1031

HEMISPHERIC PHASE AND AMPLITUDE SYNCHRONY DIFFERENCES BETWEEN SLEEP AND WAKE CONDITIONS

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Introduction: Changes in hemispheric synchrony have been observed during the transition from wakefulness to sleep using coherence and correlation measures. The purpose of the present study is to assess differences in hemispheric synchrony between sleep and wakefulness using more sensitive measures in the phase and amplitude domains.
Methods: Data for this analysis came from 21 patients (mean age 45) who underwent a Multiple Sleep Latency Test for evaluation of their hyper-somnolence. The first nap was segmented in 2.5s epochs for spectral decomposition (FFT). Epochs were sleep/wake classified using a sleep index (alpha + beta)/ (theta + delta), and values <.30 were classified as waking and those >.30 as sleep. Several synchrony measures were calculated from O1-A1 and O2-A2 derivations as follows: The twelve Hz signal was extracted from both EEG channels using a first-order complex Gaussian continuous wavelet transform (CWT). Complex CWT results were angular transformed and unwrapped to produce phase time series. Synchrony Index (SI) the averaged normalized phase distances between both EEG channels was calculated; Synchrony Entropy Index (SEI), the deviation of phase differences from the uniform distribution was quantified using an approach based on Shannon's entropy. The Hilbert transformation of the real component of the CWT results yielded the demodulated amplitude. The Synchrony Amplitude Index (SAI), the entropy estimation of the demodulated amplitude differences, was calculated.
Results: ANOVA demonstrated that phase synchrony measures increased from wake to sleep, SI, (wake=0.179, sleep=0.224, $p<.001$), SEI (wake=0.39, sleep=0.6, $p<.001$). In contrast the amplitude synchrony measure decreased during this transition, SAI (wake=0.232, sleep=0.181, $p<.001$).
Conclusion: Previous findings that have shown increased hemispheric synchrony in the wake to sleep transition may have conflated phase and amplitude domains. We propose that wake to sleep transitions are represented differently in the phase and amplitude synchrony domains.

1032

EFFECTS ON MAINTENANCE OF WAKEFULNESS TEST OF RECOVERY SLEEP DOSE FOLLOWING SLEEP RESTRICTION

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Introduction: Little is known about the sensitivity of the Maintenance of Wakefulness Test (MWT) to differing sleep period lengths (TIB) following chronic sleep restriction. This study investigated the effect of varying TIB sleep doses on a night following 5 nights of sleep restriction (SR).
Methods: N=118 healthy subjects (age=30.12±6.98yr, 64f) participated in a controlled laboratory protocol. Subjects underwent 2 nights of baseline sleep (TIB=10h) followed by 5 nights of SR (TIB=4h) and a night of varying time in bed for recovery sleep (R1). On R1 subjects were randomized to one of six TIB sleep doses (0h n=13; 2h n=18; 4h n=24; 6h n=16; 8h n=17; 10h n=21 TIB). Modified single trial (30min) MWTs were conducted between 1430h-1600h on the day after the second baseline night, after the fifth SR night (SR5) and after R1. Sleep latency was defined as time to the first appearance of a brief sleep (10sec microsleep). N=9 served as 10h TIB per night control subjects.
Results: MWT sleep latency (±SD) after 5 nights of SR was 10.48±8.6min, which differed significantly from baseline 19.12±10.3min ($p<0.001$). At SR5, sleep latency was shorter in the sleep restricted group (10.48±8.6min) versus the control group (22.26±9.32min; $p<0.01$). R1 sleep doses of ≤6h TIB (0h, 2h, 4h, 6h) yielded MWT sleep latencies significantly below those at baseline (all $p<0.05$). R1 0h and 2h TIB decreased ability to stay awake significantly below that on SR5 ($p<0.05$). R1 10h TIB improved ability to stay awake significantly above SR5 ($p<0.05$), however 8h TIB did not ($p>0.05$).
Conclusion: The MWT was sensitive to SR and to the dose of subsequent TIB for sleep. However, MWT showed significant recovery only after a sleep dose of 10h TIB. Analyses are underway to determine the nature of physiological sleep obtained in the 10h TIB.
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1033

A CLASSIFICATION SCHEME FOR MEDICATION USE IN CLINICAL RESEARCH SAMPLES

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Introduction: Medication use data is usually collected in clinical research. However, there exists no standardized method for categorizing these data, either for sample description or for the study of medication use as a variable. We developed a simple, empirically based classification scheme for medication use. Here we describe the development technique and to illustrate its potential, we apply it to samples where sleep disorders are prominent.
Methods: The development sample included 480 participants (45 men: mean age=56.56; 18 women: mean age=58.11, S.D.=12.97), recruited for a large, multi-stage study on daytime functioning in sleep disorders. As part of the questionnaire battery, participants provided information on medication use. Participants also underwent polysomnographic assessment of their sleep. Based on the Canadian Compendium of Pharmaceuticals and Products (CPS) and subsequent consensus grouping, medications were coded into 16 specific therapeutic classes. A principal components factor analysis was carried out to reduce the number of classes into usable categories. Hypotheses were developed and tested on: 63 individuals with Sleep Apnea/Hypopnea Syndrome (SAHS), 24 with Chronic Fatigue Syndrome (CFS), and 48 Healthy Controls.
Results: The factor analysis yielded 5 categories: 1) Psychoactive medication, 2) Metabolic syndrome/pain related medication, 3) Respiratory/gastric/urologic related medication, 4) Infection/skeletal/lifestyle related medication, and 5) Hormonal medication. As a preliminary test of validity of the classification scheme for medication use we tested and confirmed the hypotheses (a) that participants with SAHS use significantly more Cardiovascular medication than do CFS or Control participants and (b) that CFS participants use significantly more Psychoactive medication than do SAHS or Control subjects.
Conclusion: The categorization system we developed is easy to use and appears to discriminate some populations. We believe that a standardized classification scheme for medications commonly found in clinical populations could be of great benefit to researchers by allowing (1) better sample descriptions and (2) clearer conceptualizations of the role of medications in the etiology, maintenance, and treatment of illness.