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Comparison of a single-channel EEG sleep study to polysomnography

Brendan P. Lucey, MD^{1,2,*}, Jennifer S. McLeland¹, Cristina D. Toedebusch¹, Jill Boyd¹, John C. Morris, MD^{1,2,3}, Eric C. Landsness, MD, PhD¹, Kelvin Yamada, MD^{1,2}, and David M. Holtzman, MD^{1,2,3}

¹Department of Neurology, Washington University School of Medicine, St Louis, MO

²Hope Center for Neurological Disorders, Washington University School of Medicine, St Louis, MO

³Knight Alzheimer's Disease Research Center, Washington University School of Medicine, St Louis, MO

Summary

An accurate home sleep study to assess electroencephalography (EEG)-based sleep stages and EEG power would be advantageous for both clinical and research purposes, such as for longitudinal studies measuring changes in sleep stages over time. The purpose of this study was to compare sleep scoring of a single-channel EEG recorded simultaneously on the forehead against attended polysomnography. Participants were recruited from both a clinical sleep center and a longitudinal research study investigating cognitively-normal aging and Alzheimer's disease. Analysis for overall epoch-by-epoch agreement found strong and substantial agreement between the single-channel EEG compared to polysomnography ($\kappa=0.67$). Slow wave activity in the frontal regions was also similar when comparing the single-channel EEG device to polysomnography. As expected, stage N1 showed poor agreement (sensitivity 0.2) due to lack of occipital electrodes. Other sleep parameters such as sleep latency and REM onset latency had decreased agreement. Participants with disrupted sleep consolidation, such as from obstructive sleep apnea, also had poor agreement. We suspect that disagreement in sleep parameters between

*Corresponding author: Address: Campus Box 8111, 660 S. Euclid Avenue, Washington University School of Medicine, St Louis, MO 63110, Phone: 314-362-4342, Fax: 314-747-3813, luceyb@neuro.wustl.edu.

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Critical revision of the manuscript for important intellectual content: B.P.L., J.S.M., C.D.T., J.B., J.C.M., E.C.L., K.Y., D.M.H.

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the single-channel EEG and polysomnography is partially due to altered waveform morphology and/or poorer signal quality in the single-channel derivation. Our results show that single-channel EEG provides comparable results to polysomnography in assessing REM, combined stages N2 and N3 sleep, and several other parameters including frontal slow wave activity. The data establish that single-channel EEG can be a useful research tool.

Keywords

Ambulatory; inter-rater agreement; sleep stage scoring

Introduction

Accurately measuring sleep stages representative of an individual's sleep at home is difficult due to the strengths and limitations of the most common methods for monitoring sleep-wake patterns. Attended polysomnography (PSG) is the gold standard for sleep monitoring. Its strengths include the ability to obtain detailed information on sleep latencies, time in each sleep stage, wake time after sleep onset (WASO), and other sleep parameters, as well as diagnose sleep disorders like obstructive sleep apnea (OSA) and periodic limb movement disorder (PLMD). A significant limitation of PSG is that it requires attached electrodes and other sensors to collect data about normal and abnormal physiologic changes during sleep, including electrodes to monitor brain activity (EEG: electroencephalography), eye movements (EOG: electrooculography), muscle activity (EMG: electromyography), heart rhythm, respiratory effort, airflow through the mouth and nose, and audible snoring. PSG is also performed in a sleep laboratory and the new environment may further disturb an individual's sleep, i.e. the first night effect (Agnew et al., 1966). However, PSG to monitor only sleep stages could be performed with electrodes for EEG, EOG, and chin EMG. Further, full ambulatory PSG with sensors placed by a trained technician and the individual sleeping at home unattended is feasible and has been performed in >2000 subjects in one study (Luca et al., 2015).

Since even limited PSG to monitor only sleep stages may be financial prohibitive, unattended sleep monitoring that included sleep stages and EEG power but did not require sensor placement by a technician would have important clinical and scientific application for longitudinal research studies and clinical trials. Previous attempts to develop such a home-based, forehead-derived single-channel EEG sleep monitoring system either were tested in a laboratory with sensors placed by a sleep technologist (Dyson et al., 1984; Werth et al., 1995) or did not allow for access to the raw data if needed in order to visually review sleep stage scoring (Shambroom et al., 2012).

Sleep staging and spectral power measurement with a single-channel EEG (single EEG) recorded on the forehead presents multiple challenges compared to PSG that may affect our results. First and most obviously, the forehead channel will not detect an alpha rhythm and we predict this will limit assessment of wake and stage N1. Second, waveforms used for sleep staging differ in topographical distribution. K-complexes, for example, are mainly distributed over the medio-frontal leads while delta waves peak both medio-frontally and

occipitally (Happe et al., 2002). Further, sleep spindles have an anterior-posterior bimodal distribution and the single EEG will permit review of anterior sleep spindles only (Werth et al., 1997). Also age-related changes in spindle density, amplitude, and duration (but not frequency) are greatest anteriorly (Martin et al., 2013). Third, there are limitations on the single EEG capacity to assess topographical differences in slow wave activity (SWA) reported in adults (Finelli et al., 2001) or the posterior-to-anterior shift in maximal SWA activity reported during childhood (Kurth et al., 2010). NREM power also undergoes an antero-posterior shift during consecutive NREM sleep periods that cannot be assessed with only a single forehead derivation (Werth et al., 1996). We also predict that measuring SWA during REM will be less accurate than from a central derivation on a PSG due to eye movements.

The purpose of this study was to compare visual sleep stage scoring of PSG to a sleep-monitoring device worn on the forehead that records a multi-night single-channel EEG (Sleep Profiler[®], Advanced Brain Monitoring, Carlsbad, CA). We determined agreement between the single EEG and PSG for multiple sleep parameters including epoch-by-epoch sleep stage scoring, total sleep time, sleep latency (first epoch of N1), rapid eye movement (REM) onset latency, WASO, sleep efficiency (total sleep time/time in bed), time in each sleep stage, and SWA power. We performed between-montage comparison (PSG vs. single frontal EEG derivation) and inter-rater variability of the single-channel EEG sleep scoring compared to PSG. This study was not supported by Advanced Brain Monitoring and the company was not involved in data collection or drafting this manuscript.

Methods

Participants

Twenty-nine participants underwent diagnostic or split-night PSGs with concurrent single EEG recordings. At our center, a diagnostic PSG is an all-night sleep study without the initiation of positive airway pressure (PAP) therapy. A split-night PSG is a sleep study where a decision to start PAP to treat sleep apnea during the first 2 hours of sleep is made based on criteria specified by the ordering sleep medicine physician. Twenty-seven subjects were recruited from patients scheduled for a clinical sleep study at our center. Two cognitively-normal participants were also recruited from the Knight Alzheimer's Disease Research Center at Washington University. The study protocol was approved by the Washington University Institutional Review Board. All subjects provided written informed consent and were compensated for their participation in the study.

Sleep Monitoring

PSG was performed using standard electrode montages (EEG: F3-M2, F4-M1, C3-M2, C4-M1, O1-M2, O2-M1; EOG; chin EMG; electrocardiography (ECG); chest belt; abdominal belt; right leg EMG; left leg EMG; thermistor; pressure transducer airflow) and recorded with Polysmith[®] (Nihon Kohden, Irvine, CA). The PSG data was collected at 200 Hz. For the EEG, the low and high frequency filters were set at 0.53 Hz and 35 Hz, respectively. The single EEG device was worn on the forehead (Figure 1) and recorded at 256 samples/second from 3 frontal sensors placed at approximately AF7, AF8, and Fpz (Sleep Profiler Scoring

Manual, 2015). The electrodes at AF7 and AF8 are labeled Fp1 and Fp2, respectively, by the device software and are referred to as such throughout this paper. Although Fp1-Fp2, Fp1-Fpz, and Fp2-Fpz channels were available for review from the single EEG studies, only the Fp1-Fp2 channel was used for scoring. The single EEG hardware applies a 0.1 Hz low frequency filter and a 67 Hz high frequency filter.

Single EEG recording began simultaneously with the start of PSG. For patients diagnosed with sleep apnea and started on PAP therapy as part of a split-night PSG (9/29 participants), only the diagnostic portion of the study was available for analysis due to concern about interfering with the titration portion of the PSG by wearing both a PAP interface and the single EEG.

A total of 21,266 epochs were recorded. Any epochs from either single EEG or PSG that were unscorable due to movement or electrode artifact were excluded resulting in 19,326 epochs available for analysis. The majority of epochs unscorable due to artifacts were from the single EEG (>95%). Approximately 25% of all single EEG artifacts were due to movement during wake, 20% were due to sweat artifact, and 55% were due to poor electrode contact. Although sleep stages can be auto-scored using a web-based portal with an automated scoring algorithm (Stepnowsky et al., 2013; Popovic et al., 2014), we exported each single EEG record as a European Data Format (EDF) file for visual sleep stage scoring in Polysmith[®] with low and high frequency filters applied at 0.3 Hz and 30 Hz respectively.

Sleep Stage Scoring

All PSGs were scored by two registered sleep polysomnographic technologists (scorer 1 and scorer 2) using standard American Academy of Sleep Medicine (AASM) criteria (Iber, 2014) based on 6 EEG channels, 2 EOG channels, and 1 EMG channel recording. Both scorer 1 and scorer 2 perform monthly assessments through the AASM inter-scorer reliability program and averaged >92% agreement with the gold standard over the preceding year. Sleep stage scoring on the single EEG device used the Fp1-Fp2 channel because the inter-electrode distance is greater than between Fp1-Fpz and Fp2-Fpz (~12.5 cm vs. 6 cm), magnifying waveforms important for sleep stage scoring such as K-complexes and sleep spindles.

Sleep stage scoring on the single EEG was adapted from AASM criteria (Table 1). Figure 2 shows 6-second epochs of selected graphoelements at each sleep stage. Wake was defined as the presence of alpha rhythm and/or eye blinks for >50% of an epoch. Stage N1 was defined by attenuation of the alpha rhythm and/or slow eye movements for >50% of an epoch without the presence of K-complexes or sleep spindles. Stage N2 began at the first sleep spindle or K-complex and ended with arousal or shift to another stage. Following an arousal, epochs not meeting other criteria were scored as stage N1 until the next sleep spindle or K-complex, signifying reemergence of stage N2.

For stage N3, 20% of a single EEG epoch must show delta waves with amplitudes +/- 30 μ V (60 μ V peak-to-peak), rather than +/- 37.5 μ V (75 μ V peak-to-peak) on PSG, due to a narrow 0.1-0.6 Hz band-stop filter that reduces respiration artifact and has the additional effect of attenuating the amplitude of delta waves during slow wave sleep (Sleep Profiler

Scoring Manual, 2015). Further, the frontal location of the single EEG electrodes attenuates delta activity compared to the central region electrodes with referential recording on PSG.

REM sleep was scored based on rapid eye movements and low amplitude mixed frequency EEG. Rapid eye movements were defined as irregular, sharply peaked movements with an initial deflection usually lasting <500 milliseconds. There was no standardized amplitude criteria. When REM sleep followed stage N2, it was usually recognized at the first rapid eye movement. If epochs preceding the first rapid eye movement showed only low amplitude mixed frequency EEG, then they were back-scored as REM until reaching the last sleep spindle or K-complex. When REM followed stage N1 or N3, then REM began at the first rapid eye movement. After an arousal, epochs were still scored as REM if rapid eye movements appeared immediately after the arousal; otherwise, subsequent epochs were scored as stage N1, N2, or N3 if the appropriate criteria above were met after an arousal.

The two expert scorers sequentially scored the single EEG studies and then several weeks later sequentially scored the PSG studies. The study filenames were not randomized or blinded.

Spectral Power Analysis

Three of twenty-nine subjects with PSG and single EEG recordings were excluded from the spectral analysis due to poor quality of the PSG data and one subject was excluded due to reduced sleep time (total N=25). Both PSG and single EEG studies were scored as described above, and sleep stage scoring by scorer 1 was used for the power analysis. As in previous studies (Landsness et al., 2009; Landsness et al., 2011), the EEG signal from both sets of studies were downsampled to 128 Hz for analysis in order to eliminate processing error. Although the single EEG data was already filtered during acquisition with a 0.1-0.6 band-stop filter, we applied a band-pass (2-way least-squares FIR) filter between 0.5 and 40 Hz to both the single EEG and PSG data to maintain uniformity. Spectral analysis was performed in consecutive 6-second epochs (Welch method, Hamming window, no overlap). Artifacts were excluded in a semiautomatic method. Power in the 20-30 Hz and 1-4.5 Hz band for each electrode across all epochs of a recording were displayed. The operator (6th author) then selected a threshold between the 95th and 99.5% threshold of power to remove artefactual epochs. This resulted in less than 4% of all epochs being rejected as artefactual.

Statistics

Epoch-by-epoch agreement between the single EEG and PSG studies was assessed by calculating sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy for all single EEG vs. PSG scorer and sleep stage combinations (Altman and Bland, 1994a; Altman and Bland, 1994b). Pearson's correlation coefficients, Cohen's *kappa* coefficient, and intraclass correlation coefficients (ICC) were calculated for multiple sleep parameters determined by single EEG and PSG to measure the level of entire night agreement. A *kappa* value of 0-0.2 is considered slight agreement, 0.21-0.4 fair agreement, 0.41-0.6 moderate agreement, 0.61-0.8 substantial agreement, >0.8 almost perfect agreement (Landis and Koch, 1977). Intraclass correlation coefficients (ICC) are sensitive to differences in the means of the observations and are a measure of inter-observer

agreement (Fisher, 1954). Finally, correlation coefficients may be misleading about the level of agreement when comparing clinical measures, therefore Bland-Altman plots were generated for all sleep parameters (Bland and Altman, 1986).

All statistical analyses were performed in IBM SPSS Statistics version 22.0 (IBM Co., Armonk, NY). Graphpad Prism version 6.0b for Mac (Graphpad Software, San Diego, CA) was used to generate graphs, linear regression best-fit lines, and Bland-Altman plots.

Results

Demographics

Participant demographics are shown in Table 2. 18/29 participants had either no evidence of sleep-disordered breathing (AHI<5) or mild OSA (AHI 5-15); there were no participants with central sleep apnea. Five participants had >15 periodic limb movements per hour of sleep.

Sleep Stage Scoring Agreement

Scorers 1 and 2 had high levels of agreement when PSG-to-PSG and single EEG-to-single EEG comparisons were made (PSG-to-PSG $\kappa=0.97$; single EEG-to-single EEG $\kappa=0.94$). Compared on an epoch-by-epoch basis, the sensitivities for individual sleep stages were also high between the two scorers for both PSG-to-PSG and single EEG-to-single EEG comparisons (PSG: wake (0.982), stage N1 (0.897), stage N2 (0.987), stage N3 (0.808), and REM (0.988); single EEG: wake (0.918), stage N1 (0.878), stage N2 (0.983), stage N3 (0.838), and REM (0.97). Given the near perfect level of agreement between the two scorers, only analysis from scorer 1 is presented below. All data for scorer 2 is available in the supplement.

For single EEG-to-PSG comparisons, we initially compared stages N2 and N3 separately. Although the level agreement measured by sensitivity was high for stage N2 (0.831), it was low for stage N3 (0.294). We did not expect this very low agreement for stage N3 and attribute this finding to its low frequency in our sample (range 2.3-5.5% of all epochs) leading to large error. Measuring percent agreement for combined stage N2 and N3 produced more consistent high levels of agreement for single EEG-to-PSG comparisons similar to PSG-to-PSG and single EEG-to-single EEG comparisons, therefore all subsequent analysis combined stages N2 and N3.

Single EEG and PSG studies had high sensitivity and specificity for wake, combined stages N2 and N3, and REM (Table 3). Although specificity was >0.95 for stage N1, sensitivity was poor. There was also substantial overall scoring agreement between PSG and single EEG based on a κ of 0.67 for all epochs. When stage N1 was removed from the analysis, κ increased to 0.73.

Sleep Parameters

Although overall and specific sleep stage agreement was high on an epoch-by-epoch basis, we considered the possibility that other sleep parameters may not correlate between single EEG and PSG studies. In order to minimize error that would result from uncorrected

movement and electrode artifact in the single EEG studies, four participants with (x02267) 10% of single EEG epochs unscorable due to artifact were excluded from analysis. For PSG-to-PSG comparisons, all sleep parameters had ICCs of 0.99 between the two scorers. Single EEG-to-single EEG comparison were also found to have ICCs of 0.99 except for REM onset latency (0.76). Total sleep time, time in combined stages N2 and N3, and REM sleep showed high correlations between single EEG and PSG studies (Table 4). In both scatter and Bland-Altman plots, total sleep time (Figure 3A, 3D), combined stages N2+N3 (Figure 5B, 5E), and time in REM (Figure 5C, 5F) all had minimal bias on the Bland-Altman plot with all but 2-3 points located within 2 standard deviations of the mean difference.

In general, sleep parameters determined by sleep-wake and other stage transitions (e.g. sleep latency, sleep efficiency, WASO, time in stage N1) showed poor agreement with the lowest ICCs and r^2 values. These trends were also seen in the scatter and Bland-Altman plots for sleep efficiency (Figure 3B, 3E), WASO (Figure 3C, 3F), sleep latency (Figure 4A, 4C), and time in stage N1 (Figure 5A, 5D). We found that this was most likely due to difficulty identifying transitions between wake and sleep with only the Fp1-Fp2 channel on the single EEG. Review of the Bland-Altman plots for sleep latency and WASO showed that bias between single EEG and PSG studies increased as sleep latency and WASO time increased above 20 minutes and 60 minutes, respectively. These findings are similar to the bias for sleep efficiency <80%.

We conclude that the single EEG most accurately measures sleep parameters associated with consolidated sleep when compared to PSG, but agreement between single EEG and PSG decreases markedly when there are frequent sleep-wake or other sleep stage transitions. Factors that reduce sleep consolidation will decrease single EEG accuracy compared to PSG.

Spectral Power

As was previously reported by Werth et al., 1995, we predicted that the single EEG would show a similar homeostatic decline in slow wave activity (SWA: EEG power 1-4.5 Hz) compared to a frontal electrode from the PSG (F4). To test this hypothesis, we compared SWA between the single EEG and PSG across both the entire night and the first and last NREM sleep periods. Figure 6 shows a representative time course of the artifact-free SWA epochs across the course of the night with the corresponding hypnogram. SWA is shown as a percentage of the mean SWA across the entire night. Both the single EEG and PSG demonstrate similar profiles of SWA across the 8-hour time period. To quantify this change in the average SWA, we compared the average SWA between the single EEG and PSG for the first twenty minutes of the first NREM period and the first twenty minutes of the last NREM period normalized to the mean SWA across the entire night (Figure 7). Twenty-five participants had sufficient artifact-free recordings to analyze during these time periods. There was a significant correlation between both NREM periods and no significant bias. Average SWA also declined $35 \pm 12\%$ and $33 \pm 9\%$ (mean \pm SEM) in the single EEG and PSG recordings respectively over the course of the night and these changes are not significantly different between the two recording types ($p = 0.39$, $n = 25$, paired t-test).

Discussion

In the present study, we observed strong and substantial epoch-by-epoch agreement between the single-channel EEG and PSG scored by two expert sleep technologists. Total sleep time, time in sleep stages N2 and N3, and time in REM also showed strong agreement. Frequent sleep-wake and other stage transitions resulted in a marked decrease in agreement for both epoch-by-epoch and overall sleep parameter agreement. For instance, increased sleep fragmentation such as from sleep apnea lead to poorer agreement of several parameters, such as sleep latency, sleep efficiency, and time in stage N1. Finally, SWA in the frontal regions was similar between the single EEG and PSG as expected.

Prior work in sleep staging a single-channel EEG recorded from below the hairline focused on both automated scoring algorithms (Popovic et al., 2014) and visual scoring (Dyson et al., 1984; Werth et al., 1995). When compared to these studies, we found a lower level of overall agreement for epoch-by-epoch sleep stage scoring. For example, in an analysis of a scoring algorithm Popovic et al. found *kappa* coefficients ranging from 0.71-0.76 for all 5 sleep stages. Using visual scoring of a below the hairline electrode that approximated Fp2 referenced to A1, Dyson et al. reported an overall *kappa* of 0.8. Notably, these studies referenced a below the hairline frontal electrode to either A1 or A2 and sensors were placed and monitored by sleep technologists. Stepnowsky et al. used bipolar electroocular electrodes to compare sleep stage scoring to PSG and found overall agreement of *kappa*=0.62, a finding comparable to our study. Further, all of the studies above except for Stepnowsky et al. included only healthy participants without sleep disorders. Our lower overall agreement may be due to participants having a mix of sleep disorders. Since the single EEG signal was unmonitored and had greater artifact than PSG, differences in signal quality may also explain discrepancies between our study and previous work. Despite application of single EEG electrodes by a sleep technologist at the start of the night, we found that ~55% of all artifact on the single EEG studies was due to poor electrode contact. We predict there will be greater electrode artifact when the single EEG is applied by patients or research participants at home unattended.

For each sleep parameter, Bland-Altman plots showed 0-3 points outside the limits of 95% limits of agreement or 2 standard deviations. For specific subjects, the difference between single EEG and PSG measures is considerable. Total sleep time, for example, had two subjects with >100 minute discrepancy between the sleep EEG and PSG (Figure 3D). All of these extreme outliers were limited to 8 participants; 17/25 participants had sleep parameters that were consistently within the limits of agreement. Review of these 8 participants' sleep scoring and parameters found several commonalities. First, 5/8 participants had sleep fragmentation due to either moderate-to-severe OSA (N=3, all with AHI>24) or severe periodic limb movements during sleep (N=1) or both (N=1). Second, 3/8 participants had periods of sleep fragmentation during the night, particularly at sleep onset, with frequent transitions from wake and stage N1 that were frequently not scored correctly on the sleep EEG due to lack of occipital electrodes to record alpha activity. There were also periods of REM sleep scored as wake in these participants due to lack of rapid eye movements on the single EEG. In this situation, scoring REM sleep on the single EEG is difficult without a chin EMG.

Compared to PSG-to-PSG inter-rater scoring agreement, the single EEG demonstrated excellent agreement to PSG despite having only a single-EEG channel. In a study of >2500 experienced sleep scorers participating in the AASM inter-scorer reliability program comparing PSG-to-PSG scoring, overall inter-scorer agreement was 82.6%. Stages N1 and N3 had the lowest percent agreements, 63% and 67.4% respectively (Rosenberg and Hout, 2013). Except for stages N1 and N3, our two expert scorers achieved similar levels of agreement for a single-channel EEG compared to PSG as the inter-scorer PSG-to-PSG comparisons. Further, the single EEG sensitivities for individual sleep stages compared favorably to actigraphy sensitivities for determining wake vs. sleep on PSG (Kushida et al., 2001).

Although the single EEG-to-PSG agreement was high, a major limitation of our study is generalizability. The high agreement between single EEG and PSG scoring involved two very experienced polysomnographic technologists at our sleep center who had almost perfect agreement for both PSG-to-PSG and single EEG-to-single EEG comparisons. This study provides a baseline to compare single EEG sleep stage scoring recorded from participants in clinical or longitudinal research studies to PSGs scored by the same experts scorers. Other sleep centers will need to perform their own comparison studies if they visually score single EEG studies. We predict that a study involving a large number of expert scorers from a variety of sleep centers and experience levels would lead to lower levels of agreement.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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A



B

**Figure 1.**

A. Sleep Profiler with electrode strip flipped out 180 degrees. Each electrode location is labeled (Fz, Fp1, and Fp2) B. Sleep Profiler in use. (Used with permission from Advanced Brain Monitoring, Carlsbad, CA).

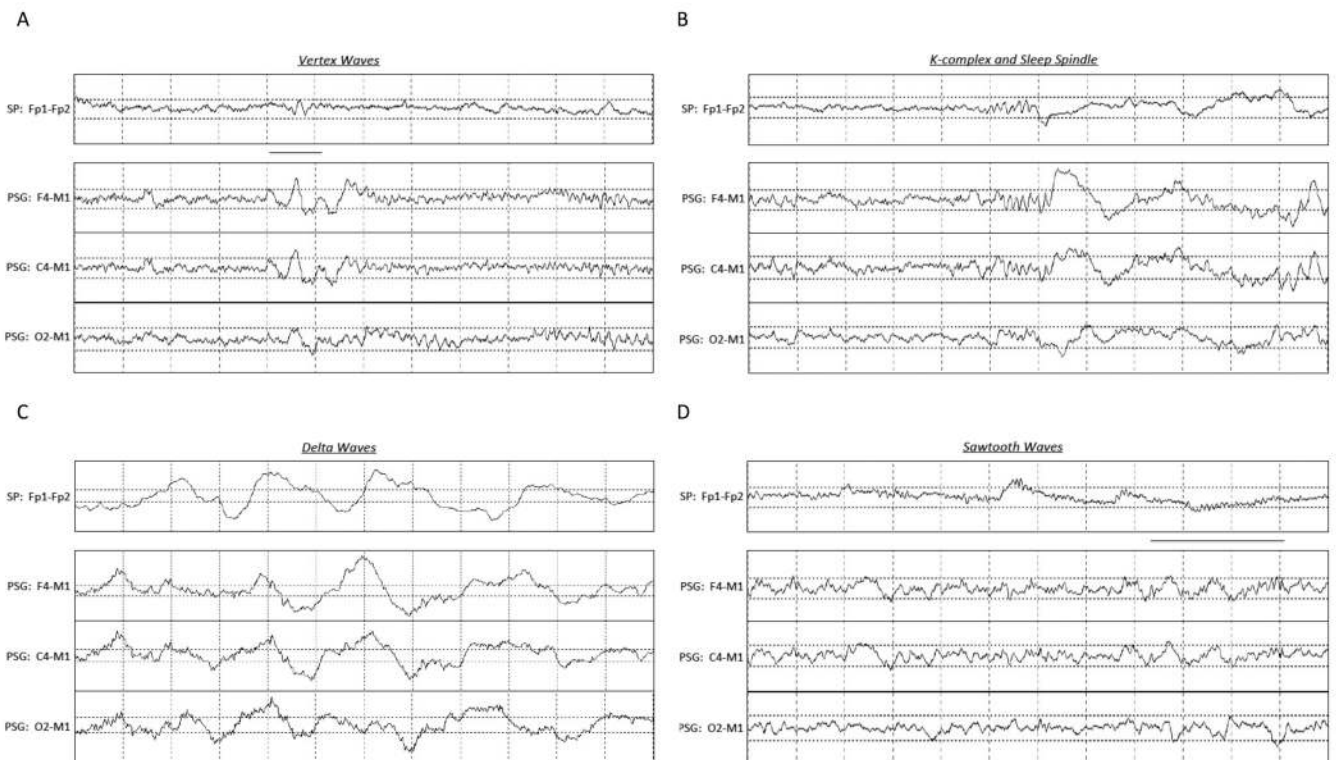


Figure 2. 6-second epochs comparing waveforms from different sleep stages recorded from the single-channel EEG (SP) and polysomnography (PSG). Vertical lines on the x-axis are 0.5-second intervals. Horizontal $\pm 30 \mu\text{V}$ reference lines are shown on all channels. A. Vertex wave (black bar). B. K-complex and sleep spindle. C. Delta waves. D. Sawtooth waves (black bar). Fp: frontopolar; F: frontal; C: central; O: occipital; M: mastoid.

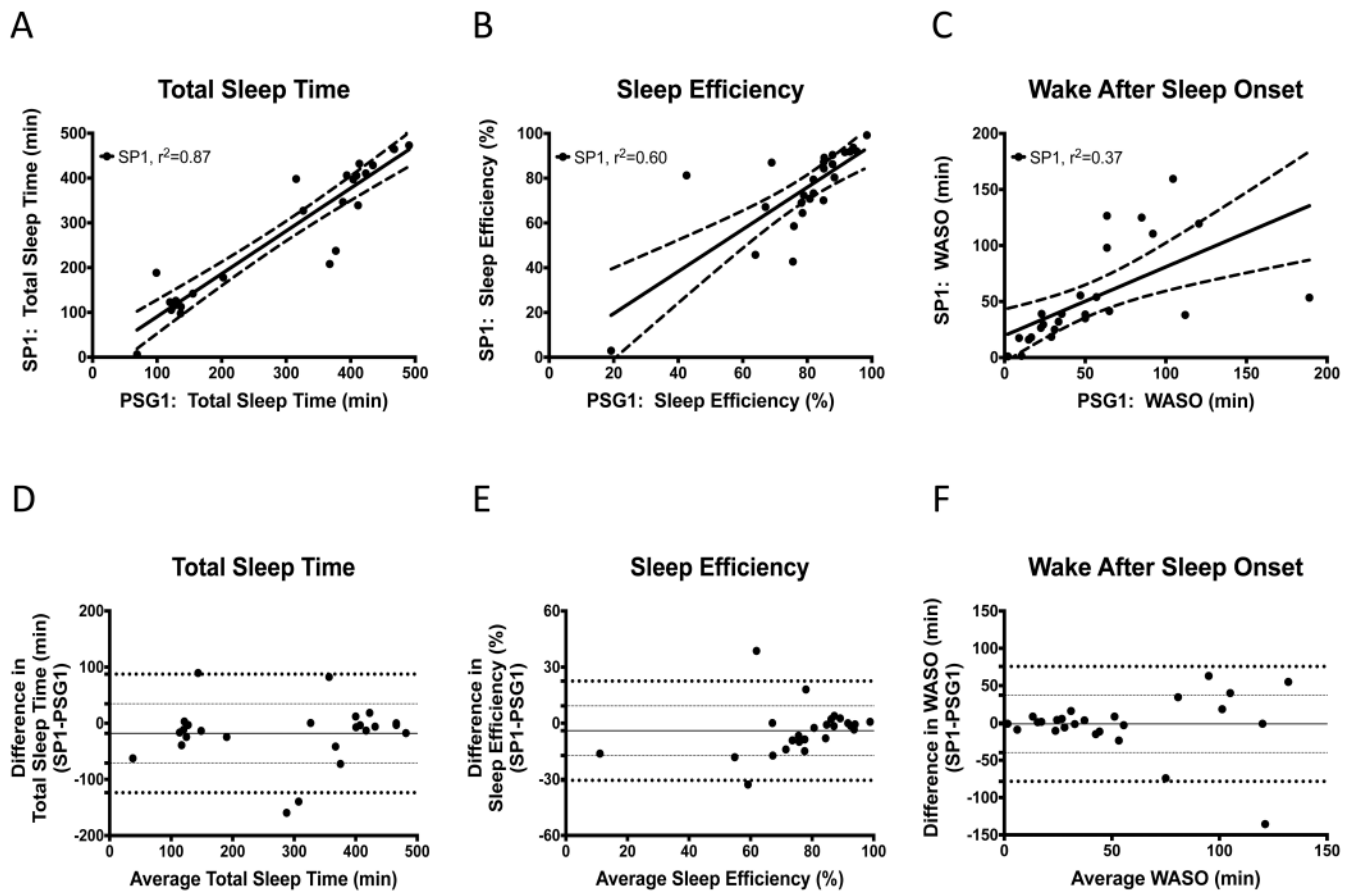


Figure 3.

Comparison of total sleep time (A, D), sleep efficiency (B, E), and wake after sleep onset (C, F) from the single-channel EEG (SP1) and polysomnography (PSG1) scored by scorer 1. A-C are scatter plots with linear regression line and 95% confidence intervals and D-F are Bland-Altman plots. For the Bland-Altman plots, difference was determined by subtracting the sleep parameter determined by single EEG from PSG. Average sleep parameter is the mean of the single EEG and PSG measurements. The mean difference is shown with the solid line, one standard deviation with small dotted lines, and two standard deviations with large dotted lines.

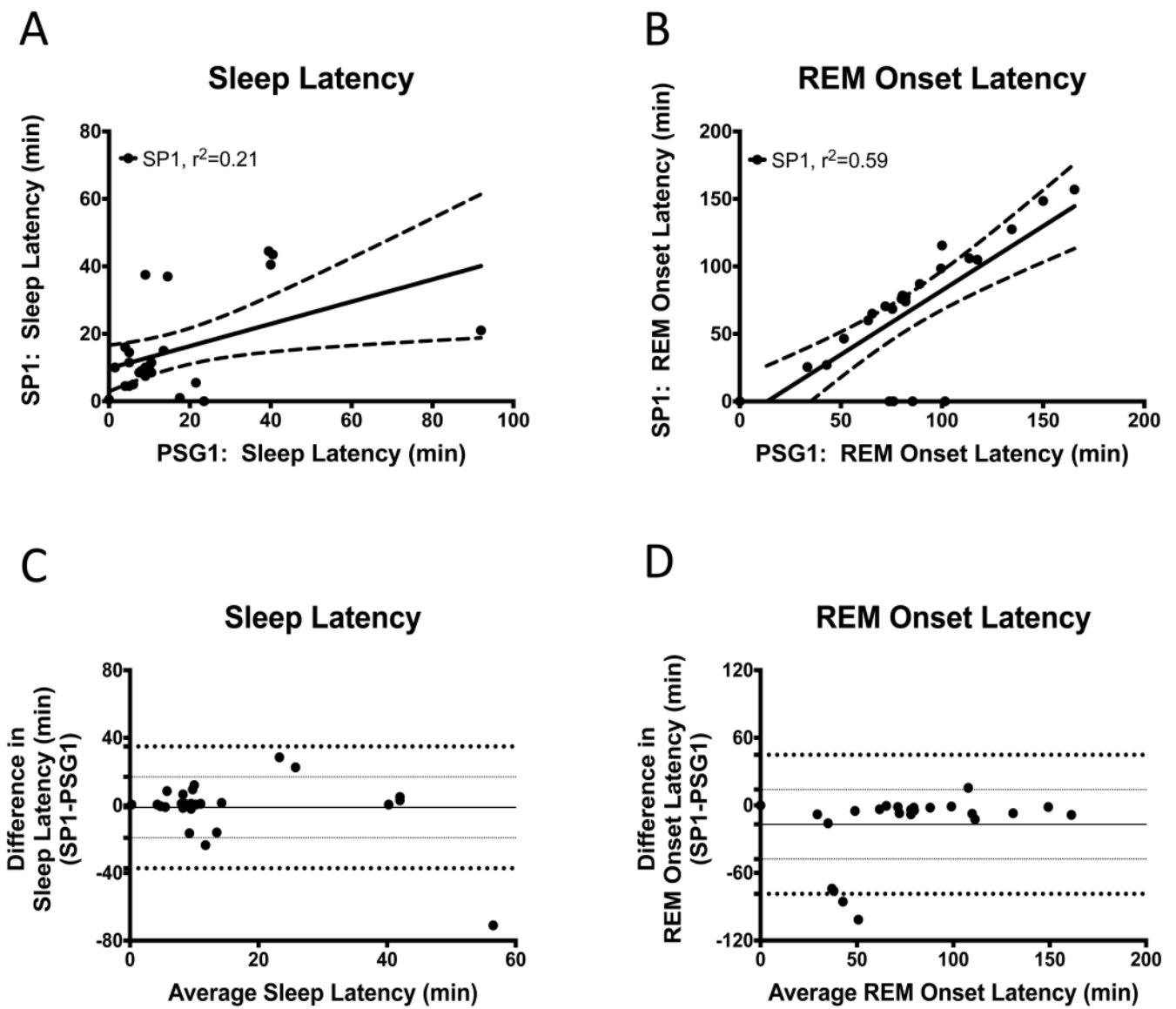


Figure 4. Comparison of sleep latency (A, C) and REM onset latency (B, D) from the single-channel EEG (SP1) and polysomnography (PSG1) scored by scorer 1. A-B are scatter plots with linear regression line and 95% confidence intervals and C-D are Bland-Altman plots. For the Bland-Altman plots, difference was determined by subtracting the latency determined by single EEG from PSG. Average latency is the mean of the single EEG and PSG measurements. The mean difference is shown with the solid line, one standard deviation with small dotted lines, and two standard deviations with large dotted lines.

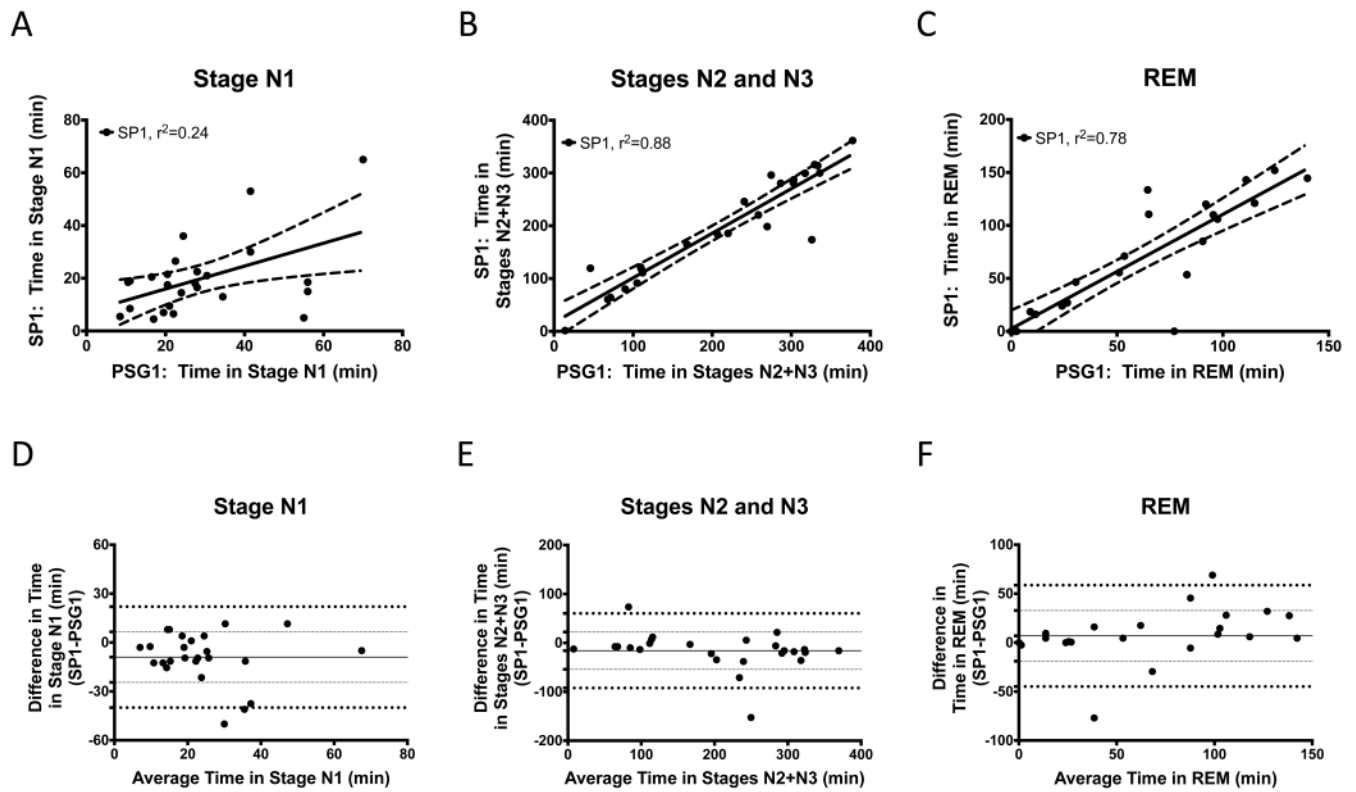


Figure 5.

Comparison of stage N1 (A, D), combined stages N2 and N3 (B, E), and REM (C, F) from the single-channel EEG (SP1) and polysomnography (PSG1) scored by scorer 1. A-C are scatter plots with linear regression line and 95% confidence intervals and D-F are Bland-Altman plots. For the Bland-Altman plots, difference was determined by subtracting the sleep stage time determined by single EEG from PSG. Average sleep stage time is the mean of the single EEG and PSG measurements. The mean difference is shown with the solid line, one standard deviation with small dotted lines, and two standard deviations with large dotted lines.

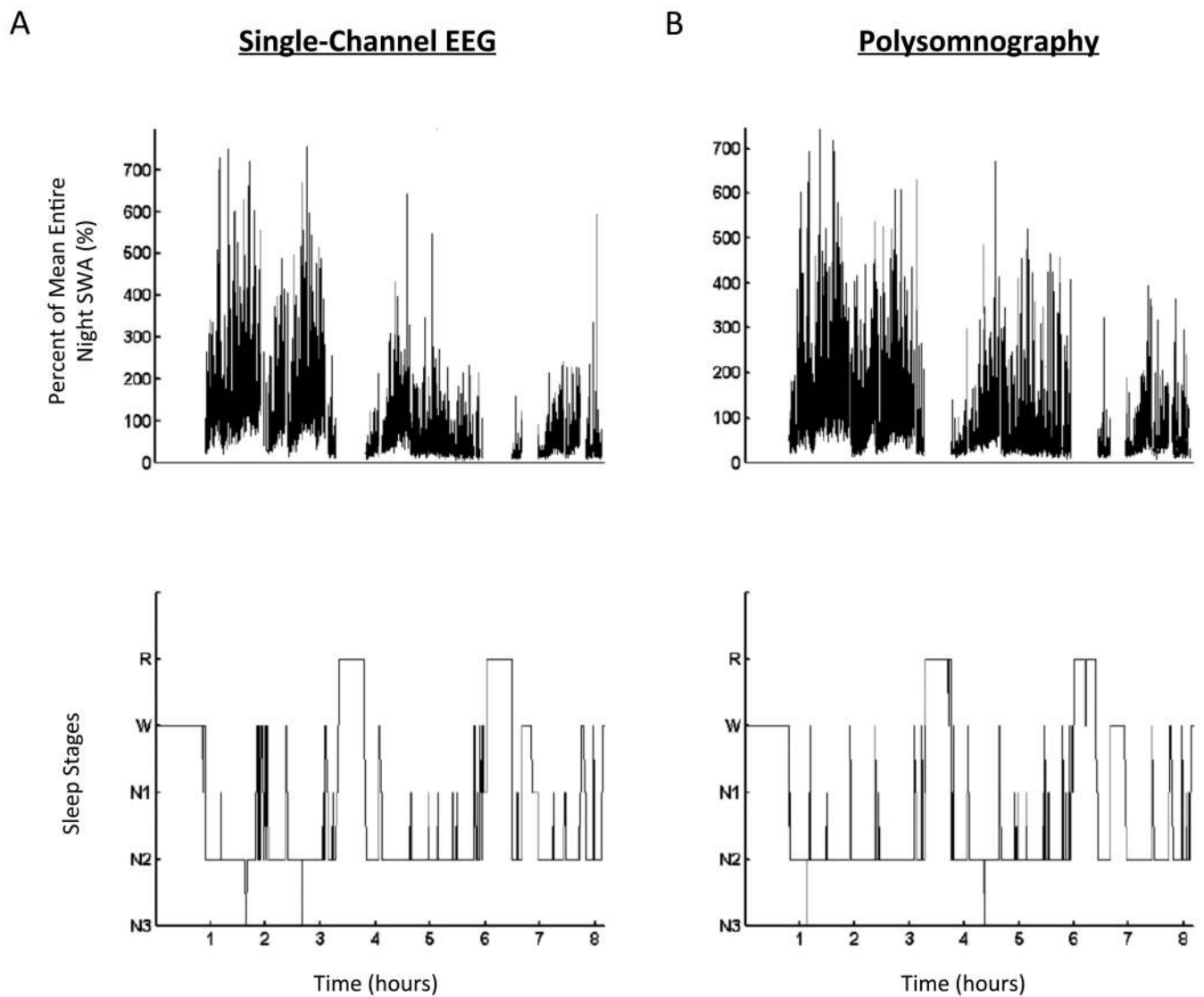


Figure 6.

Slow wave activity (SWA) and hypnogram in a single participant as measured by the single-channel EEG (A) and Polysomnography (B). SWA is expressed as a percentage of mean SWA for the entire night. Sleep stages: W = wake; N1 = NREM stage N1; N2 = NREM stage N2; N3 = NREM stage N3; R = REM sleep.

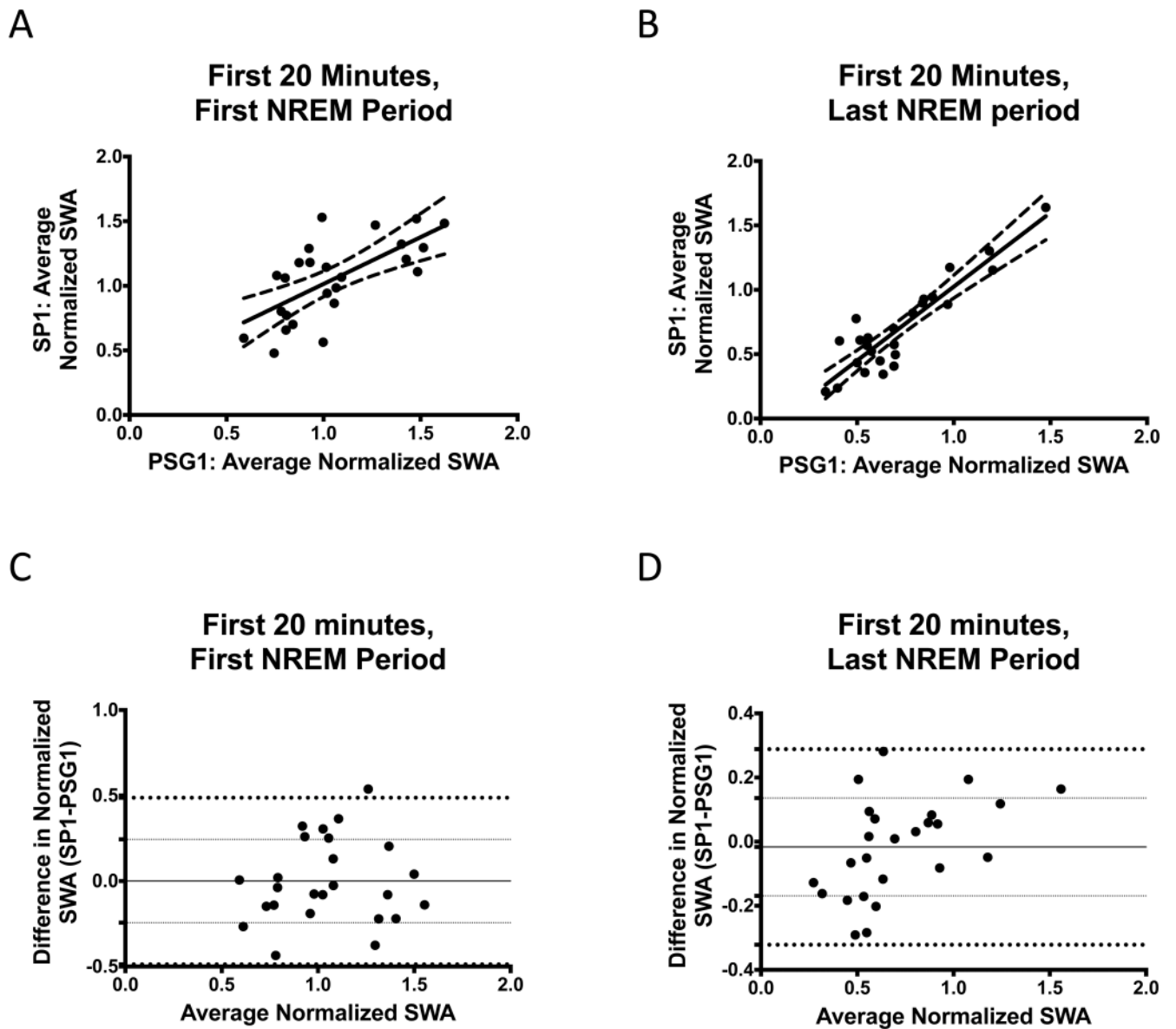


Figure 7.

For each participant, slow wave activity (SWA) during the first twenty minutes of the first NREM period (A) and last NREM period (B) of the night was normalized to the all-night average SWA. Linear regression and 95% confidence intervals are shown. PSG1: SP1: single EEG studies scored by Scorer 1. Polysomnography (PSG) studies scored by Scorer 1.

Table 1

Polysomnography vs Single-Channel EEG Sleep Stage Scoring

	Polysomnography (AASM guidelines) ¹		Single-Channel EEG	
	Channels	Scoring Criteria	Channels	Scoring Criteria
Wake	O1-M2, O2-M1:	Alpha rhythm for >50% of an epoch	Fp1-Fp2:	Alpha rhythm and eye blinks for >50% of an epoch
Stage N1	O1-M1, O2-M1: C3-M2, C4-M1:	Attenuation of alpha rhythm and slow eye movements for >50% of an epoch	Fp1-Fp2:	Attenuation of alpha rhythm and slow eye movements for >50% of an epoch
Stage N2	F3-M2, F4-M1: C3-M2, C4-M1:	K- Vertex waves complexes Sleep spindles	Fp1-Fp2:	K-complexes Sleep spindles
Stage N3	F3-M2, F4-M1, C3-M2, C4-M1:	+/- 37.5 μ V (75 μ V peak-to-peak) delta waves over 20% of an epoch	Fp1-Fp2:	+/- 30 μ V (60 μ V peak-to-peak) delta waves over 20% of an epoch
REM Sleep	EOG: Chin EMG: All EEG channels	Rapid eye movements Loss of chin tone Low amplitude mixed frequency	Fp1-Fp2:	Rapid eye movements and mixed theta-delta frequency EEG

¹Iber, 2014

AASM: American Academy of Sleep Medicine

REM: Rapid eye movement

N1: Non-REM sleep stage 1

N2: Non-REM sleep stage 2

N3: Non-REM sleep stage 3

EEG: electroencephalography

M: Mastoid

O: Occipital

C: Central

F: Frontal

Fp: Frontopolar

Table 2
Participant Demographics (N=29)

Age (years)	
Mean (standard deviation)	54 (15.7)
Range	25-80
Gender	
Male (%)	59
Female (%)	41
Ethnicity	
Caucasian (%)	62
African-American (%)	31
Other (%)	7
Apnea-Hypopnea Index (AHI)	
AHI <5	10/29
AHI 5-15	8/29
AHI 15.1-30	6/29
AHI >30	5/29
Respiratory events/hr of sleep, mean (standard deviation)	20.3 (29.8)
Range	0.1-121.4
Periodic Limb Movement Index (PLMI)	
PLMI <15	24/29
PLMI 15-45	4/29
PLMI >45	1/29
Leg movements/hr of sleep, mean (standard deviation)	9.8 (24.6)
Range	0-120.1

Table 3
Epoch-by-Epoch Comparison Between Single-Channel EEG and Polysomnography for Scorer 1 (SP1 vs. PSG1)

	Sensitivity	Specificity	PPV	NPV	Accuracy
Wake	0.776 ± 0.013	0.911 ± 0.005	0.677 ± 0.014	0.944 ± 0.004	0.885 ± 0.012
Stage N1	0.206 ± 0.02	0.957 ± 0.003	0.289 ± 0.027	0.934 ± 0.004	0.898 ± 0.034
Stage N2+N3	0.873 ± 0.006	0.889 ± 0.007	0.914 ± 0.005	0.838 ± 0.008	0.880 ± 0.006
REM	0.862 ± 0.013	0.952 ± 0.003	0.761 ± 0.015	0.975 ± 0.003	0.938 ± 0.009

SP1: Single-channel EEG studies scored by Scorer 1; PSG1: Polysomnography scored by Scorer 1; PPV: Positive predictive value; NPV: Negative predictive value. For each value of sensitivity, specificity, PPV, NPV, and accuracy, the 95% confidence intervals are shown.

Table 4
Average Sleep Parameters from Scorer 1 for Subjects with Studies $\leq 10\%$ Artifact (N=25)

	Single-Channel EEG		Polysomnography		ICC
	Mean	SEM	Mean	SEM	
TST (min)	277.3	29.6	295.4	28.9	0.96
SL (min)	15.1	2.8	16.3	3.9	0.67
REML (min)	64.6	9.7	81.4	7.8	0.72
WASO (min)	52.7	8.7	54.0	8.7	0.79
Stage N1 (min)	19.7	2.9	28.7	3.2	0.66
Stage N2+N3 (min)	195.1	19.9	211.0	22.1	0.96
REM (min)	62.5	11.0	55.7	9.0	0.92
SE (%)	74.8	4.2	78.8	3.4	0.86

TTST: Total sleep time; SL: Sleep latency; REML: REM onset latency; WASO: Wake after sleep onset; Stage N1: NREM stage 1 sleep; Stage N2+N3: Combined NREM stage 2 and stage 3 sleep; REM: Rapid eye movement sleep; SE: Sleep efficiency; Min: Minutes; SEM: Standard error of the mean; ICC: Intraclass Correlation Coefficients