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CLINICAL REVIEW

Memory consolidation in sleep disorders

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SUMMARY

In recent years sleep-related memory consolidation has become a central topic in the sleep research field. Several studies have shown that in healthy individuals sleep promotes memory consolidation. Notwithstanding this, the consequences of sleep disorders on offline memory consolidation remain poorly investigated. Research studies indicate that patients with insomnia, obstructive sleep apnea, and narcolepsy often exhibit sleep-related impairment in the consolidation of declarative and procedural information. On the other hand, patients with parasomnias, such as sleep-walking, night terrors and rapid eye movement (REM) behavior disorder, do not present any memory impairment. These studies suggest that only sleep disorders characterized by increased post-learning arousal and disrupted sleep architecture seem to be associated with offline memory consolidation issues. Such impairments, arising already in childhood, may potentially affect the development and maintenance of an individual's cognitive abilities, reducing their quality of life and increasing the risk of accidents. However, promising findings suggest that successfully treating sleep symptoms can result in the restoration of memory functions and marked reduction of direct and indirect societal costs of sleep disorders.

Introduction

Memory consolidation, the process by which labile information becomes stronger, more efficient, and more resistant to interference [1], is purported to occur during offline periods after encoding. As already observed in a pioneering study in 1924 by Jenkins and Dallenbach [2], this process seems to be facilitated when learning is followed by a sleep period. However, only in recent years has extensive research indicated that an intervening period of sleep after learning promotes the offline consolidation of declarative, perceptual, emotional, and procedural information [3]. Consequently, if sleep affects memory consolidation, it is reasonable to hypothesize that disturbed and/or altered sleep, as experienced by individuals with sleep disorders, may impair the ability to optimally consolidate information. Further, it has been estimated that about 45 million individuals in Europe [4], and between 50 and 70 million in the US suffer from sleep disorders [5], but very few research studies have investigated the consequences of sleep disorders on offline memory consolidation abilities. Yet, understanding the cognitive consequences of sleep disorders holds far-reaching implications for both future neuroscientific investigations as well as for clinical purposes. Nonetheless, only one review by Cipolli and colleagues [6] has been published on this topic. In that review, the authors supported the idea of an impaired memory consolidation process in patients with sleep disorders. The current review aims to present and integrate the newly available findings regarding sleep disorders and memory consolidation with the studies described in Cipolli and colleagues' work [6], providing a guide for future investigations trying to understand how memory impairments might be reduced via treatments targeting the sleep symptomatology, and thus, facilitating the translation from basic research to clinical practice.

The present review is organized as follows. First, I will briefly describe the main theoretical models regarding sleep and memory consolidation. Next, I will review all the available studies on sleep disorder and memory consolidation presented by type of disorder (i.e., insomnia, obstructive sleep apnea, narcolepsy, parasomnias) and the chief results will be outlined. Subsequently, these results

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Abbreviations

AHI	apnea-hypopnea index
ASRT	alternating serial reaction time task
CPAP	continuous positive airway pressure
FTT	finger tapping task
NREM	non-rapid eye movement
NC	narcolepsy with cataplexy
MA	motor adaptation
MSL	motor sequence learning
MTT	mirror tracing task
OSA	obstructive sleep apnea
PSG	polysomnography
RBD	REM behavior disorder
ROCFT	Rey–Osterrieth complex figure test
REM	rapid eye movement
SOREM	sleep onset rapid eye movement
SMR	sensorimotor rhythm
SWA	slow wave activity
SWS	slow wave sleep
TDT	texture discrimination task
WAIS	Wechsler adult intelligence scale
WPA	word-pair association task

will be discussed within a comprehensive perspective on how memory consolidation is affected by sleep disturbances. In the final section, clinical implications will be advanced and discussed.

Sleep and memory consolidation

Offline memory consolidation is typically assessed comparing performance in a memory task before and after a period of time that includes sleep and/or wakefulness. Several theoretical models have been proposed to explain the role of sleep in the consolidation of information. Beyond the dual process hypothesis [7] and the opportunistic consolidation theory [8], the models that so far have attracted the most interest in sleep research are the active system consolidation model [1] and the synaptic homeostasis hypothesis [9].

The active system consolidation model [1], based on the two-stage model of memory trace formation [10], proposes that during wakefulness, stimuli are initially encoded in parallel in two different memory systems that store information at different rates: in the hippocampus at a faster rate and in cortical networks at a slower rate. During the subsequent sleep period, specifically during non-rapid eye movement sleep (NREM, composed by stage N1, N2 and N3, the latter also known as slow-wave sleep, SWS), the pieces of information learned during wake are re-activated in both the hippocampus and in the cortex. These reactivations, through a constant dialog between the hippocampus and the neocortex, mediate the redistribution of information in cortical areas. These cortical connections are then strengthened and stabilized via synaptic consolidation, and eventually integrated with pre-existing knowledge, making the learned event less susceptible to interference [3,11]. In addition, consolidated information becomes independent from the hippocampus, which returns to an optimized state for the encoding of new material [12–14]. This model provides an elegant framework for the consolidation of hippocampus-dependent information such as declarative and explicit motor memories [3].

According to the synaptic homeostasis hypothesis [9], the encoding of information during wakefulness leads to increased strength of connections (i.e., synaptic weights). During the subsequent sleep, when external inputs are reduced, slow oscillations renormalize these synapses inducing synaptic depression. This leads to a weakening of unimportant and less integrated information, making the important (signal) relative to the spurious information (noise) more salient. This process also restores the capacity of synapses to acquire new information. Since memory formation and stabilization requires some forms of synaptic plasticity, this model offers a parsimonious explanation for the consolidation of both hippocampal (i.e., declarative memories) and non-hippocampal information (e.g., perceptual learning) [9].

Recently, it has been proposed that these models may not be mutually exclusive, in the sense that they can describe complementary processes that improve memory consolidation during sleep [15–17]. Indeed, it is possible that during NREM sleep, salient information associated with experience acquired in wake is reactivated and integrated in cortical networks [15]. This process seems to be driven by the temporal coupling of thalamo-cortical sleep oscillations, such as slow oscillations, sleep spindles and sharp-wave ripples [18–21]. In the subsequent SWS, memory reactivations can be alternated with the synaptic downscaling process in order to reduce the strength of neural networks [16], resulting in an overall synaptic reduction [9]. However, previously integrated information remains strong, thus increasing signal-to-noise ratio and reinforcing reactivated memory traces [9].

In light of these theoretical models, it is reasonable to expect that when sleep patterns are disturbed and/or altered, such as is the case of sleep disorders, the ability to optimally consolidate information may be impaired. However, as it will be described in the following paragraphs (see also Table 1), the available findings on memory consolidation and sleep disorders are quite heterogeneous.

Memory consolidation in insomnia

Insomnia is a very common sleep disorder, with prevalence rates ranging 6–10% in the general adult population [22]. Insomnia-dependent cognitive impairments, included among the diagnostic criteria of both ICD-3 [23] and DSM-5 [24], have been well described (for a review [25]). Nevertheless, memory consolidation in insomnia remains poorly investigated.

In 2006, Backhaus and colleagues hypothesized that individuals with primary insomnia would show impairment in the consolidation of both procedural (motor adaptation abilities, MA) and declarative memories. To test this idea, they recruited 16 primary insomniacs (M_{age} = 41.6 y) and 13 controls (M_{age} = 40.1 y) and had them participate in two laboratory polysomnographic recordings (PSG), an adaptation and an experimental night, respectively [26]. The evening prior to the experimental night (learning phase), participants performed a word-pair association task (WPA) and a mirror-tracing task (MTT; Figs. S1–2). In the subsequent morning, participants performed a retrieval phase per task. Although the two groups displayed a similar performance during the learning phase, the number of word-pairs recalled at retrieval remained stable for insomniacs (but increased for controls), whereas procedural memories remained stable in both groups. Nevertheless, it should be noted that the WPA paradigm used in this study included the presentation of the correct word-pair after the last retrieval session in the evening, before sleep. Consequently, this did not allow them to parse whether controls improved their performance from the level reached at the end of the learning session, or if they just showed a slower decay than insomniacs.