High-frequency EEG activity in insomnia, a technical and clinical note

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INTRODUCTION
Activity in the EEG signal with frequencies over 30 Hz is called ‘fast-frequency EEG activity’ or ‘high-frequency EEG activity’ or ‘40 Hz EEG activity’.1,2 It has been postulated that fast frequency activity during sleep plays a role in the process of ‘sensory gating’, resembling the state of consciousness during sleep3,4 and that it is also produced in the thalamocortical feedback loops which generate the sleep spindles.5 Abnormalities in fast frequency EEG activity in insomnia might therefore be of theoretical and practical interest. This paper presents the results of a pilot study that was meant to:

a) quantify fast-frequency EEG activity (30-100 Hz) in the sleep recordings of insomnia patients;
b) study the time course of this activity over the night and to test whether differences exist between the first and the second night of recording;
c) correlate fast-frequency EEG activity with other sleep parameters.

PATIENTS AND METHODS
Sixteen insomnia patients (age range 22-60 years; median 43 years; 8 women) were studied. All patients participated in an ongoing multicenter study of the effects of the drug Mirtazapine on insomnia. According to the protocol, the patients did not use any medication with known effects on the central nervous system (except for placebo) in the first two nights of the study. These two nights were used in the present study. Insomnia was defined according to the DSM IV criteria taking into account the results of polysomnography (PSG) during the first two nights. At least two of the following criteria applied: sleep onset latency was 30 minutes or longer, total sleep time was 6.5 hours or less, there were three or more awakenings. OSAS, parasomnias, circadian rhythm disorders, PLMD or other medical/psychiatric diseases with influence on sleep were excluded on the basis of anamnestic data and PSG during the first night. Sleep stages were scored according to the Rechtschaffen and Kales rules. Sleep onset latency (SOLAT) and sleep efficiency index (SEI) were taken as parameters for objective sleep quality. For both nights the Groningen sleep quality questionnaire6 and the fatigue score from the Profile Of Mood States (POMS) subjectively assessed sleep quality and fatigue the next day.

Previous studies7-9 revealed that high-frequency EEG activity has highest power during REM sleep. According to these findings, and in order to get insight in changes in fast activity during sleep, we analyzed from each night the first five minutes of EEG from the first and last REM period, as well as the first five minutes of the first and last period of deep non-REM sleep (SWS, NREM3 or 4). The advantage of choosing REM and SWS is that muscle activity is at a low level and artifacts from muscle activity will be small. Using Fourier analysis of the 4*5 minutes of EEG (C3-A2 derivation, sample frequency 200 Hz) the amplitude in microvolts/square root Hz at 30 and 55 Hz was measured. These frequency bins were chosen as we consider them representative for fast EEG activity. Intranight correlations between these eight parameters and the four clinical and PSG criteria outlined above were analyzed, as were the changes in fast activity from the first to the second night in relation to the changes in the clinical parameters.

All combinations were analyzed using the Spearman rank correlation test (two-sided). A value of p=0.05 or lower was considered to be significant.

RESULTS
All 16 patients had a (first) REM period during both nights, i.e. 32 first REM epochs were available for analysis. Due to lack of slow-wave sleep (SWS) or second REM periods, only 31 first SWS epochs, 19 last SWS epochs at the end of the night and 29 last REM epochs could be identified. Nearly all records contained visually detectable high-frequency EEG activity (an example is presented in Figure 1). Epochs of five minutes duration that were (muscle) artifact free at visual inspection and their corresponding spectra were easily obtained. Comparison of shorter epochs with and without eye movements learned that (rapid) eye movements did not contribute substantially to the fast-frequency range of the spectra. EEG amplitude at 30 and 55 Hz was low in all patients. The values ranged from 0.9-3.8 microvolts/square root Hz. For an example of a spectrum, see Figure 2. The amplitude values varied randomly within this range for first and last REM and deep sleep periods of the same night and in comparisons between both nights.

The results of the Groningen sleep quality questionnaire taken after the first night correlated significantly with the amplitude values for the first SWS: 30 Hz (r=0.52), first REM: 55 Hz (r=0.63) and second REM: 30 Hz (r=0.53), measured during the first night. The highest amplitude values were found in those patients who were the most satisfied about the quality of sleep during the same night. Night-to-night changes in the Groningen sleep quality score correlated significantly with the night-to-night differences for amplitude values at 30 Hz (r=0.56)
and in a direction opposite to that reported previously. Although there were many similarities in methods and patients, some differences exist between both studies. Our choice of amplitude values at specified frequencies instead of broadband EEG parameters might explain the discrepancy. Furthermore, some doubts may exist on the cerebral origin of all high-frequency activity in the (EEG) channels that were analyzed. Muscle activity might contaminate the results of either study. We tried to eliminate such artifacts by limiting our study to REM and SWS, both characterized by low muscle interference, and by discarding epochs that were still suspected of being contaminated with muscle activity.

We see our present study as preliminary to further investigations in the field of high-frequency EEG activity in sleep- and wake disorders. The technical aspects as outlined above will be explored. Furthermore, normal subjects and patients with other sleep- and wake disorders will be studied. We hope that these investigations will finally provide some insight in brain mechanisms underlying insomnia.

REFERENCES