P1511 / #4426

Topic: AS10 Sleep, Biological Rhythms

EFFECT OF FOOT REFLEXOLOGY ON SLEEP QUALITY AND SEVERITY OF FATIGUE IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION: A DOUBLE-BLIND RANDOMIZED CLINICAL TRIAL

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Sleep disorders and fatigue are common problems in patients with acute myocardial infarction (AMI). The aim of this study was to determine the effect of foot reflexology on sleep quality and fatigue severity in the AMI patients. In this double-blind randomized clinical trial, 90 AMI patients who had inclusion criteria were evaluated from cardiac care unit of Shahid Rajaee Hospital in Karaj, Iran from 30 may 2015 to 20 August 2016, and were randomly assigned to 3 groups: Treatment (n = 30), placebo (n = 30), and control (n = 30). Left foot reflexology in the treatment group and right foot reflexology were performed in the placebo group. The control group did not receive any intervention. Foot reflexology intervention was performed for 3 consecutive days for 20 minutes. The severity of fatigue was assessed in 3 groups of above, before, immediately and 20 minutes later of intervention by VAS. Sleep quality was assessed by Pittsburgh questionnaires and VAS, the day before and two consecutive days after intervention. The fatigue intensity score of the treatment group immediately after intervention was not significantly different from the control (P = 0.233) and placebo groups (P = 0.903). Remarkably, the control group was not significantly different from placebo (P = 1.00). The fatigue severity score in 20 minutes after intervention was significantly lower than the control group (P = 0.003), but did not differ significantly from the placebo group (P = 0.355). Also, fatigue severity score of the control group did not differ significantly from the placebo group (P = 0.218). Sleep quality with VAS and Pittsburgh on first and second day after intervention wasn't significantly different in the treatment groups compared with the control and the placebo groups and in the control group than the placebo group (P > 0.05). Findings showed that foot reflex massage 20minutes after intervention induced of significant reduction in fatigue severity in the AMI patients.

Declaration of Interest Statement: None

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P1512 / #4430

Topic: AS10 Sleep, Biological Rhythms

THE EFFECT OF CURCUMA LONGA ON SLEEP QUALITY IN PATIENTS WITH ACUTE CORONARY SYNDROME: A TRIPLE-BLIND RANDOMIZED CLINICAL TRIAL

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Sleep disorders are common problems in acute coronary syndrome patients. The aim of this study was to determine the effect of Curcuma Longa on sleep quality in patients with acute coronary syndrome. This triple-blind clinical trial was conducted on 270 patients in the Coronary Care Unit of Kowsar Hospital in Semnan, with random allocation to three groups (intervention, placebo and control) in 2020- 2021. The intervention group received 500 mg of Curcuma Longa tablets and the placebo group received 500 mg of starch, Oisel and lactose tablets at 9 a.m. after breakfast once a day for 3 days. The control group received daily interventions. Sleep quality was evaluated by the standardized sleep quality questionnaire of Saint Mary's Hospital in the morning of the first day before the intervention and in the morning of the second and third days after the intervention. The average score of sleep quality before the intervention in the intervention group was higher than that of in the placebo group (P = 0.021), but on the second and third days of the intervention, it was lower in the intervention group compared to the placebo (P < 0.001) and control groups (P < 0.001). The results showed that in the patients with acute coronary syndrome, Curcuma Longa improved the quality of sleep. Therefore, Curcuma Longa can be used to improve the sleep quality of these patients.

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Topic: AS10 Sleep, Biological Rhythms

RESPIRATION ASSOCIATES WITH HIPPOCAMPAL ELECTROPHYSIOLOGICAL STATE IN URETHANE-ANESTHETIZED RATS

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Breathing and heartbeat synchronize to each other and to brain function and affect perception and cognition in humans. Convincing evidence links especially nasal respiration to hippocampal electrophysiology, but it is not quite clear how cardiorespiratory rhythms modulate memory consolidation in the hippocampus. In experiment 1 we studied if respiration and heartbeat synchronize to the occurrence of spontaneous hippocampal oscillations in urethane-anesthetized (1.3 g/kg, i.p.) adult male Sprague-Dawley rats (n = 8). We recorded local-field potentials from left and right dorsal hippocampus using 32-electrode linear silicon probes together with respiration (piezo) and heartbeat (electrocardiogram). Each animal

was recorded for several hours. Changes in respiration, but not in heart rate, paralleled changes in the oscillatory state of the hippocampus: CA1 sharp-wave ripples (SPW-Rs) were more frequent when respiration slowed down. In half of the rats, dentate gyrus gamma bursts were more abundant during inspiration than expiration while in the rest of the rats gamma was distributed equally across the respiration cycle. In contrast, we found no connection between the cardiac cycle phase and hippocampal oscillations. In experiment 2 we studied the effect of atropine (50 mg/kg, i.p., n = 8), a muscarinic acetylcholine receptor antagonist, on the phenomena detected in experiment 1, using the same measures. Atropine increased breathing rate and abolished theta in the hippocampus in all rats. The occurrence of SPW-Rs was still associated to slowing down of respiration, while the link between respiration phase and dentate gyrus gamma bursts was abolished by atropine. These results suggest that cholinergic signaling via muscarinic receptors regulates breathing and hippocampal state and is responsible for the possible link between respiration cycle phase and hippocampal function. Future studies should address the relevance of these findings in naturally sleeping rats, using more selective manipulation methods.

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P1514 / #2202

Topic: AS10 Sleep, Biological Rhythms

SLEEP IS BI-DIRECTIONALLY MODIFIED BY AMYLOID BETA OLIGOMERS

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Disrupted sleep is a major feature of Alzheimer's Disease (AD), often arising years before symptoms of cognitive decline but how increased Aβ burden leads to disruptions in sleep remains unknown. We hypothesised that Aβ may be regulating sleep independently of neuronal cell death. To test this, we took advantage of the ability to directly deliver Aβ to the brain of larval zebrafish. We developed a high-throughput in vivo injection assay in which the amount and type of the Aβ oligomers can be controlled. We generated Aβ with different oligomeric sizes and then assessed how each Aβ preparation affected sleep and wake in zebrafish using automated video-monitoring. We then used *in situ* hybridisation for neuronal activity markers to identify cells that respond to the short and long Aβ oligomers. We generated CRISPR-mediated knockouts of putative Aβ receptors and in the zebrafish orthologs of Amyloid Precursor Protein (APP) to uncover the mechanisms of Aβ-induced sleep alterations. We demonstrate that Aβ acutely and reversibly enhances or suppresses sleep as a function of oligomer length independently of neural death. Genetic disruptions revealed that short Aβ oligomers induce acute wakefulness through Adrenergic receptor b2 (Adrb2) and Progesterone membrane receptor component 1 (Pgrmc1), while longer Aβ forms induce sleep through a Prion Protein (PrP) signalling cascade. We also demonstrate that short and long Aβ oligomers induce opposing changes in neuronal activity consistent with the behavioural results. We also show that zebrafish produces endogenous Aβ and larvae that lack *appb* have reduced sleep. Our data indicate that Aβ participates in a bi-directional sleep/wake switch. Alterations to the brain's Aβ oligomeric milieu, such as during the progression of AD, may therefore disrupt sleep via changes in acute signalling events. Thus, our data provides an explanation of the well-observed associations of abnormal sleep patterns with AD risk.

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Topic: AS10 Sleep, Biological Rhythms

ROLE OF LAYER 5 CORTICAL SYNCHRONY IN GENERAL ANESTHESIA

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General anesthetics act through diverse pharmacological mechanisms to induce a similar loss of consciousness. Recent correlational findings suggest that the common neurophysiological feature driving the loss of consciousness accross anesthetics may be a state of brain-wide synchrony that selectively involves layer-5 pyramidal neurons accross the entire cortex. However, causal evidence to support or refute this hypothesis is still lacking. To address this issue, here we combined chemogenetic manipulation with *in vivo* electrophysiology in mice under the effect of inhaled isoflurane. The levels of administered anesthesia were modulated during the electrophysiology recording sessions in order to induce a global, cortex-wide change of neuronal synchronization. Chemogenetic manipulation was performed using DREADDs and was restricted to a fraction of layer-5 pyramidal neurons in the motor cortex. We found that changing the levels of administered anesthesia modifies the activity of layer-5 cortical neurons, with an expected variation in the depth of unconsciousness. We also showed that chemogenetic depolarization of layer-5 pyramidal neurons of the motor cortex increases their activity and dramatically disrupts their synchrony between hemispheres, but is not sufficient to reverse the loss of consciousness nor to reduce the depth of anesthesia. These results suggest that cortex-wide synchrony of layer-5 pyramidal neurons is not a necessary feature for the loss of consciousness induced by general anesthesia.

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