



Impaired memory consolidation during sleep in patients with functional memory disorder

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ABSTRACT

Functional memory disorder (FMD) is characterized by mnemonic and attentional deficits without symptoms of mild cognitive impairment or dementia. FMD usually develops in subjects with high psychosocial stress level and is classified to the somatoform disorders. We assessed memory performance (procedural mirror tracing task, declarative visual and verbal memory task) and other cognitive functions before and after one night of sleep in 12 FMD patients (mean age: 51.7 yrs, 7 females) and 12 healthy subjects matched for age, gender and IQ. Memory performance and other neurocognitive tasks did not differ between the groups at baseline. After one night of sleep, FMD patients showed an impairment of declarative memory consolidation compared to healthy subjects (visual task: $p = 0.004$; verbal task: $p = 0.039$). Spectral analysis of sleep-EEG indicated an increased cortical excitation in FMD. We hypothesize that a hyperarousal state in FMD might contribute to sleep disturbance implicating negative effects on declarative memory consolidation.

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1. Introduction

A number of authors describe that 6–12% of the patients seen in memory clinics complain about everyday memory dysfunctions without fulfilling criteria for mild cognitive impairment or dementia (Almeida et al., 1993; Schmidtke et al., 2007). Typically, patients report on significant mnemonic and attentional deficits in daily living but show normal results in standard neuropsychological tests. Recent research suggests that these symptoms might form an entity in its own right (Manes et al., 2008; Derausne et al., 1999; Barker et al., 1994). While some authors have preferred the term “memory complainer”, more precise criteria for this group of patients have been developed and a diagnostic entity called “functional memory disorder” (FMD) has been suggested (Schmidtke et al., 2008; Metternich et al., 2009, see Table 1). These criteria are congruent with the definitions of functional memory complaints/disorder suggested by other authors (see Ponds et al., 1995; Berrios et al., 2000)

According to a recent description FMD has been conceptualized as “an acquired medical and psychological condition with significant failure of memory and concentration that occurs in daily living, is unrelated to organic factors, and is assumed to be caused by distress and psychological dysfunction” (Schmidtke et al., 2008; Metternich et al., 2008). Typical complaints of FMD patients include subjective perception of deficits in prospective and working memory and deficits in attention and long term-memory (see Table 1). These problems usually fluctuate with stress levels and have generally begun within the past 5–10 yrs. FMD patients can credibly relate a great number of such mnemonic failures in their everyday lives. A typical profile of patients who develop FMD has been observed: their educational, professional, and socioeconomic attainment level is often above-average (Schmidtke et al., 2008, see also Berrios et al., 2000). The etiology of FMD is not completely understood. However, an etiological model has been suggested, according to which the complaints are an expression of actual stress-induced lapses of memory as well as low memory-related self-efficacy beliefs, memory-related anxieties, a perfectionism pertaining to one’s own cognitive abilities, and chronic psychosocial stress. Together, these symptoms form a circulus vitiosus, similar to the presumed etiology of somatoform disorders (Metternich et al., 2009). This model has been helpful in explaining the symptoms of FMD and in establishing and evaluating a specific new therapy (Metternich et al., 2008, 2010). Memory

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Table 1
Clinical FMD criteria and typical symptoms of FMD (according to Schmidtke et al., 2008).

Clinical FMD criteria
1. Complaint of acquired (>6 months) dysfunction of memory that, as perceived by patients, significantly affects their level of functioning in professional and/or private life
2. Presence of external and/or subjective factors, addressed as psychosocial burden factors that cause significant psychological stress
3. Verbal memory and attentional capacity above -1.5 SD (age-corrected), as assessed by standardised tests
4. Absence of a recognizable other cause of cognitive impairment. A physical examination, not including imaging, was routinely performed
5. Absence of major psychiatric disease, e.g. psychosis, major depression, dissociative disorder, obsessive-compulsive disease, etc. (previous or present). Patients with dysthymia or adjustment disorder with depressed mood were only included if the Beck Depression Inventory score was ≤ 15
Typical symptoms of FMD
<i>Items related to a deficit of working memory and concentration</i>
1. Forgetting errands on the way to their execution
2. Rapid forgetting of essential parts of a personal or telephone conversation
3. Experiencing disruptions of the thread of thoughts in conversations
4. Absent-mindedness and day-dreaming during conversations
<i>Items related to a deficit of the registration of new contents and prospective memory</i>
5. Forgetting important contents of conversations, appointments and errands (time scale of days)
6. Experiencing difficulties understanding and registering the contents of news, reading and lectures
<i>Items related to a deficit of retrieval</i>
7. Blocks of retrieval of well-known names, phone numbers, PIN codes, etc. (can typically be retrieved at a later time)
8. Committing errors, or experiencing "blackouts" during routine activities at work, at home, while driving, etc.
9. Difficulties finding words

dysfunctions caused by major psychiatric disorders exclude a diagnosis of FMD (Fischer et al., 2008; Isley et al., 1995). FMD patients usually show slightly elevated depression scores (Metternich et al., 2009) which might be a result of psychosocial distress (Pittenger and Duman, 2008; Potter et al., 2009; Schmidtke et al., 2008).

There is a symptom overlap of a major depressive disorder and of FMD regarding reported memory deficits (Schmidtke et al., 2008). In major depression, they are discussed to be mediated by hypercortisolism and disturbed sleep (Antonijevic et al., 2000), and there is evidence for impairment of declarative memory consolidation in depressed patients (MacQueen et al., 2002). Similarly, slow wave sleep is reduced in depression (Riemann et al., 2001), which proves to be an important condition for normal declarative memory consolidation (Plihal and Born, 1997). In FMD, however, there is on the one side no evidence for a memory impairment when retrieval takes place on the same day (Metternich et al., 2009). On the other side, the role of sleep has not yet been investigated notwithstanding the large number of studies providing evidence that sleep plays an important role in memory consolidation (McGaugh, 2000; Stickgold, 2005; for review see: Ficca and Salzarulo, 2004; Peigneux et al., 2001; Ellenbogen et al., 2006a).

Different methods have been used to assess the correlation between sleep and memory. Many authors investigated for example the relevance of sleep stages (Plihal and Born, 1997; Karni et al., 1994; Smith and Mac, 1994) for memory formation. Other studies suggest that the interaction of different sleep stages is essential for successful memory consolidation (Ficca et al., 2000; Stickgold et al., 2000a; Mednick et al., 2003). fMRI and PET studies showed that patterns of brain activity seen while practicing a memory task are "replayed" during subsequent sleep (Maquet, 2000). It has also been demonstrated that the consolidation of memory is related to a reactivation in the hippocampal region during sleep (Peigneux et al., 2003). Furthermore, molecular and electrophysiological studies support the hypothesis that sleep and sleep-specific brain activity patterns, such as sleep spindles or theta-waves, foster neural

plasticity believed to underly memory consolidation (Cantero et al., 2003; Louie and Wilson, 2001; Ribeiro et al., 1999; Sejnowski and Desthene, 2000; Smith, 1995).

Although depression and FMD are different diagnostic entities, they might share some similar underlying physiological mechanisms involved in memory consolidation. On the one hand, disturbed sleep might lead to impaired memory consolidation, on the other hand, hypercortisolism might be both linked to sleep disturbance and to memory consolidation (Kumari et al., 2009; Elzinga et al., 2005).

Primary objective of the present study was to assess overnight memory consolidation and sleep in FMD patients for the first time. We hypothesized that FMD patients show significantly attenuated sleep-related memory consolidation in comparison to healthy controls. Procedural and declarative memory was investigated separately by different memory tasks. Further explorative analyses of sleep were conducted using polysomnography and spectral analysis of sleep EEG.

2. Methods

2.1. Subjects

FMD patients were recruited in the Center for Geriatric Medicine and Gerontology of the University Medical Center in Freiburg, Germany. Inclusion criteria for the study were: patients aged between 40 and 60 yrs who fulfilled the diagnostic criteria of FMD (Schmidtke et al., 2008, see Table 1) or healthy subjects (no memory or sleep complaints). Exclusion criteria for patients and healthy controls were: (1) serious medical diseases, (2) treatment with anticonvulsants, sedatives, psychotropic drugs or medications that have an effect on following receptors: serotonin, histamine, dopamine, acetylcholine and beta-receptors in the last 2 weeks, (3) smoking more than 10 cigarettes a day, (4) current or past drug or alcohol abuse, (5) major psychiatric disorder (according to DSM IV/ICD-10), (6) IQ < 90 as determined by SPM (Raven et al., 1999, adapted short version containing 32 homogeneous items), (7) insufficient German language skills, and (8) education level below CSE (Certificate of Secondary Education). In both groups sleep habits and subjective sleep quality were monitored by questionnaires (PSQI, Buysse et al., 1989) and sleep diary for 2 weeks prior to the memory investigations.

All participants underwent a complete physical examination including urinalysis and blood work (blood cell count, thyroid hormones, liver enzymes, creatinine, BUN, CRP) and psychiatric assessment including a structured clinical interview according to DSM-IV criteria (SKID, DIA-X-interview) to rule out any relevant somatic and psychiatric disorder. Twelve healthy subjects were selected from a large pool of controls participating in sleep studies using the same study design. They were matched to the group of FMD patients regarding gender, educational level and age. Control subjects were recruited by advertisement from the clinic staff and their relatives. Written informed consent was obtained from all participants during the screening session. The study was approved by the local ethics committee.

2.2. Procedures and tasks

All participants spent one night in the sleep laboratory with polysomnographic monitoring over 8 h (time in bed), usually from 10:30 pm to 6:30 am according to standard procedures (Rechtschaffen and Kales, 1968). Patients and healthy subjects were advised to keep a regular sleep pattern, to avoid alcohol and caffeine before the polysomnographic assessment.

Memory performance and general neurocognitive performance (including attention, working memory and cognitive speed) were assessed from 7:00 to 9:00 pm prior to sleep (T1, learning condition) and at 7:00 to 9:00 am after sleep (T2, recall condition). Following encoding in the evening, participants were prepared for polysomnographic assessments and monitored until bed time by trained staff members to keep the level of novel sensory input and motor activity (interference) low and comparable between the groups. An interval of 30 min between awakening and recall served to minimize potential effects of sleep inertia. The testing sessions required about 60 min. The procedure had been developed for a previous study (Nissen et al., 2006).

Attention was assessed by TAP (testing battery for attention assessment, Zimmermann and Fimm, 2000) which included different aspects of attention. First tonic alertness was tested. Subjects had to press a button as fast as possible when a cross became visible on a PC-screen. Phasic alertness included an audio warning before the cross-popped up on the screen. For both tests reaction time was measured. Divided attention was assessed by reaction time and the number of correct and incorrect reactions with respect to a visual and an acoustic stimulus.

Psychomotor performance was tested by Trail Making Test (Reitan, 1958) which contains linking numbers (1–25) that are pre-printed a sheet of paper (Version A) and linking numbers (1–13) and letters (A–L) alternately (1-A, 2-B, 3-C, etc., Version

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