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Original article

Visual declarative memory is associated with non-rapid eye movement sleep and sleep cycles in patients with chronic non-restorative sleep

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Abstract

Objective: Sleep contributes to processes of memory, but many questions still remain open. The aim of this study was to test the role of different aspects of sleep for memory performance in a group of patients with chronic non-restorative sleep.
Methods: Forty-two consecutive patients (mean age 40.3 years; 31 women) with non-restorative sleep were included. All subjects underwent polysomnography for diagnostic reasons and obtained the following diagnoses (International Classification of Sleep Disorders, ICSD): psychophysiological or idiopathic insomnia ($N = 18$), paradoxical insomnia ($N = 13$), mild hypersomnia ($N = 6$), and dysthymic disorder ($N = 5$). Patients with sleep-related breathing disorders or restless legs were not included. Prior to polysomnography on the second night and the next morning, neuropsychological tests were performed. Declarative memory was tested by the Rey–Osterrieth Complex Figure Test and a paired associative word list. Procedural learning was assessed by a mirror-tracing skill.
Results: Visual declarative memory performance was significantly associated with total sleep time, sleep efficiency, duration of non-rapid eye movement (NREM) sleep and number of NREM–REM sleep cycles, but not with specific measures of REM sleep or slow wave sleep.
Conclusions: Further indications of a role of sleep, and in particular of NREM sleep and sleep organization, for visual declarative memory were found.

Keywords: Sleep disorders; Neuropsychology; Rey–Osterrieth figure test; Polysomnography; Non-rapid eye movement sleep; Sleep cycles

1. Introduction

A growing body of evidence supports the role of sleep for memory processes (for review: [1,2]). However, many questions still remain open, for example whether memory consolidation is linked to a particular sleep stage and whether different types of memory are differentially influenced by the sleep stages (for review: [3,4]). It was found that consolidation of procedural memory is linked to rapid eye movement (REM) sleep [3], and in animal studies it was suggested that the activation of ponto-geniculo-occipital (PGO) waves is part of the mechanism for REM sleep-dependent learning improvement [5]. In contrast, declarative memory was assumed to be associated with slow wave sleep (SWS; [6,7]). These results were obtained by experiments with partial sleep deprivation: sleep in the first half of the night with a high amount of SWS had a different effect on memory consolidation as sleep in the second half of the night with a high amount of REM sleep. Thus, these results

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could be confounded, because encoding and retrieval are tested at different, and also at unusual, times, such as at 02:00 h. In a recent study of whole-night sleep, an association between SWS and visual declarative memory was only found in patients with schizophrenia but not in healthy controls [8].

With regard to more generalized aspects of sleep, the influence of a lesser, but chronic, deprivation of sleep was studied by Van Dongen et al. [9]. A restriction of sleep periods to 6 h per night over 14 consecutive days resulted in significant deficits in working memory performance in young healthy subjects, suggesting an importance of total sleep time for sleep-associated memory processes [9]. Another approach has emphasized the importance of sleep organization and the regular occurrence of non-REM (NREM)–REM sleep cycles for verbal memory consolidation [10–12]. Total sleep time and number of sleep cycles are possibly correlated, but due to a study with experimental manipulations of sleep a better retention of verbal material was associated with the number of sleep cycles but not with duration of either REM sleep or NREM sleep [11]. Recently, sleep spindles – one of the most prominent features of stage 2 sleep – were thought to play a functional role in sleep-related memory processes in humans [13–16].

In patients with sleep disorders such as primary insomnia, studies on the issue of memory functions have shown conflicting results. Whereas few studies have shown an impairment of memory performance in patients with insomnia in comparison to healthy controls [17,18], in most of the studies no significant differences have been found (for review: [19,20]). This might be due to the procedure of recruiting the subjects by advertisements, since these participants have often shown only marginal objective sleep disturbances [19,21].

The aim of this study was to test the role of different aspects of sleep for memory performance. We studied a group of clinical patients with non-restorative sleep presenting a high range of sleep parameters to avoid confounding effects of artificially provoked sleep deprivation. With reference to the guideline "non-restorative sleep" developed by the German Sleep Research Society [22] and to the Diagnostic and Statistical Manual of Mental Disorders, Fourth edition (DSM-IV) [23], non-restorative sleep is described as the feeling that sleep is light, restless or of poor quality. We correlated electroencephalographic (EEG) sleep measurements with memory performance the morning after polysomnography. In particular, the following hypotheses were tested: memory performance should be associated with total sleep time and number of NREM–REM cycles. Additionally, performance in declarative memory should be correlated to the amount of SWS and sleep spindles, and procedural memory to the amount of REM sleep. Since rapid eye movements have been suggested as correlates of PGO waves in REM sleep [24], REM density as a parameter of phasic REM sleep activity should also be associated with memory performance.

2. Methods

2.1. Subjects

We included 42 consecutive patients (mean age 40.3 years, age range 16–67 years; 31 women) with non-restorative sleep, who underwent polysomnography for diagnostic reasons. Mean duration of sleep complaints was eight years (range 1–25 years) and the mean score on the Pittsburgh Sleep Quality Index (PSQI; [25]) was 11.0 (range 3–19). Any relevant medical condition was assessed by medical history, physical examination and routine laboratory investigation including urinary drug screening. Not included in the study were subjects who (a) had a medical disorder (e.g., rheumatoid arthritis or thyroid disease) which compromises sleep, (b) were taking antidepressants or any other psychotropic medication or drugs, (c) had >2 apneas or hypopneas per hour during the first night in the sleep laboratory, or (d) suffered from restless legs syndrome or had a periodic limb movement arousal index >5 per hour. Four patients showed a borderline positive benzodiazepine screening in agreement with reported singular benzodiazepine intake in the week before the study and remained in the study. The patients obtained the following diagnoses according to the International Classification of Sleep Disorders (ICSD) [26]: psychophysiological insomnia ($N = 15$), idiopathic insomnia ($N = 3$), paradoxical insomnia ($N = 13$), mild hypersomnia ($N = 6$; range of mean sleep latency in the multiple sleep latency test (MSLT): 5–9 min), and dysthymic disorder ($N = 5$). All patients gave their informed written consent. The study was approved by the local ethics committee and conformed to the Declaration of Helsinki.

2.2. Procedure

All patients underwent sleep polysomnography for diagnostic reasons for two nights in the sleep laboratory. On both nights, a standard clinical polysomnogram including EEG activity (C3-A2 and C4-A1), electro-oculographic (EOG) activity, submental and lower limb electromyographic (EMG) activity and electrocardiographic (ECG) activity were performed. Chest and abdominal movements, airflow and oxygen saturation were measured only on the first night. In patients with >2 apneas or hypopneas per hour, respiration was again monitored on the second night and they were not included in the study. Patients with complaints of daytime sleepiness additionally underwent a MSLT after