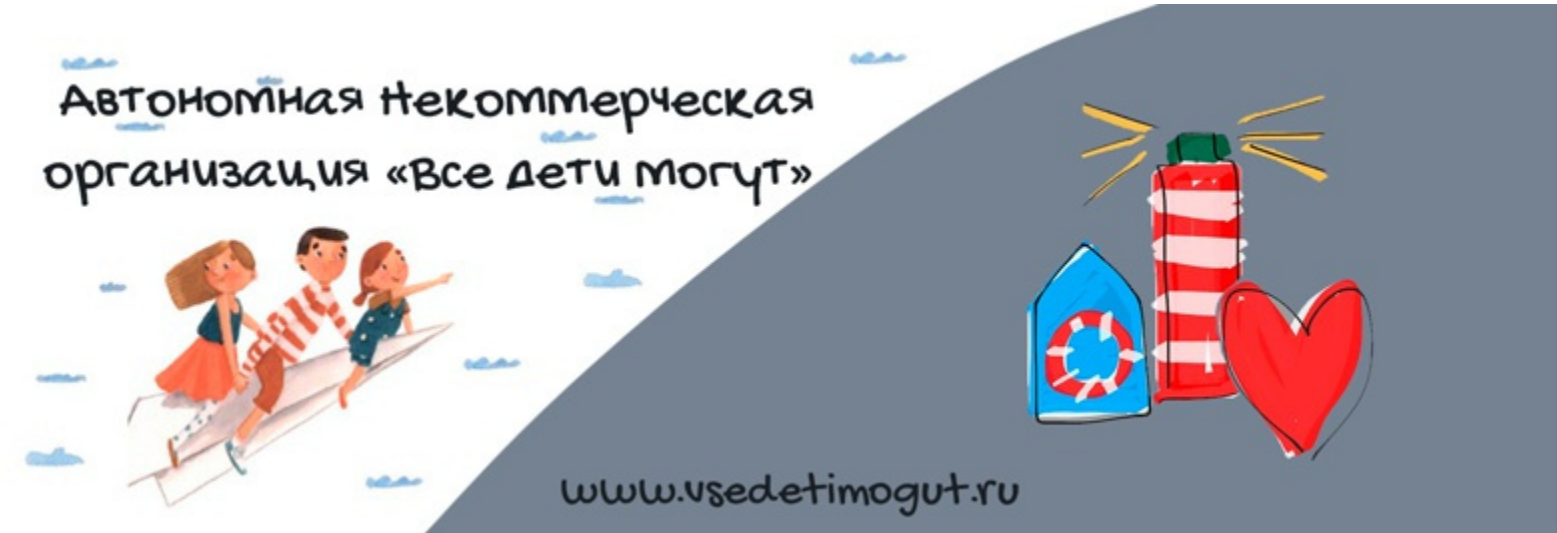
# Характер сна у взрослых пациентов с церебральным параличом



# Sleep pattern in adults patients with cerebral palsy

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## Sleep pattern in adults patients with cerebral palsy

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*Introduction:* Patients with cerebral palsy (CP) are in risk to sleep respiratory disorders, due to the presence of neuromuscular alteration. There are many studies evaluating sleep pattern using questionnares in children, but very few using polysomnography (PSG) in adults with CP. The aim of this study was evaluate the sleep pattern in adults patients with CP through PSG.

*Materials and methods:* 22 patients with diparegic CP,11 female and 11 male, mean neck circumference 35.4±2.6, mean age 26.9±5.8 and mean BMI 22.0±3.63. All CP patients were recruited from the Training Program in Dentistry for Persons with Disabilities, UNESP-SP-Brazil. Inclusion criteria were patients with partially preserved cognition function and ability to respond to verbal commands. Patients or caregivers that did not sign the informed consent did not participate. All patients underwent the PSG exam at Sleep Disorders Laboratory- UNINOVE University-SP-Brazil. After the habituation night, the PSG for the evaluation of sleep pattern was performed. This study was approved by the Human Research Ethics Committees of UNESP/SJC, n. 25000.058696/2010–74.

*Results:* 45.0% (10) patients presented obstructive sleep apnea (OSA). Mean sleep stage 1,2,3,and REM were 35%, 41%,10% and 14% respectively. Mean oxyhemogloblin median and minimum were 96% and 91% respectively. The mean total sleep time was 210 min. Many patients had difficulty to sleep during PSG.

*Conclusion:* This the first study to evaluate the sleep pattern, through PSG exam, in a group of adults with CP. The difficulty to sleep, represented for short total sleep time, may be due to neuromuscular disease characteristics. Although, even with a short sleep time, 45% of patients presented OSA. Short-sleep patterns and OSA may interfere with habilitation activities and community adjustment in this population. Diagnose of sleep pattern should be taken as early as possible to achieve improvement of their quality of live.

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## Increased cortical arousal propensity in opiate users with complex sleep apnea syndrome

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*Introduction:* Complex sleep apnea syndrome (CompSAS) presumably involves unstable ventilatory control mechanisms, possibly including cortical brain arousal indexed by NREM cyclic alternating pattern (CAP) sleep microarchitecture. CompSAS has been associated with opiate use, but may also be idiopathic or associated with underlying cardiac disease. We aimed to determine whether cortical arousal propensity indexed by NREM CAP differed between CompSAS opiate users (OU) and non-opiate users (NOU), and OSA controls.

*Materials and methods:* A retrospective analysis of clinical and diagnostic polysomnographic data of 39 consecutive CompSAS patients (18 OU, 21 NOU) and 18 OSA controls without CompSAS matched for age, gender, body mass index, and polysomnographic apnea-hypopnea index (AHI) was performed. Polysomnograms were manually analyzed for CAP and log transformed CAP A Ratio Index (ARI, with higher values indicating higher sleep-preservation propensity) according to standard methods using Hypnolab scoring software (ATES Medica Labs, Verona, Italy). Groups were compared utilizing Wilcoxon Rank Sum tests, and multivariable regression was performed to determine associations between predictor variables and CompSAS.

*Results:* AHI ($p = 0.66$) and arousal indices ($p = 0.42$) were similar between OU, NOU, and OSA controls. CAP rate was lower in OU than NOU or OSA controls (66 vs. 77 vs. 77, $p = 0.13$). OU had lower CAP A1 and higher A2 indices (A1: 52 vs. 94 vs. 88, $p = 0.096$; A2: 39.7 vs. 24.0 vs. 21.1, $p = 0.068$), resulting in a signifi cantly lower ARI in OU ($p = 0.02$) with ARI below 0.55 associated with CompSAS ($p = 0.0026$).

*Conclusion:* CompSAS OU demonstrated a higher cortical arousal propensity when compared to CompSAS NOU and OSA controls, resulting from reduced A1 (slow, sleep promoting) and higher A2 (fast, sleep fragmenting) CAP rhythms. Our fi ndings suggest that opiates alter cortical arousal mechanisms that could induce central apnea, possibly causing postarousal/sleep-onset central apneas during positive airway pressure treatment.

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