INFORMATION ON THE EFFECTS OF EEG AND ECG FEATURES ON HYPNOSIS

EEG Spectral Properties and Associated ECG-Based Heart-rate Variability in People With Insomnia Versus Healthy Sleepers

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INTRODUCTION

Insomnia is a sleep disorder characterized by difficulty falling asleep, staying asleep, daytime impairment or distress, or waking up too early and not being able to fall back asleep. The American Academy of Sleep Medicine defines insomnia as difficulties in initiating and maintaining sleep for a minimum period of 1 month, despite adequate opportunity and favorable conditions for sleep.1

Sleep disorders disrupt normal central and autonomic nervous system function during sleep, which may manifest as anorexia in electroencephalography (EEG)/electrocardiography (ECG) coupling.2,3

People with insomnia have increased EEG (8–12 Hz) and (11–16 Hz) power during rapid eye movement (REM) sleep, and increased 0.5–4 Hz power and increased HR (R–R, n), and in power during non-REM (NREM) sleep.1

People with insomnia also have notably lower EEG-based heart-rate variability (HRV) during NREM and REM sleep compared with healthy sleepers.1

This study aimed to compare the sleep architecture, central and autonomic nervous system functions, and EEG/ECG coupling of healthy sleepers versus people with insomnia using polysomnography.

Methods

This study used publicly available, de-identified, polysomnography data from 9 people with insomnia1 and 12 age-matched, healthy sleepers drawn as a subset from an IRB-reviewed, exempt status study conducted by Sleep Number Corporation. The insomnia cohort was free of neurological disease and did not affect the central nervous system.1

All insomnia phenotypes were included in the analysis, as the sample was insufficient to stratify by insomnia phenotype. 

Sleep architecture was characterized for all study participants and the following bands were measured for REM and NREM sleep: spectral EEG power in the A, R, and (0.5–4 Hz) bands, mean heart rate, and mean HRV metrics (Figure 1).

The A band was included to identify patterns associated with micro-arousals. HRV metrics included standard deviation of normal-to-normal intervals (SDNN), absolute power of the high frequency band (HF, 0.15–0.4 Hz), absolute power of the low frequency band (LF, 0.04–0.15 Hz), and LF power, and ratio of LF to HF.2

Normal-to-normal intervals are defined as R–R intervals after outlier removal.

Values for EEG power were log-transformed before analysis, as the range of values differed greatly between subjects.

The LF/HF ratio were predictors of EEG coupling revealed that HR, SDNN, HF, LF, and the power of the low frequency band divided by total power. Values for EEG power were significantly higher for healthy sleepers than insomnia patients, with the exception of lower HF power in NREM sleep in people with insomnia, as shown in the following equation (adjusted R² = 0.705, F = 0.713).

A second generalized linear model analysis was performed to account for collinearity and interactions, which significantly increased model predictability (adjusted R² = 0.974, F = 0.113; Table 3).

These results show that NREM in healthy sleepers is associated with a lower LF/HF—low central nervous system activity—and high HF, indicating high parasympathetic autonomic nervous system activity.

In our study, we observed significantly lower LF/HF in the insomnia group compared with healthy sleepers. These results may indicate challenges in falling asleep, as the increase in LF/HF in NREM is a hallmark of the hypnagogic (falling asleep) phase.6

These results suggest that, in people with insomnia, the parasympathetic dominance in NREM sleep is challenged by higher LF, which may decrease sleep quality.

Further, these results are consistent with previous research noting associations between LF/HF and micro-arousals during sleep.9

Adding the interaction between the SDNN time-domain metric significantly increased HRV metric significantly increased adjusted R².10

Further research with a larger population is needed to confirm these results.

RESULTS

The coils were similar in demographic characteristics, baseline sleep architecture, and cardiovascular parameters during sleep (Table 1).

In our study, we observed significantly lower LF/HF in the insomnia group compared with healthy sleepers. These results may indicate challenges in falling asleep, as the increase in LF/HF in NREM is a hallmark of the hypnagogic (falling asleep) phase.6

We also observed that power in NREM, likely a marker of restlessness, negatively correlated with HF—reflecting parasympathetic activity—and positively correlated with LF, reflecting sympathetic activity.9

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FIGURE 2: TEMPORAL ALIGNMENT OF EEG- AND ECG- DERIVED METRICS DURING A SLEEP SESSION.

TABLE 1. DEMOGRAPHIC, SLEEP ARCHITECTURE, AND CARDIOVASCULAR ATTRIBUTES OF THE STUDY COHORT.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Healthy cohort</th>
<th>Insomnia cohort</th>
<th>Fisher</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>55.0 ± 17.2</td>
<td>51.9 ± 32.3</td>
<td>0.705</td>
</tr>
<tr>
<td>Gender (%)</td>
<td>66.7 ± 33.3</td>
<td>58.3 ± 25.0</td>
<td>0.64</td>
</tr>
<tr>
<td>Males (%)</td>
<td>57.1 ± 36.2</td>
<td>45.9 ± 38.2</td>
<td>0.03</td>
</tr>
<tr>
<td>Sleep metrics (min)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>REM</td>
<td>66.1 ± 22.4</td>
<td>51.9 ± 32.3</td>
<td>0.03</td>
</tr>
<tr>
<td>NREM</td>
<td>65.6 ± 23.3</td>
<td>51.9 ± 32.3</td>
<td>0.03</td>
</tr>
<tr>
<td>Cardiovascular metrics, mean ± SD</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (beat/min)</td>
<td>66.6 ± 26.8</td>
<td>67.6 ± 9.6</td>
<td>0.64</td>
</tr>
<tr>
<td>SDNN (ms)</td>
<td>50.2 ± 32.0</td>
<td>51.9 ± 32.3</td>
<td>0.50</td>
</tr>
<tr>
<td>LF (0.04–0.15 Hz) power</td>
<td>0.9 ± 0.1</td>
<td>0.9 ± 0.1</td>
<td>0.70</td>
</tr>
<tr>
<td>LF/HF</td>
<td>4.1 ± 2.7</td>
<td>2.6 ± 1.2</td>
<td>0.02</td>
</tr>
<tr>
<td>REM power</td>
<td>0.8 ± 0.1</td>
<td>0.9 ± 0.1</td>
<td>0.80</td>
</tr>
<tr>
<td>REM/HF</td>
<td>0.0 ± 0.0</td>
<td>0.0 ± 0.0</td>
<td>0.70</td>
</tr>
</tbody>
</table>

A generalized linear model analysis for EEG/ECG coupling revealed that HR, SDNN, HF, LF, and the LF/HF ratio were predictors of EEG coupling during NREM sleep in people with insomnia, as shown in the following equation (adjusted R² = 0.705, F = 0.713).

The coefficients are validated at the level of the sample and not for permission to reuse any content. Medical writing support was provided by Sandra Page, PhD, University of Oxford. This study used publicly available, de-identified, polysomnography data from 9 people with insomnia1 and 12 age-matched, healthy sleepers drawn as a subset from an IRB-reviewed, exempt status study conducted by Sleep Number Corporation.

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