SLEEP INSTABILITY IN FRONTOTEMPORAL DEMENTIA

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Objective:

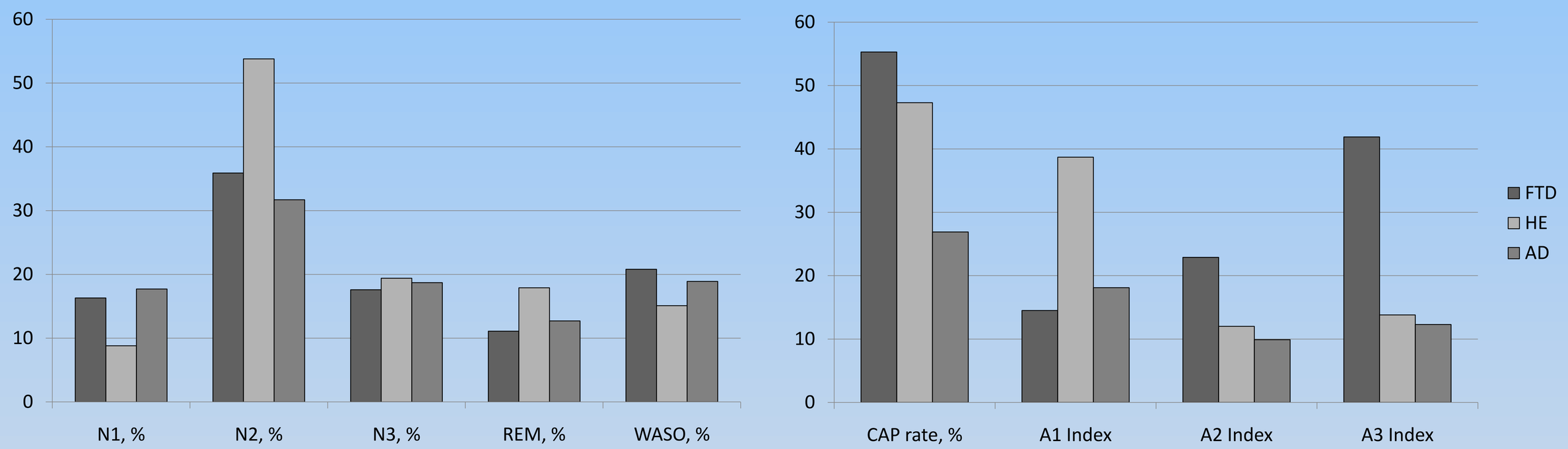
Frontotemporal dementia (FTD) represents a major issue among neurodegenerative dementias, particularly in early cases. Although sleep disorders significantly impair patients' and caregivers' quality of life in FTD, as well as in other forms of neurodegenerative dementias, such as Alzheimer 's Disease (AD), polysomnographic (PSG) reports are very few in literature. Aim of our study was to investigate sleep microstructure, by means of Cyclic Alternating Pattern (CAP), in FTD.

Methods and materials:

Ten behavioral variant FTD (6 M, 4 F; mean age 61.2 ± 7.3 years; disease duration: 1.4±0.7 years) underwent nocturnal sleep-lab based PSG for the evaluation of nocturnal sleep architecture and CAP parameters. Data obtained were compared with the relevant parameters obtained from age- and sex-matched mild to moderate AD, and cognitively intact elderly controls.

Results:

Compared with healthy controls, nocturnal sleep was at least as much impaired as observed in AD, and in a shorter disease duration, with decreased total sleep time and REM sleep, and increased light sleep.
CAP analysis pointed out increased sleep instability (CAP rate) for FTD, and a CAP disruption that most prominently involved slow wave activity related phases, with significantly decreased A1 index.



Sleep macrostructure and microstructure in the three groups. N1=N1 NREM; N2=N2 NREM; N3=N3 NREM; WASO=Wake after sleep onset. CAP rate=CAP time/NREM sleep x 100; A1 Index=Number of A1 per hour of NREM sleep; A2 Index=Number of A2 per hour of NREM sleep; A3 Index=Number of A3 per hour of NREM sleep. Sleep stages are reported as percentage of sleep period time.

Discussion and conclusions:

Sleep in FTD appears at least as much disrupted as observed in AD, and in a shorter disease duration. Sleep impairment is obvious at both macrostructural and microstructural level, and may be specifically related to the specific frontal lobe involvement in the neurodegenerative process. The pattern of alterations seems somewhat peculiar, probably due to the anatomical distribution of neurodegeneration, with a major impact on frontal lobe generated sleep transients and a substantial sparing of posterior cortex – related phenomena.
These data suggest the potential role of sleep parameters, disrupted from the early phases of the disease, as in-vivo biomarkers, and confirm the importance of correctly address sleep, and its disorders, in individuals with neurodegenerative diseases.

References

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