AGE RELATED CHANGE ON NREM SLOW OSCILLATIONS REBOUND AFTER SLEEP DEPRIVATION

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Introduction: The hypothesis that aging is associated with alterations in the build-up function of the homeostatic process is still a matter of debate. Higher homeostatic pressure leads to higher neuronal synchronisation in NREM sleep, that is detectable by electroencephalography (EEG) as low-frequency, high-amplitude waves (slow-wave oscillations; SWO). SWO are proposed as a maker of synaptic strength homeostasis and to play a crucial role in memory and brain plasticity. However, the impact of aging on SWO rebound after sleep deprivation is unknown.

Methods: Sixty-one healthy volunteers with no sleep disorders were separated in two groups: 29 Young (14 M, 15 W; 27 y ± 5), and 32 Middle-aged (14 M, 18 W; 52 y ± 5). Each subject participated in a baseline nocturnal sleep and a daytime recovery sleep (after 25-hour of wakefulness). SWO detection was performed on artefact free sections of NREM sleep for Fp1, F3, C3, P3, and O1 (linked-ears), with an automatic algorithm, using the following criteria: 1) Negative peak <-40 uV; 2) Peak-to-peak amplitude >75uV; 3) Duration of negative deflection > 125 ms and < 1500 ms; and 4) Duration of positive deflection < 1000 ms.

Results: Three-way ANOVAs (Factors: Age group, Sleep condition, Derivation) were performed on SWO peak-to-peak amplitude (uV) and density (number of SWO/minutes of NREM sleep). Middle-aged subjects showed lower SWO density and SWO amplitude compared to the young, during both sleep condition. Enhanced homeostatic pressure increased both SWO density and SWO amplitude during recovery sleep. This effect varied with cerebral topography and with age group for SWO density only. The increase in SWO density after sleep deprivation was more prominent in anterior derivations in both age group, and the rebound in SWO density was more important in young subjects compared to middle-aged subjects.

Conclusion: Previous results showed a frontal dominance of slow wave activity rebound following sleep loss, suggesting that these brain areas may necessitate more intense recovery. Our results show that this anterior dominance following sleep deprivation is linked to differential enhancement in the number of SWO rather than to an increase in their amplitude. Also, compared to the young, middle-aged subjects showed a reduced ability to increase SWO density but not SWO amplitude during recovery sleep after sleep loss. Together, these results suggest that SWO amplitude and SWO density are regulated by different mechanisms.