学 位 論 文 の 要 旨

氏 名 松本 貴久

学 位 論 文 名 Effect of Risperidone on Polysomnography in Healthy Subjects

発 表 雑 誌 名 (巻, 初頁～終頁, 年) Sleep and Biological Rhythms (in press)

著 者 名 Takahisa Matsumoto, Soichi Mizuno, Junya Tsukada, Yasushi Inami, Jun Horiguchi

論 文 内 容 の 要 旨

INTRODUCTION

Atypical antipsychotic drug, risperidone, is a very potent antagonist of central 5-hydroxytryptamine-2 (5-HT_2) and dopamine D_2 receptors. Only two polysomnographic studies have reported the effects of risperidone on sleep. This study aimed to determine the effect of risperidone on polysomnography (PSG) in healthy subjects.

MATERIALS AND METHODS

Ten healthy, young male volunteers (mean age, 22 ± 3.7 years; range 19–31) participated in this study. None of the subjects had any history of sleep disorders or any other form of mental and somatic illness. All the subjects gave informed written consent after being explained the risks and benefits before undergoing this study. This study was approved by the Medical Ethics Committee of Shimane University School of Medicine.

Each volunteer was asked to reach the sleep laboratory at 9 pm for PSG monitoring. The subjects spent three successive nights in the laboratory. The first was the adaptation night and the second was the baseline night. On the third night, the volunteers received risperidone (1 mg tablet, per os) 30 minutes before the PSG recording. We evaluated the PSG parameters according to the standard criteria. To investigate the distribution of sleep parameters, the sleep period time (SPT) was divided into three equal periods, and each mean value of sleep parameter was calculated separately for each period.

RESULTS AND DISCUSSION

Following risperidone administration, we observed a significant increase in the percentage of stage 2 sleep to SPT (baseline, 58.5 ± 6.3%; risperidone, 65.3 ± 5.9%; $p<0.05$) and a significant decrease in the percentage of rapid eye movement (REM) sleep to SPT (baseline, 23 ± 2.3%, risperidone 17.3 ± 5%; $p<0.05$). No other significant changes were noted.

During the first period, risperidone significantly decreased ($p < 0.05$) stage 1 sleep and increased ($p < 0.05$) stage 2 sleep. During the second period, risperidone administration significantly increased ($p < 0.05$) stage 2 sleep. During the third period, it made no significant change in stage 2 sleep.

Recently, the effects of atypical antipsychotic drugs on sleep has been

investigated. Lee et al. reported that clozapine improved sleep continuity and increased stage 2 sleep in patients with schizophrenia. Sharply et al. demonstrated that olanzapine increased slow wave sleep (SWS) in healthy subjects. Cohrs et al. reported that quetiapine increased total sleep time, sleep efficiency, stage 2 sleep, and subjective sleep in healthy subjects. Yamashita et al. reported that the duration of SWS was significantly longer in patients with schizophrenia treated with risperidone than in those treated with haloperidol.

Sharply et al. reported that risperidone administration showed a significant decrease in REM sleep and a trend toward increased stage 2 sleep in their previous study. The present study demonstrated that a single dose of risperidone (1 mg) significantly increased stage 2 sleep and decreased REM sleep in 10 healthy subjects.

We clarified that this change in the stage 2 sleep was obtained during the first and second periods of sleep, and not during the third period. Moreover, we demonstrated that risperidone decreased stage 1 sleep during the first period of sleep.

CONCLUSION

The present data are new findings with respect to the effect of risperidone treatment on PSG. Our study suggests that risperidone treatment improves the quality of sleep in schizophrenia.