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MODELING SLOW WAVE ACTIVITY PATTERNS ACROSS THE SCALP

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INTRODUCTION

In models of sleep regulation, timing of sleep is linked to the temporal dynamics of slow-wave activity (SWA, EEG spectral power in ~0.75–4.5 Hz range) in the cortical NREM sleep EEG. In the original two process model of sleep regulation, SWA was used as an indicator of sleep debt. The time constant of the SWA decrease over consecutive NREM–REM sleep cycles was used to estimate the time course of process S during sleep[1]. Later, SWA expression was assumed to reflect the decay rate of process S rather than sleep debt directly. A detailed model describing the overnight SWA pattern with its intrusions of REM sleep and waking was made by Achermann[2], refining the description of the changes in SWA and process S over the night. In this model, process S and SWA are coupled by a 'gain constant', quantifying the efficiency of SWA in dissipating S.

The time course of SWA during sleep after sleep deprivation varies between cortical locations[3], suggesting that local differences in process S exist. In this study, we estimate local differences of S regulation by fitting 'gain constants' for 26 locations on the scalp. We observed higher frontal 'gain constants', suggesting that indeed regulation of S has local differences.

METHODS

Nine healthy young subjects (18–28 years) participated. Subjects did neither smoke nor use drugs, and abstained from consumption of alcohol and coffee throughout the experiment. They did not rate as extreme morning or evening types. The experiment was approved by the Medical Ethics Committee of the Academic Hospital of the University of Groningen. Subjects signed an informed consent form.

Subjects had a habituation sleep night and a baseline sleep night in the. Only baseline sleep EEGs were used here. Before sleep nights, subjects were doing their normal daily routine, until they came to the sleep laboratory at 20:00. After application of scalp electrodes subjects were asked to perform computerized test series of ~35 min duration at 22:00 and 23:00. The test series contained questionnaires and visual event related potential trials. Subjects prepared for sleep at 23:40 and went to bed around 23:55, after which the electrodes were connected to the EEG amplifiers. At 00:00 hours, lights were turned off until 08:00 hours the next morning.

EEGs were recorded using a cap system with Ag/AgCl electrodes (Electro-Cap International, Inc., Eaton, Ohio, USA), on 26 positions on the scalp (F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, P9, P10, PO7, PO8, O1, Oz, O2, PO9, PO10, O9, O10). The left earlobe was used as reference, and the inion was used as ground. Data were amplified

(500µV/V) band-pass filtered between 0.16–30Hz and sampled at 100 Hz. Besides EEGs, also EOG of the right eye was obtained, and EMG was measured on neck muscles. EEGs were scored for wake, movement time, REM sleep and NREM sleep on a 30 second basis, using standard criteria[4]. For quantification, the EEG analysis program BrainVision (Brain Products, Germany) was used. NREM sleep EEG was screened for artefacts in 3 sec intervals, clean EEG epochs were Fourier transformed, and spectral power for the 2-3Hz EEG frequency bin was averaged over the particular 30 sec interval.

The SWA/S modelling method was done according to the description of Achermann[1], with minor modifications as explained in Zavada et al[5]. EEG power data of each of the 26 derivations were entered into the SWA fitting model together with the vigilance state information to estimate the 'gain constant' for the 26 locations. To visualize scalp maps of 'gain constant' values and to carry out tests for topographic differences (TANOVA), LORETA software was used[6].
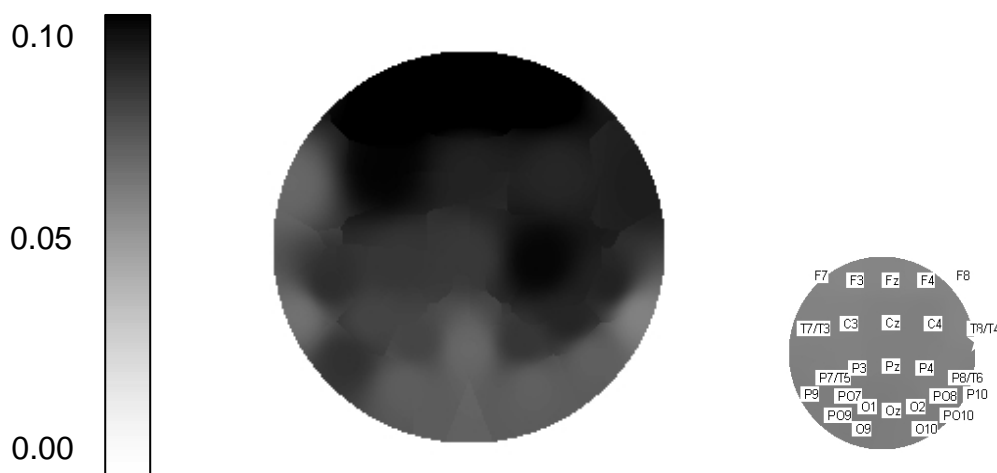

Figure 1. Distribution map of the average 'gain constant' of 26 derivations on the scalp (n = 6–9). Grey intensity ranging from 0.00 to 1.00. Derivations are indicated in the lower right corner. The emerging pattern is characterized by higher 'gain constants' predominantly in frontal areas (see text).

RESULTS AND DISCUSSION

Failures to fit occurred in 11 individual cases (subject-derivation combinations), 10 for parietal/occipital one for temporal and none in frontal and central derivations Failures typically occurred when the SWA pattern did not show a gradual decrease over consecutive NREM/REM cycles. Figure 1 shows the scalp map of average 'gain constants' (n = 6–9) . 'Gain constant' values are larger in frontal derivations compared to parietal/occipital derivations, deviating from the average gain constant (TANOVA: $p < 0.01$). Sleep regulation on parietal/occipital locations appears to deviate from that in frontal areas, sometimes to the extent that the simulation model is not capable to process the data.

Thus, sleep regulation differs across cortical locations. As a consequence, there must be location dependent (SWA related) differences in sleep processes. Since Process S was invented to describe sleep behaviour, multiple processes S are not possible. We propose another name for the sleep processes described with the gain constant: Process Z. This variable solely describes the SWA related aspects of sleep regulation, allowing for local

differences in sleep processes. It can be further investigated which 'process Z' can explain sleep behaviour best, thereby linking (locally different) brain related functional sleep processes to sleep behaviour.

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