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QUANTITATIVE ELECTROPHYSIOLOGY DURING NIGHT- AND DAYTIME INCLUDING ELECTROENCEPHALOGRAPHY AND EVENT-RELATED POTENTIAL EEG/ERP TOPOGRAPHY AND TOMOGRAPHY (LORETA) IN HYPERTHYROIDISM S. Rosales-Rodriguez<sup>1,2</sup>, B. Saletu<sup>2</sup>, P. Anderer<sup>1</sup>, B. Ludvik<sup>3</sup>, G.M. Saletu-Zyhlarz<sup>1,2</sup> <sup>1</sup>Department of Psychiatry and Psychotherapy, Medical University of Vienna, <sup>2</sup>Institute of Sleep Medicine, Rudolfinerhaus, <sup>3</sup>Department of Internal Medicine III, Medical University of Vienna, Vienna, Austria

Introduction: In contrast to the abundance of visual qualitative EEG reports in pathoendocrinology, there is a paucity of quantitative EEG findings.

Objectives: Electrophysiology may be utilized for differential diagnosis of hypersomnolence. Aims: To investigate long-term daytime sleepiness in a young female neurologist with the tentative diagnosis of narcolepsy in addition to moderate depression/anxiety and congenital thyroid hypoplasia treated with thyroxin.

Methods: Three-night polysomnography, multiple sleep latency test (MSLT), visual EEG/ERP-mapping, LORETA, psychometrics and blood analyses were performed. Results: Polysomnography revealed normal sleep efficiency and sleep architecture, but a high arousal index of up to 63/h TST. The MSLT showed a shortened mean sleep latency of 3.7 min. without REM-sleep onsets, objectifying the high Epworth Sleepiness score of 18. Visual EEG evaluation exhibited a fast alpha rhythm with intermittent theta and delta intrusions and paroxysmal activities. EEG-mapping showed an accelerated dominant frequency and ubiquitous increase in absolute (especially delta and beta) power, ERP revealed shortened N1 latency and very high amplitudes in all components (P300 > 5 SD). LORETA demonstrated significant regional increases in delta, alpha-2 and beta-1 power in the anterior cingulate, orbital, ventromedial, dorsolateral prefrontal cortex and temporal cortex, predominantly right hemispherically. Psychometry showed increased anxiety (SAS) and depression (SDS) and reduced quality of life. Finally, hormonal analysis pointed to thyrotoxicosis factitia (ICD-10 E05.4).

Conclusion: Diagnostic investigations clearly elucidated the pathogenesis of the presenting diagnosis of organic hypersomnia triggered by increased sleep-microarousals due to thyrotoxicosis factitia. Discontinuation of hormone substitution led to a normalization process that will be discussed.