What causes the differences in cardiac activity within and between subjects during sleep?

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absence of obstruction or arousal, some dial-downs were preceded by hypercapnic and/or hypoxic breathing for 30 seconds, inducing a range of ventilatory stimulation during which breath-by-breath VE and αHR were determined. The HR response to ventilatory stimulation (αHR/VE) was calculated from these observations. In each patient the increase in HR in the first two post-event breaths was compared to the increase expected from the αHR/VE at the observed post-event VE levels.

**Results:** αHR/VE ranged 0 to 0.91 beats.L-1.m in different subjects (0.43 ± 0.28). The post-event changes in HR, relative to pre-dial-down HR, ranged −2.7 to 5.8 beats.min-1 in B1 (1.2 ± 2.2; p < 0.02) and −1.7 to 6.9 beats.min-1 in B2 (2.3 ± 2.4; p < 0.001). The difference between observed and expected αHR was −0.06 ± 2.02 min-1 for B1 and 0.55 ± 2.00 min-1 for B2. Neither value was significant.

**Conclusion:** Post-event tachycardia in OSA in the absence of cortical arousal can be entirely explained by the post-event increase in ventilation.

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**0172**

**CARDIAC ACTIVITY IN ADULTS WITH AUTISM BEFORE AND AFTER SLEEP**

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**Introduction:** Poor sleep is a frequent finding in autism and has been shown to interfere with daytime functioning in adults with autism (Limoges et al., 2013). Literature in typically developing individuals (TD) shows that sleep also influences the regulation of the autonomic nervous system so that the sympathovagal tone is normally higher in the morning compared to evening. Electrocardiographic (ECG) studies suggest the existence of a sympathetic-parasympathetic disequilibrium in autism (Ming et al., 2005). This research tested whether this observation is related to sleep or not.

**Methods:** 12 adults with ASD (20.8 ± 4.2 years) and 12 TD individuals (22.1 ± 4.0 years) were evaluated over two consecutive nights in a sleep laboratory using polysomnography. ECG samples were taken 5 minutes before and after sleep period. Spectral analysis of the heart rate variability was done using a commercial software and the following variables were extracted: total spectral power (TSP), absolute values of low (LFabs: sympathetic tone) and high (HFabs: parasympathetic tone) frequency spectral powers and normalized values of low (LFnu) and high (HFnu) frequency spectral power. Analyses were performed using repeated measures ANOVAs.

**Results:** Preliminary results show significant differences between evening and morning values in both groups, with higher morning values for TSP (p = 0.012), LFabs (p = 0.001) and LFnuf (p = 0.004). No significant difference was found for the HFabs but HFnu was significantly lower in the morning (p = 0.007). No significant differences were found between the ASD and TD groups.

**Conclusion:** These results suggest that the effect of nocturnal sleep was similar in both groups with higher sympathetic activity in the morning. Interesting results show a higher parasympathetic influence on heart rate variability in the evening for TD and ASD participants. Further analyses will focus on ECG activity during sleep, for each of the sleep stages.

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**0173**

**WHAT CAUSES THE DIFFERENCES IN CARDIAC ACTIVITY WITHIN AND BETWEEN SUBJECTS DURING SLEEP?**

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**Introduction:** It is known that cardiac activity varies across sleep stages. However, it has not been quantitatively investigated in what aspects the cardiac activity is influenced by within-/between-subject differences. The differences can be caused by many factors such as subject demographics, time and (cardiac) physiology. We hypothesize that these factors affect the cardiac activity during sleep. Therefore, we try to quantify these effects leading to cardiac variations within and between subjects, which can be potentially used to help separate sleep stages.

**Methods:** We considered overnight heartbeats, obtained from electrocardiographic signals, from 165 healthy adults (age 51.8 ± 19.4 years). Sleep stages were scored on 30-s epochs with polysomnography according to R&K rules. To investigate the abovementioned effects on cardiac activity, we applied multilevel models that consider structural variables at hierarchical levels. Two cardiac parameters were analyzed: mean heart rate (HR) and standard deviation of heartbeat intervals (SDNN). The models (with two levels: subject and time) included variables regarding effects from sleep stages (wake, REM, light and deep sleep), demographics (age, gender and body mass index), time of night and physiological differences within and between subjects.

**Results:** For both parameters, all the effects mentioned above were found to be significant (Wald Z-test, p < 0.05). Further, when excluding the variance caused by sleep stage, the variances explained by demographics, time, physiology within subjects and physiology between subjects respectively accounted for 3.4%, 4.0%, 12.4% and 80.2% of the total variance for HR, and 13.7%, 2.4%, 40.9% and 43.0% for SDNN.

**Conclusion:** Demographics, time and within-/between-subject physiological differences have significant effects on cardiac activity during sleep. The major effects come from the differences within and between subjects in physiology, accounting for > 80% of total variance (except sleep stage). Practically, for cardiac-based sleep staging, the main challenge is to reduce these within-/between-subject differences.

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**0174**

**AUTONOMIC CHANGES AFTER SLEEP RESTRICTION - EVIDENCE OF AN ALLOSTATIC MECHANISM**

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**Introduction:** Chronic sleep problems are commonly associated with mood difficulties, which are potentially linked to neurophysiological hyperarousal. It has been previously shown that total sleep deprivation interferes with autonomic function resulting in reduced vagal tone. However, research using partial sleep deprivation (PSD) protocols, which more closely approximate a real-world model of insomnia, is limited. Here we assessed the effects of repeated PSD on physiological and self-reported responses to emotional stimuli.

**Methods:** After a 2-min baseline, 22 participants (28% men, ages 25 ± 2.2) viewed validated neutral and sad film clips. Electrocardiography data used to derive heart rate (HR; beats/min) and heart rate vari-