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UNILATERAL VISUAL STIMULATION REDUCES REM SLEEP LATENCY

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INTRODUCTION

Sleep is hypothesized to play an important role in brain recovery. The necessity to recover is thought to be use dependent. Longer use by elongated waking results in intensified sleep, as can be measured by higher slow wave activity in the non-rapid-eye-movement (NREM) sleep EEG. Increased use of specific brain areas results in locally intensified sleep in those specific brain areas (Kattler et al. 1994).
The visual cortex is known to be able to increase its metabolism up to 40% with specific checkerboard stimulation (Fox & Raichle 1984). We used checkerboard stimulation to investigate the effects of enhanced visual cortex activity on sleep, and report here on the effects on sleep architecture.

METHODS

The study was carried out with 10 healthy young male (5) and female (5) subjects (18-25 years). The subjects did not smoke nor use drugs, and refrained from consuming alcohol and coffee throughout the experiment. Subjects scored as normal chronotype. Subjects signed an informed consent. The study was approved by the Medical Ethics Committee of the Academic hospital of the University of Groningen.
Subjects were asked to schedule their sleep between 00:00 and 08:00, starting 3 days before the experimental procedure in the lab began. Subjects were habituated to sleeping in the lab on day 1 and were scheduled to sleep in the lab from 00:00 to 08:00. On day 2, subjects slept in the lab between 00:00-08:00 after a day of normal activity (Baseline). On day 3, test sessions with unilateral checkerboard stimulation were performed every hour between 09:00 and 23:00. Subsequently, people were allowed to sleep between 00:00-08:00 (Recovery).
Visual stimulation during test sessions was carried out by means of an inverting checkerboard on either the left or right half of a 17" computer monitor, aiming for stimulation of the visual cortex of the contra-lateral brain hemisphere. Subjects were positioned at 35 cm from the screen, corresponding to an angular exposure of ~24º. Subjects were asked to focus on a plus sign in the centre of the screen.
A checkerboard inverting 4 times per second was presented on either the left (5 subjects) or the right (5 subjects) half of the screen. During part of the visual stimulation, subjects performed screen related tasks to ensure attention to remain focussed on the screen. The stimulation time amounted to 12 minutes per test session.

Sleep architecture was assessed using EEGs measured with a 28 Ag/AgCl electrode cap system, which has been described elsewhere (Strijkstra et al. 2003). Sleep EEGs were scored for vigilance states using standard scoring procedures. REM sleep latency was calculated as time between first sleep onset (NREM sleep stage 2) and first REM.

RESULTS AND DISCUSSION

Table 1 shows descriptive data of the sleep patterns during baseline and recovery sleep. Sleep onset, total sleep time, total NREM sleep time, total REM sleep time and movement time was not statistically different between baseline and recovery nights. REM sleep latency was significantly reduced, i.e. subjects went into REM sleep earlier during the recovery night compared to the baseline night.

Visual stimulation was expected to enhance local brain metabolism in the occipital cortex, and thus induce a local need for additional repair. However, NREM sleep time or percentage was not increased, nor did we observe an increase in the power of EEG slow waves during NREM sleep, including power derived from the EEG traces recorded directly above the visual cortex (data not shown).

With regard to the hypothesis that enhanced local brain metabolism causes enhanced sleep need and thus should be reflected by increased NREM sleep slow wave activity, the apparent lack of NREM sleep related differences may be based on the possibility that a normal day has similar metabolic effects as the visual stimulation procedure in the lab. This can not be easily tested directly. However, there is evidence indicating that our visual stimulation procedure at least causes an increase in subjective sleepiness (Boerema et al. 2003), suggesting that a normal day is less demanding than our test day with stimulation procedure. One might have expected that this change in subjective sleepiness would have had some NREM sleep related counterpart.

The shorter REM latency could possibly be the trivial result of residual habituation effects to the recording procedure, although other signs of residual habituation effects, such as decreases in wake after sleep onset and increases in REM duration, were not found (LeBon et al. 2001).

However, reduced REM sleep latency has also been found as a result of increased visual stimulation and increased visual attention (DeGennaro et al. 1995). Visual stimulation thus may affect sleep architecture via processes related to the increased activity of the visual (attention) systems during prior waking. Possible ‘repair’ following visual stimulation may be reflected in REM sleep related variables such as REM sleep latency, rather than NREM sleep EEG power.

Table 1. Means and standard errors of different sleep variables during baseline and recovery sleep, and P-values of paired t-tests comparing sleep variables between the nights).

	Baseline		Recovery		t-test
	Mean	SEM	Mean	SEM	P-value
Sleep onset (time)	0:18:33	0:06:59	0:20:39	0:04:20	0.3060
1st NREM-REM duration (time)	1:21:00	0:15:06	1:20:03	0:08:30	0.8749
REM sleep latency (time)	0:56:18	0:10:30	0:45:42	0:12:26	0.0444
1st REM duration (time)	0:24:42	0:08:49	0:34:21	0:16:03	0.1343
Fraction REM of 1st cycle	0.303	0.075	0.422	0.166	0.0571
Number of NREM-REM cycli	5.5	0.882	5.65	0.747	0.6849
Total NREM duration	4:28:27	0:54:47	4:38:57	0:48:50	0.4394
Total REM duration	3:03:18	0:53:45	2:59:18	0:45:58	0.8024
Total sleep time (minutes)	436.6	11.350	429.8	18.108	0.2616
Movement time (minutes)	20.05	7.574	22.75	12.488	0.3862
Wake time (minutes)	21.05	5.310	26.35	12.360	0.2291

REFERENCES

1 Boerema,A.S., Van Bunnik,B.A.D., Strijkstra,A.M., Wijers,A.A., Beersma,D.G.M., Daan,S. 2003. Unilateral visual stimulation results in increased subjective sleepiness. In 'Sleep Wake Research in The Netherlands, Vol.14, Van Bemmel A.L., Beersma,D.G.M., Hofman,W.F., Ruigt,G.S.F., Vos,P.J.E. (Eds.), NSWO, Leiden, The Netherlands, pp.15-17.

2 DeGennaro,L., Violani,C., Ferrara,M., Casagrande,M., Bertini,M. 1995. Increase of REM duration and decrease of REM latency after a prolonged test of visual attention. Int.J.Neurosci. 82:163-168.

3 Fox,P.T., Raichle,M.E. 1984. Stimulus rate dependence of regional cerebral blood flow in human striate cortex, demonstrated by positron emission tomography. J. Neurophysiol. 51, 1109-1120.

4 Kattler H., Dijk, D.J. & Borbély, A.A. 1994. Effect of unilateral somatosensory stimulation prior to sleep on the sleep EEG in humans. J. Sleep Res. 3:159-164.

5 LeBon,O., Staner,L., Hoffmann, G., Dramaix,M., San Sebastian,I., Murphy,J.R., Kentos,M., Pelc,I., Linkowski,P. 2001. The first-night effect may last more than one night. J. Psychiatric Res. 35:165-172.

6 Strijkstra,A.M., Beersma,D.G.M., Drayer,B., Halbesma,N. & Daan,S. 2003. Subjective sleepiness correlates negatively with global alpha (8-12 Hz) and positively with central frontal theta (4-8 Hz) frequencies in the human resting awake electroencephalogram. Neurosci. Lett. 340, 17-20.