of sleep medication vs. no use (adjusted OR=4.72, 95% CI: 1.91-11.64) during the month following stroke.
Conclusion: Short sleep duration, poor sleep quality, and use of sleep medication during the 1-month following stroke were all associated with depression 6-months post-stroke. Since depression is associated with worsened functional recovery, prevention and/or treatment of poor sleep should be prioritized following a stroke to encourage optimal psychological and physiological health.
Support (If Any): R01HL141494

0932

USING NOVEL EEG PHENOTYPES AND ARTIFICIAL INTELLIGENCE TO ESTIMATE OSA SEVERITY

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Introduction: EEG studies are widely used for monitoring and diagnosis of neurological conditions including epilepsy, seizure disorders, among others. Ambulatory EEG, EEG-video monitoring, and long-term EEG monitoring typically result in several full nights of sleep EEG data. In this work, we leverage artificial intelligence methods that achieved breakthrough performance in related domains with large clinical EEG datasets, to explore our hypothesis that neurological phenotypes that highly correlate with sleep disordered breathing can be extracted from overnight EEG recordings. Furthermore we hypothesize that these EEG phenotypes can be used to accurately predict a patients OSA severity, without accompanying cardiopulmonary data.
Methods: We used cross-sectional analyses of adult patients (N = 4650) who completed an overnight PSG study. All signals were excluded from analysis except for the standard 10-20 EEG sensor array, to simulate an ambulatory or video-EEG acquisition for the present study. Global phenotypic features were derived from the patients full-night sleep architecture and fragmentation profiles. Local phenotypic features were derived by analyzing biomarker patterns and respiratory cycle-related EEG changes exhibited in the EEG signals directly. Artificial Intelligence methods including Bidirectional-LSTM and Deep-CNN were trained, optimized, and evaluated to model the relationship between global and local EEG phenotypes and OSA severity. Performance for predicting moderate and severe OSA (AHI ≥ 15) was evaluated using randomized 10-fold cross-validation.
Results: The best performance was obtained by a combination of the Bidirectional-LSTM and Deep-CNN architectures, with an average accuracy, sensitivity, and specificity of 91.1%, 86.9%, and 99.5% respectively for predicting moderate and severe OSA.
Conclusion: This and prior work have demonstrated a promising opportunity to estimate OSA severity with a host of EEG study types using applied artificial intelligence. Future research involving a cohort of ambulatory EEG subjects, controlled for OSA severity, can validate the efficacy of this approach in the clinical setting. Following further validation, AI based risk estimates could be incorporated into diagnostic EEG reports, to provide clinicians with additional means for identifying patients with moderate and severe OSA that may benefit from follow-up diagnosis and treatment.
Support (If Any):

0933

SELECTIVE SLEEP DEPRIVATION AND SEIZURE RECURRENCE INJUVENILEMYOCLONICEPILEPSY

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Introduction: It is well known the association of sleep deprivation and the risk of seizure recurrence in epileptic patients, however the role of selective patterns of sleep deprivation is yet to be defined in certain epilepsy syndromes with its associated implications on seizure recurrence and functionality. We wanted to evaluate patterns on selective sleep deprivation in patients with juvenile myoclonic epilepsy (JME).
Methods: We analyzed sleep deprivation in 2 different forms: Late night sleep delay (LNSD) defined by more than 1.5 hours of the habitual sleep onset time missed, and early morning (EMSD); of 1.5 hours or more missed in the early morning awakening. We compared the recurrence of seizures in a known population of 15 JME patients that fulfilled clinical and neurophysiological criteria, we analyzed a sleep detailed history that included logs and observation by family members, variables associated with sleep schedule and total sleep time were noted. We evaluated the onset of sleep and morning awakening times in periods that included 3-month blocks before the occurrence of seizures. All the patients were compliant with the anti-epileptic treatment.
Results: A relationship between all forms of sleep deprivation and seizures was seen in all the patients. LNSD and EMSD were clearly associated with increased frequency and duration of early myoclonic jerks in the first 2 hours after awakening. There was as well increase in seizure frequency with both types of sleep deprivation; however, the most significant predisposing factor was EMSD of 90 minutes or more of the habitual waking time. LNSD and seizures was observed in 25 % of the patients compared to 75% with EMSD ($p < 0.01$).
Conclusion: Sleep deprivation is clearly associated in the generation of seizures in different seizure syndromes. Early sleep morning awakening is a more selective contributor for seizure recurrence than delayed sleep time in patients with Juvenile Myoclonic Epilepsy and this selective deprivation can increase the seizure frequency as much as 3 times more than the late-night sleep. Further testing on other forms of epilepsy is needed.
Support (If Any): None

0934

EFFECTS OF SLEEP QUALITY AND PAIN INTENSITY ON NEGATIVE AFFECT IN IDIOPATHIC PARKINSON'S DISEASE

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