaneously.

II. METHODS

A. DATA COLLECTION, DEMOGRAPHICS, AND SLEEP SCORING

The participants in this study were 11–14-year-old Icelandic children from the Icelandic EuroPreval/iFAAM (Integrated Approaches to Food Allergen and Allergy Risk Management) birth cohort study [21]–[23]. The Europrevall study in Iceland [24] included a questionnaire two years before the present study to the parents on the snoring and witnessing of apneas in their children. All children who were reported to snore at least three times a week or had witnessed apneas at least once a week (n = 109) in the questionnaire were invited to participate in the present study. Out of the invited, 55% agreed to participate (n = 60). Moreover,
children without reported snoring or apneas were invited to participate as age- and gender-matched controls \((n = 58)\). In the present study, all participants were investigated as a single group leading to a study population of 118 participants. However, a total of four SAS or PSG recordings had <4 hours of successful recording of EEG, two studies failed, and one participant declined the full usage of data; thus, these participants were excluded from the present study. The final study population included 111 participants (Figure 2). The demographic information of the participants is summarized in Table 1. This study was approved by the Ethical Committee of Landspitali - the National University Hospital of Iceland and the National Bioethics Committee of Iceland (VSN 18-206) and an informed written consent was obtained from a parent and/or legal guardian of all participants.

The participants went through a simultaneous type II PSG and a SAS recording conducted with two separate Nox A1 devices (Nox Medical, Reykjavik, Iceland). The equipment was setup by experienced sleep technologists at the Landspitali University Hospital, Children’s Hospital, Reykjavik, Iceland and the measurements were conducted during a single night at the participants’ home. The PSG included a recording of EEG (F4-M1, C4-M1, O2-M1 signals with the F3-M2, C3-M2, and O1-M2 signals as backup), EOG (E1-M2, and E2-M2), and chin EMG that were used for identifying the sleep stages. The SAS setup included a recording of frontal EEG (AF4, AF3, AF8, and AF7) and EOG (E1, E2, E3, and E4) with placements approximately 1 cm above and below each eye (Figure 1). Patches with two electrodes were used to provide redundancy in case of electrode failure while also allowing the symmetric form and a mechanically stable structure. The EEG channels were referenced to the averaged EOG channel measured below the eyes, i.e. to channel E3E4. This referencing is used to minimize the eye movement artefacts in the EEG.

**B. DATA ANALYSIS**

The recordings were annotated by expert sleep technologists in accordance with the AASM manual, version 2.5 [4] using Noxturnal version 5.1 software (Nox Medical, Reykjavik, Iceland). The sleep stages were identified from the PSG recordings. Only the epochs which were identified as Stages W (wake), N1, N2, N3, or R (rapid eye movement sleep) were included in the analysis. The EEG signals from both montages, AF4-E3E4 and AF3-E3E4 from SAS and F4-M1 and F3-M2 from PSG, were exported from Noxturnal software with a sampling frequency of 200 Hz. No preprocessing of the signals was conducted. All epochs were first extracted in a subject-by-subject manner, and then pooled based on the sleep stage. The total number of analysed epochs was 115312, out of which 3471 were Stage W, 28139 Stage N1, 53204 Stage N3, 22290 Stage R, and 8208 Stage N.
TABLE 2. Comparison of median lower and upper envelope amplitude levels within different sleep stages from PSG (F4-M1 and F3-M2) vs. SAS (AF4-E3E4 and AF3-E3E4).

|         | Lower envelope (µV) | % difference | Upper envelope (µV) | % difference |
|---------|---------------------|--------------|---------------------|--------------|
|         | F4-M1 | AF3-E3E4 | | F4-M1 | AF3-E3E4 | |
| Stage W | -38.9 | -17.9 | 54.1 | 25.7 | 19.2 | 25.3 |
| Stage N1 | -15.9 | -21.6 | -35.7 | 32.4 | 3.0 | 90.7 |
| Stage N2 | -25.6 | -19.3 | 24.6 | 29.5 | 9.0 | 69.4 |
| Stage N3 | -39.9 | -21.6 | 46.0 | 34.0 | 17.9 | 47.3 |
| Stage R | -24.1 | -19.5 | 19.3 | 26.7 | 7.3 | 72.7 |
| Average | -28.9 | -20.0 | 21.6 | 29.6 | 11.3 | 61.1 |

|         | F3-M2 | AF3-E3E4 | % difference | F3-M2 | AF3-E3E4 | % difference |
|---------|-------|----------|--------------|-------|----------|--------------|
| Stage W | -39.4 | -19.5 | 50.5 | 26.5 | 18.8 | 28.8 |
| Stage N1 | -18.9 | -19.0 | -0.01 | 30.1 | 6.1 | 79.7 |
| Stage N2 | -26.2 | -21.1 | 19.4 | 28.8 | 9.5 | 66.9 |
| Stage N3 | -40.1 | -23.4 | 41.7 | 32.5 | 17.4 | 46.6 |
| Stage R | -23.6 | -19.6 | 16.9 | 27.0 | 9.0 | 66.8 |
| Average | -29.6 | -20.5 | 25.6 | 29.0 | 12.2 | 57.8 |

Median lower envelope and upper envelope amplitudes were determined from each epoch without removing artefacts. The percentage difference is computed comparing how much higher the PSG voltages are than the SAS voltages. Negative percentages indicate that SAS amplitudes are higher than PSG amplitudes. The statistical significance of the differences between SAS and PSG upper and lower envelope amplitude-distributions were evaluated using Wilcoxon’s rank-sum test. Statistical significance was set to $p < 0.001$ (bolded in the table). Abbreviations: PSG = polysomnography, SAS = Self Applied Somnography, W = wake, R = rapid eye movement.

For amplitude level analyses, upper and lower envelope curves of each epoch were computed using a 50-point cubic spline interpolation between detected minimums and maximums (Figure 3). Envelopes were analysed to identify possible voltage drift and amplitude levels in all sleep stages, as only Stage N3 has a defined peak-to-peak threshold to the AASM sleep scoring manual [4]. Subsequently, the mean values of the lower and upper envelopes were computed. This procedure was performed similarly for all analysed signals. After computing the means, the median and the distribution of means of upper and lower envelopes were determined. These were compared between PSG and SAS and the statistical significance was determined by Wilcoxon’s rank-sum test. Additionally, the upper and lower envelope amplitudes were investigated via box plots to better visualize the differences in the morphology of the distributions. No variables were assumed to be normally distributed. As the sample sizes (i.e. number of epochs) were large, the threshold for statistical significance was chosen to be 0.001. All the analyses were conducted using Matlab (version 2018b; The MathWorks, Natick, MA, USA).

In addition to amplitude analyses, the frequency content of each epoch was determined. The power spectrums were computed using Welch’s method. The analysed frequency range was 0.3–35 Hz [4], with 50% overlapping of the subsequent partitions, Hamming window, and window size of 1125 points. Before computing the spectrums, each epoch was normalized by subtracting the mean and dividing the values with the standard deviation of the corresponding signal. After the computation of spectrums, the median power spectrum of each signal within each sleep stage was determined.

Moreover, based on preliminary results, the frequency band 10 – 15 Hz was further analyzed to investigate a shift in alpha-band peak power between PSG and SAS. Within this frequency range, the peak power frequency was determined for all Stage N2 and Stage N3 epochs from all recordings. From these, the empirical cumulative distributions were determined and the statistical differences between PSG and SAS was further evaluated using Kolmogorov-Smirnov test.

### III. RESULTS

#### A. AMPLITUDE COMPARISON

The SAS channels AF4-E3E4 and AF3-E3E4 showed consistently lower median amplitudes based on envelope-means in all sleep stages compared to corresponding PSG channels F4-M1 and F3-M2, respectively (Table 2). The only exception was the median lower envelope of SAS in Stage N1 for AF4-E3E4. In addition, the upper and lower envelopes of SAS channels had a broader or similar interquartile range compared to interquartile ranges of PSG envelopes in Stages N2, N3, and R (Figures 4 and 5). In Stages W and N1, the interquartile ranges were broader in PSG channels, except in F3-M2 in Stage N1 (Figures 4 and 5).

#### B. SPECTRAL ANALYSIS

In frequency-domain comparison, the overall spectral morphology was similar between PSG and SAS. However, a substantial difference in absolute and relative power content was observed between the median power spectrums (Figure 6). F4-M1 and F3-M2 channels had consistently higher powers in the whole analysed frequency range (Figure 6). In Stages N2 and N3, a small but systematic and statistically significant ($p < 0.001$) shift in alpha-band peak power towards lower frequencies was observed in both SAS channels compared to PSG channels (Figure 7). In Stage N2, the median alpha-band peak power frequency was 11.84 Hz and 11.92 Hz for F3-M2 and F4-M1, respectively. In the SAS channels,
the median frequencies were 11.43 Hz and 11.41 Hz for AF4-E3E4 and AF3-E3E4, respectively. In Stage N3, the frequencies were 11.56 Hz for F4-M1, 11.54 Hz for F3-M2, 11.28 Hz for AF4-E3E4, and 11.27 Hz for AF3-E3E4. Only negligible differences between right and left channels were observed in amplitude or frequency-domain comparisons between AF4-E3E4 and AF3-E3E4 or F4-M1 and F3-M2, except during Stage W (Table 2, Figure 6, and Figure 7).

**IV. DISCUSSION**

In the present study, we investigated the differences in EEG signal characteristics between type II PSG and a Self Applied Somnography (SAS) setup. We found consistently lower EEG signal amplitudes when EEG was measured using the SAS setup. The amplitudes of SAS-EEG signals were over 40% lower in median within all sleep stages. Moreover, the frontal EEG channels of PSG showed substantially higher power in the whole clinical frequency range compared to the SAS setup. These results imply that preprocessing of the raw EEG signals, relying on the SAS setup, is required prior to manual sleep staging.

The amplitudes of the SAS-EEG signals were consistently lower than those of the PSG-EEG. The most likely explanation is the different referencing of the signals. The locations of the electrodes used for the EOG-based referencing (channel E3E4) differ from the traditional mastoid electrodes (M1 or M2). It results in similar EEG waveforms but different

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**FIGURE 4.** Comparison of minimum and maximum amplitude levels in different sleep stages between F4-M1 (PSG) and AF4-E3E4 (SAS). The red line in the box indicates the median value, edges of the box represent the interquartile range and whiskers indicate 99.3% coverage of the data. Extreme outliers are not presented. Abbreviations: PSG = polysomnography, SAS = Self Applied Somnography, W = wake, R = rapid eye movement.
absolute characteristics. Moreover, the electrodes AF4 and AF3 from the fixed montage are also located close to the actual F4 and F3 placements but not in the same exact locations, which may have a minor additional effect. Furthermore, the SAS relies on self-adhesive electrodes whereas the EEG electrodes used for PSG have an additional conducting paste. This can lead to differing skin electrode impedances and may have a minor effect on the obtained results.

The observed differences in the raw EEG signal characteristics may pose a challenge in detecting Stage N3 due to differences in delta-band power. Stage N3 is characterized by delta oscillations located in 0.5–2 Hz frequencies [4]. These oscillations have to reach a fixed 75 µV peak-to-peak amplitude to allow the scorer to differentiate the Stage N3 from Stage N2 [4]. Stage N2 is characterized by theta-frequencies, sleep spindles, and K-complexes located around 9–16 Hz frequency band [4], [25], but may also have simultaneous delta-oscillation with lower peak-to-peak amplitudes compared to Stage N3 [26]. The present results show that the SAS setup produces significantly lower amplitudes within Stage N3 compared to type II PSG. Aside from differences in the spectral power, the only noticeable difference between the frequency content of SAS and PSG was a small but systematic shift in alpha-band peak power frequency in Stages N2 and N3 (Figure 6 and Figure 7). The EOG-referencing and the placement of the SAS electrodes could contribute to the shift in alpha-peak power. However,
FIGURE 6. Logarithmic median power spectral density (PSD) estimates for channels AF4-E3E4 (SAS), AF3-E3E4 (SAS), F4-M1 (PSG), and F3-M2 (PSG) in all sleep stages and the relative difference in spectral power content between SAS and PSG. Abbreviations: PSG = polysomnography, SAS = Self Applied Somnography, W = wake, R = rapid eye movement.
FIGURE 7. The empirical cumulative distributions for alpha-band peak power frequencies between 10 – 15 Hz for channels AF4-E3E4 (SAS), AF3-E3E4 (SAS), F4-M1 (PSG), and F3-M2 (PSG) in sleep stages N2 and N3. Abbreviations: PSG = polysomnography, SAS = Self Applied Somnography.

the alpha shift was small, ranging from 0.27 to 0.49 Hz, and thus should not affect the visual scoring of Stage N2. More studies are warranted to verify the scoring concordance between PSG and SAS.

It is noteworthy, that the observed differences in the spectral and amplitude content between PSG and SAS are possible to overcome. As seen in the median spectrums, the spectral morphology is highly similar between SAS and PSG. Thus, even though amplitude levels differ, the frequency characteristics produced by the SAS setup are comparable to PSG. Besides the decreased delta-power in SAS channels, the only observable difference was the lower alpha-band peak power frequency in Stages N2 and N3. However, the observed shift was minor, and should not affect the identification of sleep spindles and other alpha-band characteristics of EEG. This indicates that for sleep staging, similar scoring as with PSG may be achieved by digitally preprocessing (e.g. amplifying certain frequency bands) the raw SAS EEG signals or lowering the amplitude threshold required for scoring Stage N3 when analyzing EEG measured with SAS. However, the studied EEG signal characteristics do not provide information on individual epoch-by-epoch differences but rather on the averages. Therefore, further studies are required to validate how well a manual scorer succeeds in identifying different sleep stages from SAS recordings compared to PSG.

The present study of EEG signal characteristics of SAS was conducted in a pediatric population aged 11–14 years; thus, the results cannot be generalized to other populations. Measuring the EEG with a self-applicable electrode set has different practical issues in children compared to adults. For instance, the electrode placement with a fixed electrode set differs due to the differing facial size. Therefore, further studies are required to confirm the technical quality of SAS recording in an adult population as well as in a pediatric population with a broader age range. In addition, the dataset comprised two distinct groups: snorers and the control group. No comparison between these groups was conducted. However, as this was an epoch-by-epoch technical signal quality comparison, this should not affect the obtained results.

A further limitation of the present study is the slight positive bias caused by the electrode placement protocol. The SAS was placed together with the normal EEG setup by the sleep technicians. Thus, the study does not provide information on the self-application success rate. However, setup by sleep technicians was necessary to obtain a reliable comparison of the technical quality of the signals in a best-case scenario without unnecessary confounding factors. The reliability and feasibility of the use of the SAS on children in the home setting with parent setup will be a future research topic. Moreover, this study did not compare artefact tendency between EEG recordings from SAS and PSG. One of the most
common artefact in overnight EEG recordings is the sweat artefact. Sweat artefacts are visible in the delta-range and they mimic the slow-wave oscillations due to different impedance characteristics of the skin-electrode interface when sweat is present [27]–[29]. They are a common problem especially in sleep apnea populations as the patients often suffer from excessive nocturnal sweating [30], [31]. Sweat artefacts are also related to material selections of the electrodes [30], [31]. As the power in the SAS delta-band did not reach the powers of PSG in any sleep stage, it can be speculated that SAS is as susceptible to sweat artefacts as PSG-EEG. However, sweat artefact analysis was outside the scope of the current paper. Therefore, more detailed studies are warranted to investigate how susceptible the SAS setup is to sweat artefacts and whether the different referencing of the EEG signals affects artefact appearance. Finally, the PSG-EEG and SAS-EEG recordings were conducted with two separate Nox A1 amplifiers which can potentially lead to small synchronization issues. However, these limitations were acknowledged in the study design phase and the methodology was chosen correspondingly such that small differences in synchronization pose no significant issues.

V. CONCLUSION
The EEG signals recorded with the SAS setup had similar spectral morphology but consistently lower amplitudes and less power across the analysed frequency range of 0.3–35 Hz. Therefore, the raw EEG signals should be digitally preprocessed before scoring or the amplitude threshold for identifying Stage N3 from the SAS recording should be lowered. Based on the present data, we propose a cutoff value of 45 µV for peak-to-peak slow-wave oscillation to be used with SAS in pediatric studies when identifying Stage N3 if no additional preprocessing is conducted. However, further studies are required to verify the accuracy of the proposed threshold in different study populations.

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