Effects of aging and daytime recovery sleep on N-REM slow oscillations


(1) Centre d’étude du sommeil et des rythmes biologiques, Hôpital du Sacré-Cœur de Montréal, Québec, Canada
(2) Département de psychologie, Université de Montréal, Montréal, Québec, Canada

The hypothesis that aging is associated with alterations in the build-up function of the homeostatic process is still a matter of debate. Most knowledge on how age modulates the effects of sleep deprivation on NREM sleep synchronization comes from visual scoring of sleep stages and quantitative sleep EEG (e.g. spectral analyses). Spectral analysis provides important indices on sleep EEG synchronization but it does not allow identifying N-REM sleep EEG oscillations per se. We used an automatic algorithm to assess the effects of age, sleep loss and topography on N-REM sleep slow oscillations (SO; >75 µm). Twenty-four healthy volunteers with no sleep disorders were separated in two groups: Young (6W, 6M; 24.2y ± 3.3), and Middle-aged (6W, 6M; 53.8y ± 3.7). Each subject participated in a baseline nocturnal sleep and a daytime recovery sleep (after 25-hour of wakefulness). SO detection was performed on artefact free sections of NREM sleep for Fp1, F3, C3, P3, and O1 (linked-ears), with an automatic algorithm using published criteria (Massimini et al. 2004). Three-way ANOVAs (Factors: Age group, Sleep condition, Derivation) were performed on SO amplitude and density (nb/min). Compared to baseline sleep, SO amplitude increased during daytime recovery sleep and this effect was more prominent in young compared to older subjects in FP1. SO density was higher during daytime recovery sleep compared to baseline sleep in both age groups and this effect was stronger in Fp1 and F3. Results are in line with the notion that older subjects have a reduced ability to increase sleep synchronisation following sleep deprivation, particularly in anterior derivations. Interestingly, age-related difference in the effects of the sleep deprivation was observed on SO amplitude only, and not on SO density. This may be explained by age-related decline in the capacity to synchronize larger neuronal populations after sleep deprivation.
This research was supported by scholarships from the Canadian Institutes of Health Research (CIHR), and grants from CIHR, the Fonds de la Recherche en Santé du Québec (FRSQ) and the Natural Sciences and Engineering Research Council of Canada (NSERC).

Key words: slow oscillation, aging, sleep deprivation

Literature Cited