

## **Sleep cycling alternating pattern (CAP) expression is associated with hypersomnia and GH secretory pattern in Prader-Willi syndrome**

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Background and purpose: hypersomnia, sleep disordered breathing and narcoleptic traits such as rapid eye movement (REM) sleep onset periods (SOREMPs) have been reported in Prader-Willi syndrome (PWS). In a group of young adult patients with genetically confirmed PWS we evaluated sleep and breathing polysomnographically, including cycling alternating pattern (CAP), and we analyzed the potential interacting role of sleep variables, sleep-related breathing abnormalities, hypersomnia, severity of illness variables and growth hormone (GH) secretory pattern.

Patients and methods: eleven males and 7 females (mean age:  $27.5 \pm 5.5$  years) were submitted to a full night of complete polysomnography and the multiple sleep latency test (MSLT). GH secretory pattern was evaluated by a standard GH-releasing hormone plus arginine test. Sixteen non-obese healthy subjects without sleep disturbances were recruited as controls.

Results: compared to controls PWS patients showed reduced mean MSLT score ( $P < 0.001$ ), reduced mean latency of sleep ( $P = 0.03$ ), increased REM sleep periods ( $P = 0.01$ ), and increased mean CAP rate/non-rapid eye movement (NREM) ( $P < 0.001$ ). Only four PWS patients had apnea/hypopnea index (AHI)  $\geq 10$ . Conversely, significant nocturnal oxygen desaturation was frequent (83% of patients) and independent from apneas or hypopneas. In the PWS group, CAP rate/NREM showed a significant negative correlation with MSLT score ( $P = 0.02$ ) independently from arousals, respiratory disturbance variables, severity of illness measured by Holm's score or body mass index (BMI). PWS patients with CAP expression characterized by higher proportion of A1 subtypes presented less severe GH deficiency ( $P = 0.01$ ).

Conclusions: Our study suggests a relationship between hypersomnia and CAP rate, and between CAP expression and GH secretory pattern in PWS, possibly reflecting underlying central dysfunctions.

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