Atypical Heart Rate Increase after Sleep Onset

Anda Baharav¹,², Yair D. Fuxman², Arie Oksenberg³
(1) Shaare Zedek Medical Center, Sleep Disorders Clinic, Jerusalem, Israel; (2) HypnoCore, Yehud, Israel; (3)Loewenstein Hospital-Rehabilitation Center, Sleep Disorders Unit, Ra’anana, Israel.

Introduction
- The decrease in heart rate after sleep onset is a well recognized behavior. We observed an atypical increase in HR after sleep onset in several patients referred for a polysomnogram (PSG) for suspected sleep apnea.
- A lack of reduction in heart rate has been associated with increased mortality risk.¹ ²
- We hypothesize that this behavior is the result of an abnormal autonomic cardiovascular control at least during sleep in these subjects.
- We aimed to estimate the autonomic function during sleep using time frequency decomposition of heart rate variability (HRV) as a measure of autonomic cardiovascular control.

Methods
- PSG data from 8 patients that exhibited increasing HR were compared to a control group of 16 studies that exhibit the characteristic decline in HR (Fig. 1).
- HRV analysis was performed using the HC1000P software.
- Control studies were matched for age, sex, BMI and Apnea/Hypopnea Index (AHI).
  - Age 50.0±9.2, BMI 27.0±5.0, AHI was 7.0±7.3
- HRV analysis was performed as follows: ECG signals were analyzed by the HC1000P software based on time-dependant spectral analysis of the RR interval. Thus we calculated: very low frequency (VLF) which is associated with vasomotion and thermoregulation, low frequency (LF) which is associated with sympathetic and parasympathetic fluctuations, high frequency (HF) associated with mainly parasympathetic fluctuations, and the Autonomic Balance Index (ABI = LF/HF) across the night during different sleep states.
- Six parameters were compared: VLF, LF, HF, ABI, heart rate (HR) and DC which is associated with mean RR interval.
- Differences between groups were examined by two tailed t-test.

Results
- Compared with the control group, the group with rising heart rate was found to have (Fig. 2):
  - Significantly higher HR than the control group for all sleep stages (p < 0.05).
  - Lower VLF for all sleep states but did not reach statistical significance.
  - Significantly higher LF during wakefulness (p < 0.05).
  - Higher HF for all sleep stages, reaching statistical significance for the entire night (p < 0.1), light sleep (p < 0.1) and during wakefulness (p < 0.05).
  - Lower ABI during light sleep (p < 0.1).

Conclusions
- Subjects with an atypical increase in HR after sleep onset showed a significantly higher HR during all sleep/wake stages and a significantly higher LF during wakefulness, expressing higher sympathetic activity.
- Thus this atypical behavior of the HR after sleep (non-dipping HR) may represent a marker of increased cardiovascular risk in these patients.

References