Changes in Sleep Architecture following Motor Learning Depend on Initial Skill Level

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Abstract

■ Previous research has linked both rapid eye movement (REM) sleep and Stage 2 sleep to procedural memory consolidation. The present study sought to clarify the relationship between sleep stages and procedural memory consolidation by examining the effect of initial skill level in this relationship in young adults. In-home sleep recordings were performed on participants before and after learning the pursuit rotor task. We divided the participants into low- and high-skill groups based on their initial performance of the pursuit rotor task. In high-skill participants, there was a significant increase in Stage 2 spindle density after learning, and there was a significant correlation between the spindle density that occurred after learning and pursuit rotor performance at retest 1 week later. In contrast, there was a significant correlation between changes in REM density and performance on the pursuit rotor task during retest 1 week later in low-skill participants, although the actual increase in REM density failed to reach significance in this group. The results of the present study suggest the presence of a double dissociation in the sleep-related processes that are involved in procedural memory consolidation in low- and high-skill individuals. These results indicate that the changes in sleep microarchitecture that take place after learning depend on the initial skill level of the individual and therefore provide validation for the model proposed by Smith et al. [Smith, C. T., Aubrey, J. B., & Peters, K. R. Different roles for REM and Stage 2 sleep in motor learning. *Psychologica Belgica, 44,* 79–102, 2004]. Accordingly, skill level is an important variable that needs to be considered in future research on sleep and memory consolidation. ■

INTRODUCTION

A number of studies have reported a beneficial effect of sleep on memory consolidation (for a review of human studies see Walker & Stickgold, 2006, and Smith, 2001; for a review of animal studies see Smith, 2003). Many of these studies have tried to link different sleep stages (i.e., Stage 2 sleep, slow wave sleep [SWS], and rapid eye movement [REM] sleep) with different types of memory (e.g., declarative and nondeclarative memory). There has been some evidence to suggest that SWS and REM sleep facilitate declarative memory for neutral (Peigneux et al., 2004; Plihal & Born, 1997, 1999) and emotional (Wagner, Gais, & Born, 2001) material, respectively. Regarding nondeclarative memory, both REM sleep (Smith, Nixon, & Nader, 2004; Wagner, Hallschmid, Verleger, & Born, 2003; Maquet et al., 2000; Stickgold, LaTanya, & Hobson, 2000; Plihal & Born, 1997, 1999; Smith, 1993; Buchegger, Fritsch, Meier-Koll, & Riehle, 1991) and Stage 2 sleep (Fogel & Smith, 2006; Nader & Smith, 2003; Walker, Brakefield, Morgan, Hobson, & Stickgold, 2002; Smith & MacNeill, 1994) have been identified as being important for the memory consolidation. There have been at least two different ways to explain these discrepant findings with respect to sleep stages and nondeclarative memory: the complexity of the task (Smith, 2001) and the novelty of the task (Smith, Aubrey, & Peters, 2004).

Smith (2001) proposed a distinction between simple procedural tasks and complex procedural tasks. In line with this dichotomy, simple procedural tasks have been linked to Stage 2 sleep. In the study by Smith and MacNeill (1994), for example, subjects learned the pursuit rotor task and were then randomly assigned to one of several sleep deprivation conditions: total sleep deprivation for the whole night, total sleep deprivation for the last half of the night, selective deprivation of REM only, and non-REM (NREM) awakenings. Control subjects were allowed to sleep as normal. All subjects were retested 1 week later. Smith and MacNeill reported that subjects in the total sleep deprivation condition performed significantly worse than did subjects in the control and REM deprivation conditions. Because sleep in the last half of the night consists largely of REM sleep and Stage 2 sleep and because the subjects in the selective REM deprivation condition were not impaired at retest, the authors concluded that Stage 2 sleep was important for the consolidation of this simple motor task. Other research by Smith and colleagues (Fogel & Smith, 2006; Nader & Smith, 2003) has shown that the number and density of Stage 2 sleep spindles increases significantly in subjects following acquisition of simple

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motor tasks. These findings are particularly interesting because it has been suggested that sleep spindles represent an ideal mechanism to promote synaptic plasticity (Destexhe & Sejnowski, 2001; Steriade, 1999).

On the other hand, according to the complexity explanation, REM sleep has been associated with the consolidation of complex procedural tasks (Smith, 2001). For example, Smith, Nixon, et al. (2004) have recently reported that REM densities increased significantly after subjects learned the Tower of Hanoi and the Mirror Trace tasks, with the largest increases being observed in the later part of the night. Furthermore, the postacquisition increase in REM densities was significantly correlated with the amount of improvement on the tasks at retest 1 week later. Using a partial sleep deprivation approach, Plihal and Born (1997) showed that exclusion of late sleep (which comprises a large portion of REM sleep) impaired performance on the Mirror trace task more than exclusion of early sleep (which comprises a large portion of SWS). Thus, converging evidence from different approaches has suggested that REM sleep is involved in facilitating procedural learning of tasks that are somewhat complex or cognitive in nature.

Although the simple/complex dichotomy seems to explain some of the findings that have linked sleep stages to procedural memory consolidation, one of the problems with this explanation is that there is no easy way to decide which tasks are simple and which tasks are complex. In addition, it would be difficult to argue that some of the tasks that have been linked to REM sleep (e.g., the serial reaction time task and the visual texture discrimination task) are complex in nature (Maquet et al., 2000; Stickgold et al., 2000). We have recently put forth an alternative model in an attempt to account for the different roles of REM sleep and Stage 2 sleep in motor learning (Smith, Aubrey, et al., 2004). In order to better ground our model within the larger field of cognitive neuroscience, we have attempted to incorporate previous research on (a) the neural networks involved in motor learning and (b) the role of individual differences in task acquisition. Regarding the first point, our model builds upon the neuroanatomical model of motor learning proposed by Doyon and Ungerleider (2002) that discriminates between an early fast stage of motor learning and a slower late stage of motor learning. Doyon and Ungerleider further describe how each of these stages is dependent upon the activity of two well-studied motor systems within the brain: the corticocerebellar system and the corticostriatal system. The exact manner and time course of the involvement of these two systems in motor learning depends on the type of task being performed. For example, the corticocerebellar system is involved during the initial acquisition of motor sequence learning, whereas the corticostriatal system is involved in consolidating motor sequences for such tasks. According to our model, different motor tasks do not rely on different sleep stages. Rather, the role of each sleep stage is dependent upon the novelty or skill level of the individual. We have suggested that postacquisition REM sleep will be important if consolidation of a task requires the programming of new motor sequences (i.e., if the task is novel to an individual or if he or she does not have much skill on that task), which has been hypothesized to rely predominantly on the corticocerebellar system. In contrast, postacquisition changes in Stage 2 sleep will be important if consolidation of a task does not require the programming of new motor sequences (i.e., if the task is not novel, or the individual is already proficient at the task). Familiar sequences have been hypothesized to rely predominantly on the corticostriatal system (see Smith, Aubrey, et al., 2004, for a more detailed description on how REM sleep and Stage 2 sleep may be linked to the corticocerebellar and corticostriatal systems).

Regarding the role of individual differences in task acquisition, previous research has shown that such differences in perceptual, cognitive, or motor abilities may influence the results of behavioral and neuroimaging studies on learning by adding unwanted variability, thus potentially obscuring interesting patterns within samples (Frutiger et al., 2000; Ackerman, 1987). In addition, there is evidence that as individuals become more familiar with a certain task or obtain more expertise in processing certain classes of stimuli, they will begin to recruit different brain areas to perform those tasks and process those stimuli (Gauthier, Tarr, Anderson, Skudlarski; & Gore, 1999; Sakai et al., 1998; Jenkins, Brooks, Nixon, Frackowiak, & Passingham, 1994; Grafton et al., 1992). Accordingly, we have made our model on sleep and motor learning a dynamic one in which the sleep stage that is important in consolidating performance on a given motor task will depend upon the initial skill level that an individual demonstrates while performing that task.

We feel that one of the major advantages of our model on sleep and motor learning is that it attempts to link sleep stages, and more specifically the phasic events in these stages, to the different phases of motor learning that have been identified. Despite the groundbreaking efforts made by some investigators to show that sleep is important for consolidating motor learning tasks (for reviews, see Walker & Stickgold, 2006; Smith, 2001), an important gap still remains between the fields of sleep and memory on the one hand and the cognitive neuroscience of motor learning on the other hand. Indeed, much of the work in this area has concluded that sleep does facilitate motor learning, but it is not entirely clear on how sleep is specifically involved in motor consolidation. A better understanding of *how* sleep benefits motor learning is critical to advancing our knowledge of motor learning in general. Our model builds upon and extends earlier work and attempts to bridge the gap between the literature on sleep and memory and the

literature on the cognitive neuroscience of motor learning. However, the validity of our model has not yet been examined.

The purpose of the present study was to examine the validity of our (Smith, Aubrey, et al., 2004) model with respect to the roles of REM sleep and Stage 2 sleep in the consolidation of a procedural motor task—the pursuit rotor task. Like complexity, novelty is a construct that is difficult to operationalize in a laboratory setting. With this in mind, we decided to focus instead on the participants' initial level of skill on the task, which, it could be argued, is likely to be related to task novelty. To assess the initial skill level of the participants, we divided the sample into low- and high-skill groups depending on their initial level of performance on the pursuit rotor task. We predicted that participants in the high-skill group would show a significant increase in the density of Stage 2 sleep spindles, but no increase in REM density, following acquisition of the pursuit rotor task. We also predicted that the postacquisition increase in Stage 2 spindle density, but not REM density, would be correlated with performance on the pursuit rotor task at retest 1 week later in the high-skill participants. In contrast, participants in the low-skill group were hypothesized to show significant increases in REM density but not Stage 2 sleep spindle density. The postacquisition increase in REM density, but not Stage 2 sleep spindle density, was expected to be correlated with performance on the pursuit rotor task 1 week later at retest.

METHODS

Participants

A total of 24 participants took part in the study. All participants were undergraduate students enrolled at Trent University. The mean age was 20.54 years (SD = 2.40). There were 12 women and 12 men. At the outset, we excluded any individuals with irregular or unusual sleeping patterns, medical conditions or medication consumption, as well as conspicuous substance abuse that would disrupt cognitive performance or sleeping architecture. This study was approved by the Trent University Ethics Committee. All participants signed informed consent forms and received $25 per night (and bonus course credit if applicable) for the study.

Materials

The pursuit rotor task was used as a measure of motor procedural learning. To perform this task, participants had to track a moving light on a photoelectric device with a hand-held stylus using their nondominant hand. An electronic counter was used to determine how long each participant was able to keep the stylus on target during each 30-sec trial (as determined by the experimenter using a stop watch). The light moved around a rectangular pattern in a clockwise direction at a rate of 32 rpm.

The Multidimensional Aptitude Battery II (MAB-II; Jackson, 1998) was used to assess the Full-scale IQ, Verbal IQ, and Performance IQ of each subject. This IQ test is similar to the Wechsler Adult Intelligence Scale-R and these two scales are correlated quite highly (r = .91).

In-home sleep recordings were performed using Suzanne (Tyco Healthcare Group LP, Mansfield, MA) portable polysomnographic systems. These systems record physiological data at a sampling rate of 120 Hz onto 32-MB PC flash memory cards, which are then later downloaded offline onto a PC computer for subsequent analyses. We recorded the electroencephalogram (EEG), electrooculogram (EOG), and electromyogram (EMG) using silver-plated electrodes. The EEG (C3 and C4) and EOG (left and right eyes) were monopolar recordings and were referenced to electrodes placed on the contralateral mastoid bone (A1 and A2). The EMG channel was bipolar. Low- and high-pass software filters for EEG and EOG were set at 0.03 to 30 and 0.03 to 2 Hz, respectively. The low-pass software filter for the EMG channel was set at 10 Hz and no high pass was used. The skin was prepared using Nuprep abrasive cleanser and Ten-20 conductive paste along with tape or gauze was used to affix the electrodes in place.

Procedure

Sleep recordings were acquired for three consecutive nights for all participants. The purpose of the first night was simply to get the participants used to sleeping with all of the electrodes on. The data from this first night were not analyzed. The second night served as the participants ''baseline'' night of sleep so that changes in sleep architecture following learning could be made. On the afternoon (at approximately 4:00 p.m.) of the third day, participants performed 30 trials of the pursuit rotor task using their nondominant hand. Each trial lasted for 30 sec and trials were grouped into blocks of 5 with a brief pause in between each block. This acquisition phase of the pursuit rotor task took approximately 20 min. The sleep was recorded later that night and these data served as the ''acquisition'' night. Participants performed their retest session on the pursuit rotor task 1 week later.

On each night, a research assistant would go to the home of the participant to hook up the sleep-recording apparatus and then turn on the unit before leaving the home. Participants were then allowed to go to sleep at their preferred time. The research assistant would then go to the participant's home the following morning to turn the unit off and bring it back to the Trent University sleep laboratory to download the data. Participants were required to perform the acquisition and retest phases of

the pursuit rotor task at the Trent University sleep laboratory. To avoid any impact of performing the IQ testing on the baseline and acquisition nights, the MAB-II was administered to participants either on the day of their first night (acclimatization night) or after their acquisition night. The MAB-II was scored according to standard procedures (Jackson, 1998).

Data Analysis

The primary dependent variable for the pursuit rotor task was the total time on target (TOT). A median split procedure on the TOT for the first three trials during the acquisition phase of the pursuit rotor was used to divide the sample into low-skill ($n = 12$) and high-skill ($n = 12$) participants. To determine whether the two groups would differ in their performance at the end of the acquisition phase, an independent t test was used to compare the two skill groups on their TOT during the last three trials of the acquisition phase. We also performed independent t tests on the TOT during the first three trials and the last three trials of the retest phase. Independent t tests were also used to compare the two skill groups in terms of their age and their IQ (Full-scale, Verbal, and Performance) as assessed by the MAB-II (Jackson, 1998). The distribution of men and women between the two skill groups was assessed by using a chi-square analysis.

Sleep stages were scored[1] according to standard criteria (Rechtschaffen & Kales, 1968) using Sandman software (Version 7.2; Melville Diagnostics, Ottawa, Ontario, Canada). To determine whether acquisition affected sleep macroarchitecture differently in the two skill groups, 2 (group: low skill; high skill) × 2 (night: baseline; acquisition) analyses of variance (ANOVAs) were performed for the number of minutes awake (after sleep onset), the number of minutes of Stage 1 sleep, the number of minutes of Stage 2 sleep, the number of minutes of SWS (Stages 3 and 4 combined), the number of minutes of REM sleep, and the total time (TT = awake + Stage 1 + Stage 2 + SWS + REM). Sleep spindles were counted visually from the C3 or C4 channels in epochs of Stage 2 sleep over the entire night according to the following criteria: Spindles had to be in the 12- to 16-Hz frequency range, they had to be at least 0.5 sec in duration, and they had to resemble the typical fusiform spindle morphology. Spindle densities were calculated by dividing the total number of spindles by the total number of minutes in Stage 2 sleep throughout the night. REMs were counted visually from epochs of REM sleep over the entire night. Using the right or left EOG recording, eye movements had to be conjugal and they had to exceed 15 μV. To test our predictions regarding the effect of acquisition on sleep microarchitecture (spindle and REM densities) in the two skill groups, 2 (group: low skill; high skill) × 2 (night: baseline; acquisition) ANOVAs were performed. Cohen's effect sizes (Cohen & Cohen, 1983) were also computed to provide a measure of the magnitude of changes using the following formula: mean (acquisition night) − mean (baseline night)/standard deviation (baseline night). To assess whether changes in sleep microarchitecture (spindle and REM densities) were related with pursuit rotor performance (acquisition and retest phases), Pearson correlation coefficients were computed. All statistical analyses were performed using Version 11.5 of SPSS. The alpha level was set at .05 for all analyses.

RESULTS

Age did not differ significantly between the low-skill ($M = 20.42$; $SD = 2.54$) and the high-skill ($M = 20.67$; $SD = 2.35$) groups ($p > .80$). There were no significant differences between the low- and high-skill groups in terms of their Full-scale IQ ($M = 108.96$; $SD = 10.36$ vs. $M = 110.08$; $SD = 9.77$), their Verbal IQ ($M = 107.17$; $SD = 10.69$ vs. $M = 107.33$; $SD = 10.21$), or their Performance IQ ($M = 108.96$; $SD = 10.36$ vs. $M = 110.08$; $SD = 9.77$); $p > 60$ for all. In terms of sex distribution, there were seven men and five women in the low-skill group, which did not differ significantly from the number of men ($n = 5$) and women ($n = 7$) in the high-skill group, $p > .40$.

Pursuit Rotor Performance

The performance of the low-skill and high-skill groups on the pursuit rotor task during the acquisition and retest phases is shown in Figure 1. As expected, the difference between the two groups on the first three trials during acquisition was quite large (remember that the TOT for these three trials was used to divide the sample into the two skill groups; hence no statistical comparison was made). The difference between the two skill groups remained consistent throughout the remainder of the acquisition and retest phases. The difference between the low-skill ($M = 36.23$; $SD = 7.47$) and high-skill ($M = 46.65$; $SD = 7.54$) groups in terms of the TOT during the last three trials of the acquisition phase was significant, $t(22) = 3.40$, $p = .003$. The low-skill ($M = 40.39$; $SD = 7.48$) and the high-skill ($M = 44.55$; $SD = 8.86$) groups did not differ in their performance on the first three trials during retest 1 week later ($p > .20$). However, by the end of the retest session, the high-skill group ($M = 53.40$; $SD = 6.21$) performed significantly better than the low-skill group ($M = 46.19$; $SD = 7.88$) on the last three trials, $t(22) = 2.49$, $p = .021$. One additional point worth mentioning is that both groups appeared to reach their own asymptotes (approximately 45 and 55 sec in the low- and high-skill groups, respectively, out of a maximum of 90 sec) during the retest session, suggesting that there was a ceiling effect on this task at retest.

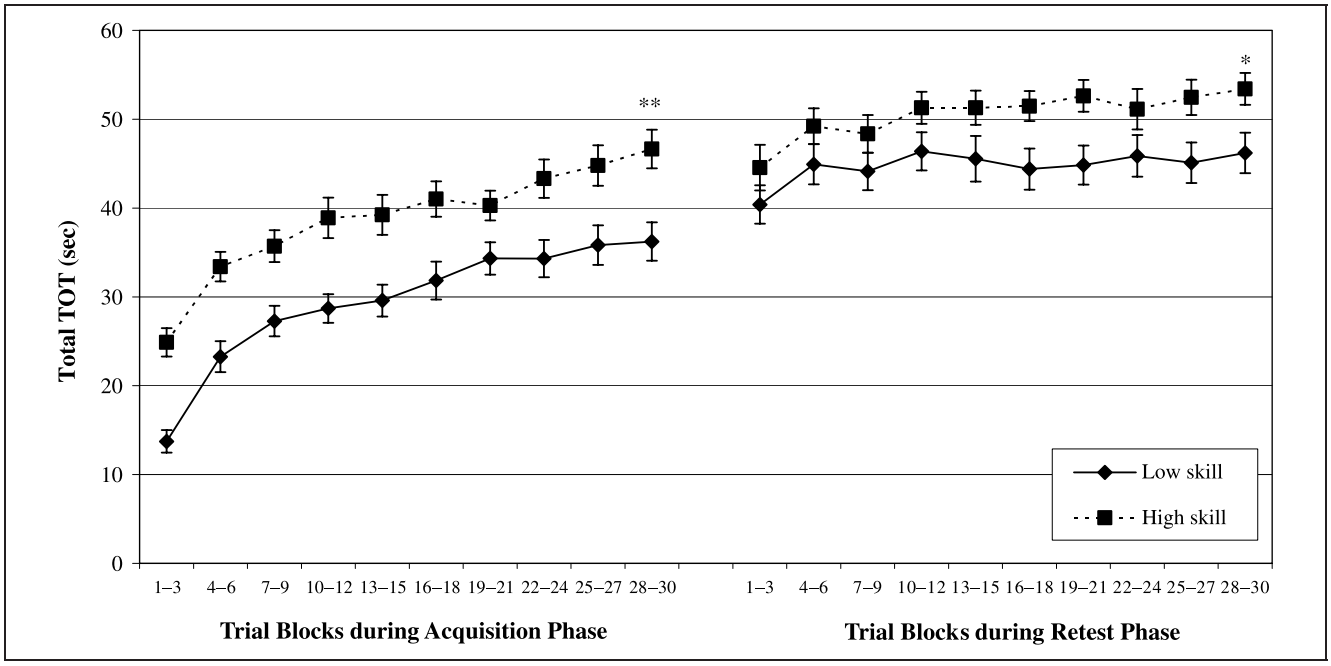

Figure 1. Pursuit rotor performance during acquisition and retest sessions.
**$p < .01$;
*$p < .05$.

Sleep Macroarchitecture

Table 1 contains the number of minutes and percent of total time (TT) spent awake (after sleep onset) and in each sleep stage for the baseline and acquisition nights. There was a significant Group × Night interaction, $F(1,22) = 7.68$, $p = .011$, for TT. Follow-up paired t tests revealed that there was a significant increase in TT for the high-skill group, $t(11) = 2.40$, $p = .035$, whereas the decrease in TT observed for the low-skill group only approached significance, $t(11) = 2.00$, $p = .071$. Neither the main effect of night nor the main effect of group was significant for TT ($p > .10$ for both).

Similar ANOVAs were performed for the number of minutes awake and in each sleep stage to determine the source of the group differences in overall TT. The main effect of night and group and the interaction between group and night were not significant for time spent awake ($p > .30$ for all). There was a main effect of night for time spent in Stage 1, indicating that there was a significant decrease in this stage for both groups between the baseline and acquisition nights. The main effect of group and the interaction term failed to reach significance ($p > .20$ for both). There was a significant Group × Night interaction for time spent in Stage 2 sleep, $F(1,22) = 4.87$, $p = .038$. Follow-up paired t tests indicated that participants in the high-skill group spent significantly more time in Stage 2 sleep on the acquisition night than on the baseline night, $t(11) = 2.25$, $p = .046$. The decrease in Stage 2 sleep for the low-skill group did not reach significance ($p > .20$). Regarding SWS, the main effects of night and group were not significant ($p > .70$ for both). However, the Group × Night interaction came close to reaching significance, $F(1,22) = 3.61$, $p = .071$. Finally, for REM sleep duration, there were statistical trends for the main effect of night, $F(1,22) = 3.32$, $p = .082$, and the main effect of group, $F(1,22) = 3.83$, $p = .063$. The Group × Night interaction also approached significance, $F(1,22) = 3.48$, $p = .076$. In summary, there was a significant increase in the number of minutes between sleep onset and offset (i.e., TT) across the two nights in the high-skill group compared to the low-skill group. This increase was driven primarily by more time spent in Stage 2 sleep and, to a lesser extent, more time spent in SWS and REM sleep.

Because there were differences between the groups in TT, we also performed Group × Night ANOVAs for the percent of TT spent awake and in each sleep stage (see Table 1). There were only two effects worth mentioning. There was a main effect of night for Stage 1, $F(1,22) = 12.76$, $p = .002$, indicating that the percent of TT spent in Stage 1 decreased in both groups. The main effect of group for REM sleep approached significance, $F(1,22) = 4.21$, $p = .052$: The percent of TT spent in REM sleep was greater overall in the high-skill group than in the low-skill group. None of the other main effects and interaction terms were significant ($p > .10$ for all).

Sleep Microarchitecture

Descriptive data for the spindle and REM densities for each group are reported in Table 2. The Group × Night ANOVA for spindle density yielded a significant main

Table 1. Sleep Macroarchitecture Data for the Low-skill (n = 12) and High-skill (n = 12) Groups on the Baseline and Acquisition Nights

	Baseline Night		*Acquisition Night*	
	No. of Minutes	*% of Total Time*[a]	*No. of Minutes*	*% of Total Time*
Awake[b]				
Low skill	26.83 (24.32)	5.67 (5.50)	14.32 (11.59)	3.12 (2.49)
High skill	28.43 (30.39)	5.57 (4.90)	27.43 (25.20)	4.86 (4.32)
Stage 1				
Low skill	8.54 (7.18)	1.73 (1.44)	4.92 (6.58)	1.00 (1.20)
High skill	7.04 (5.14)	1.51 (1.04)	5.49 (5.10)	0.98 (0.90)
Stage 2				
Low skill	247.17 (35.02)	50.65 (7.33)	234.21 (47.97)	51.67 (4.33)
High skill	220.04 (48.91)	46.83 (8.09)	253.74 (44.86)	47.49 (6.18)
SWS				
Low skill	97.17 (37.07)	19.65 (6.53)	88.88 (30.62)	20.35 (7.86)
High skill	91.96 (29.60)	20.23 (7.38)	103.25 (43.26)	19.11 (6.21)
REM				
Low skill	109.79 (30.00)	22.30 (5.04)	109.50 (38.03)	23.86 (6.21)
High skill	121.42 (35.06)	25.85 (5.71)	147.06 (37.11)	27.56 (5.98)
Total time				
Low skill	489.50 (38.42)	–	451.82 (76.72)	–
High skill	468.89 (74.15)	–	536.97 (88.59)	–

Data entries are mean (*SD*).

[a]Total time = no. of minutes awake + no. of minutes in Stage 1 + no. of minutes in Stage 2 + no. of minutes in SWS + no. of minutes in REM sleep.

[b]Awake = wakefulness after sleep onset.

effect of night, $F(1,22) = 11.72$, $p = .002$, indicating that there was an overall significant increase in spindle density across the two nights. The two groups did not differ in their overall spindle densities as revealed by a nonsignificant main effect of group ($p > .60$). Of particular interest, there was a significant Group × Night interaction, $F(1,22) = 9.00$, $p = .007$. Follow-up paired t tests revealed that there was a significant increase in spindle density across the two nights for the high-skill group, $t(11) = 4.50$, $p = .001$, but not for the low-skill group ($p > .70$). Cohen's effect sizes (d) were also calculated to provide a measure of the magnitude of change in spindle density for each group (see Table 2). The increase in spindle density for the high-skill group was just over half of a standard deviation ($d = .60$); in contrast the magnitude of increase was just above 0 ($d = .03$) in the low-skill group.

Table 2. Sleep Microarchitecture for the Low-skill (n = 12) and High-skill (n = 12) Groups on the Baseline and Acquisition Nights

	Baseline Night	*Acquisition Night*	*d*
Spindle density			
Low skill	6.02 (1.81)	6.08 (1.87)	.03
High skill	5.29 (1.46)	6.16 (1.58)	.60
REM density			
Low skill	13.94 (5.68)	16.55 (5.66)	.46
High skill	13.72 (6.72)	14.96 (7.08)	.18

Data entries are mean (*SD*). d = Cohen's effect size.

The Group × Night ANOVA for REM densities revealed a significant main effect of night, $F(1,22) = 4.87$, $p = .038$, indicating that there was an overall increase in REM density across the two nights. The main effect of group did not reach significance ($p > .70$), nor did the Group × Night interaction ($p > .40$). These results show that the two groups did not differ in their overall REM densities or in the degree of change in their REM densities across the two nights. In terms of magnitude, however, the effect size of the increase in REM density was almost one half of a standard deviation ($d = .48$) in

Table 3. Correlation Coefficients for Pursuit Rotor Performance and Sleep Microarchitecture Data in the Low-skill (n = 12) and High-skill (n = 12) Groups

	Low-skill Group		*High-skill Group*	
	TOT Acquisition	*TOT Retest*	*TOT Acquisition*	*TOT Retest*
TOT during acquisition phase	–	.543 (.068)	–	.872 (<.001)
Spindle density on acquisition night	.109 (.736)	−.182 (.571)	.546 (.066)	.636 (.026)
Change[a] in spindle density	.460 (.132)	−.011 (.973)	.532 (.075)	.439 (.153)
REM density on acquisition night	−.131 (.685)	.091 (.778)	.118 (.714)	−.234 (.463)
Change[a] in REM density	.564 (.056)	.654 (.021)	.111 (.732)	−.196 (.541)

Date entries are Pearson correlation coefficients (and p values). TOT = total time on target.

[a]Change = acquisition night minus baseline night.

the low-skill group compared to a much smaller effect in the high-skill group (d = .18). These data suggest that the increase in REM density was relatively large in the low-skill group, but because participants in the high skill group also showed an increase, albeit of lesser magnitude, the Group × Night interaction failed to reach significance.

Correlational Analyses

Table 3 contains the correlation coefficients for the pursuit rotor performance measures and the sleep microarchitecture data. In the low-skill group, there was a significant correlation between the change in REM density (acquisition night minus baseline night) and the TOT during retest 1 week later (TOT-retest), $r(11) = .654$, $p = .021$. The correlation between the change in REM density and TOT during acquisition (TOT-acquisition) approached significance, $r(11) = .564$, $p = .056$. These results show that the TOT during acquisition was related to the amount of change in REM density and this change in REM density was also correlated with the TOT during retest. A similar situation emerged in the high-skill group for spindle density on the acquisition night. There was a significant correlation between spindle density and TOT-retest, $r(11) = .636$, $p = .026$, and a trend for the correlation with TOT-acquistion, $r(11) = .546$, $p = .066$. These findings indicate that in the high-skill group the TOT during acquisition was related to the spindle density later that night, which was also related to the TOT during retest. Of particular relevance for the present study is the opposite pattern of correlations between the two groups for the correlations among TOT-retest, spindle density on acquisition night, and the change in REM density across nights (see Figures 2 and 3). These results suggest that, as predicted, different memory-related phasic sleep processes were occurring in the two skill groups following acquisition.

One additional point worth mentioning about Table 3 is that the correlation coefficients between the TOT-

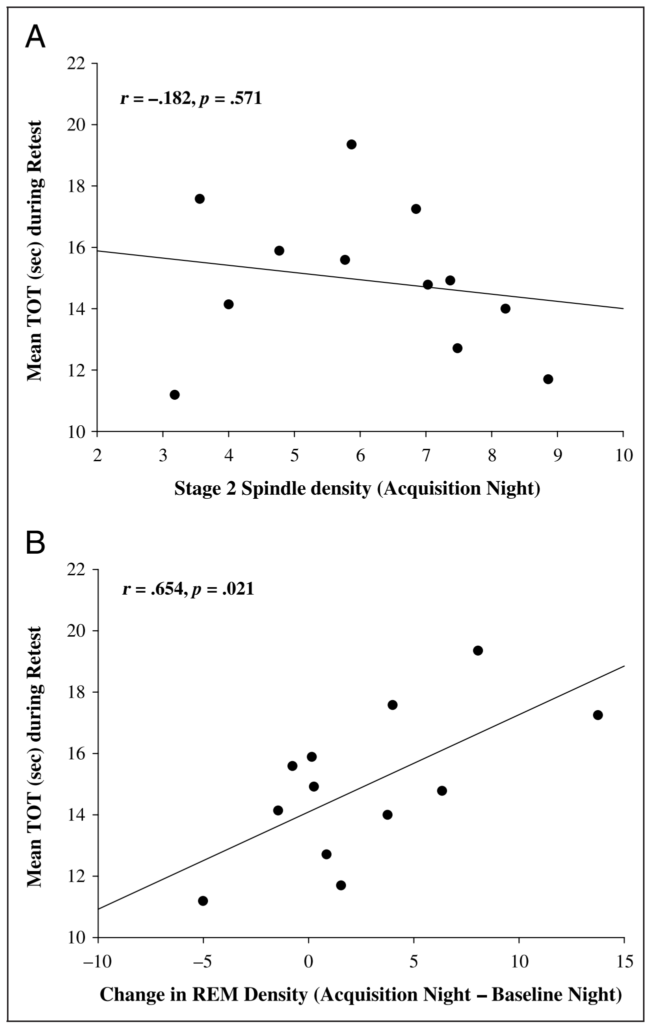

Figure 2. Low-skill group. (A) Scatterplot of the relationship between Stage 2 spindle density on acquisition night and pursuit rotor performance at retest in low-skill group (n = 12). (B) Scatterplot of the relationship between the change in REM density (acquisition night minus baseline night) and pursuit rotor performance at retest in low-skill group (n = 12).

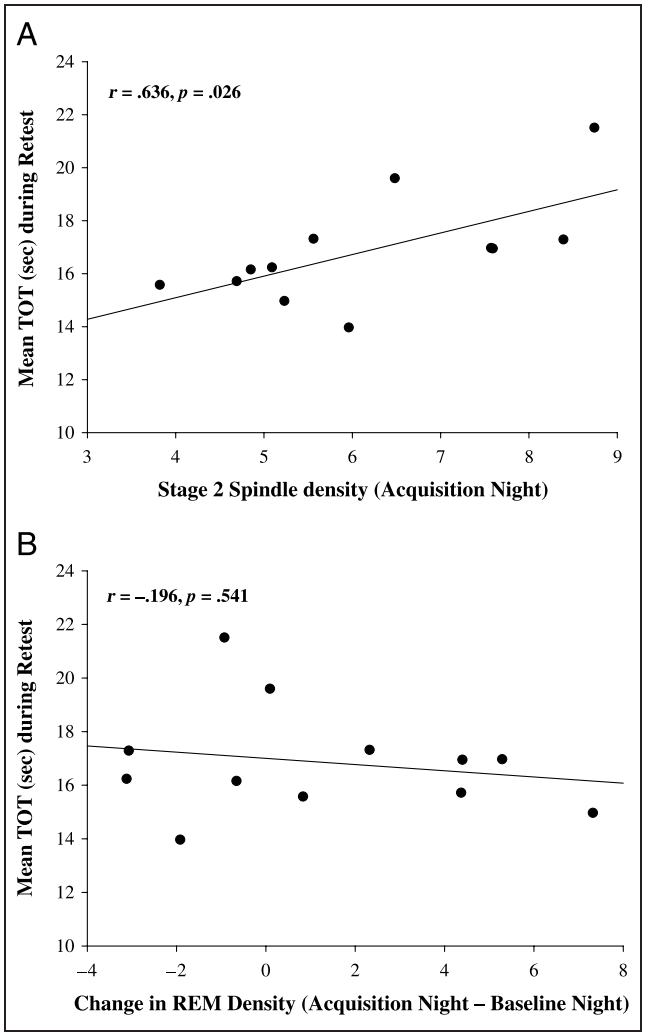

Figure 3. High-skill group. (A) Scatterplot of the relationship between Stage 2 spindle density on acquisition night and pursuit rotor performance at retest in high-skill group (n = 12). (B) Scatterplot of the relationship between the change in REM density (acquisition night minus baseline night) and pursuit rotor performance at retest in high-skill group (n = 12).

acquisition and TOT-retest phases were different in the two groups. This correlation was quite robust in the high-skill group, $r(11) = .872$, $p < .001$; in contrast, the correlation was much smaller in the low-skill group, $r(11) = .543$, $p = .068$. These results indicate that performance on the pursuit rotor task during acquisition was much more predictive of subsequent performance on this task 1 week later in the high-skill group than in the low-skill group. We decided to conduct post hoc hierarchical regression analyses to determine whether changes in sleep microarchitecture (i.e., spindle and REM densities) mediated the correlations between TOT-acquisition and TOT-retest differently in the two skill groups. Separate analyses were performed for spindle and REM densities in each group. For each of these analyses, the ability of TOT-acquisition to account

Table 4. Hierarchical Regression Analyses for Variables Predicting TOT-retest in the Low-skill Group (n = 12)

Variable	R^2	ΔR^2	p
Initial model			
TOT-acquisition alone	.295	–	.068
Model 2			
Step 1: Spindle density on acquisition night	.033	–	.571
Step 2: TOT-acquisition	.354	.321	.064
Model 3			
Step 1: Change[a] in REM density	.427	–	.021
Step 2: TOT-acquisition	.472	.045	.406

TOT = total time on target.

[a]Change = acquisition night minus baseline night.

for the variance in TOT-retest (r^2) was determined before and after statistically controlling for the sleep density measure of interest. The results of these analyses for the low-skill group are reported in Table 4. Initially, in the low-skill group, TOT-acquisition accounted for 29.5% of the variance in TOT-retest. After controlling for spindle density, the predictive ability of TOT-acquisition increased slightly to 32.1%. In contrast, after controlling for the change in REM density, the predictive ability of TOT-acquisition dropped to 4.5% (an attenuation of over 84%). These results suggest that the ability of TOT-acquisition to predict TOT-retest was largely mediated by the changes in REM density observed following learning in the low-skill group.

An opposite pattern of results emerged in the high-skill group (see Table 5). Initially, TOT-acquisition was a

Table 5. Hierarchical Regression Analyses for Variables Predicting TOT-retest in the High-skill group (n = 12)

Variable	R^2	ΔR^2	p
Initial model			
TOT-acquisition alone	.760	–	<.001
Model 2			
Step 1: Spindle density on acquisition night	.404	–	.026
Step 2: TOT-acquisition	.796	.392	.002
Model 3			
Step 1: Change[a] in REM density	.039	–	.541
Step 2: TOT-acquisition	.847	.808	<.001

TOT = total time on target.

[a]Change = acquisition night minus baseline night.

strong predictor of TOT-retest, being able to account for 76.0% of the variance. After controlling for spindle density on the acquisition night, however, the predictive ability of TOT-acquisition dropped by almost half to 39.2%. Even after controlling for spindle density, TOT-acquisition still accounted for a significant amount of unique variance in predicting TOT-retest. There was a slight increase (6.31%) in the predictive ability of TOT-acquisition after controlling for the change in REM density. These results suggest that the ability of TOT-acquisition to predict TOT-retest was partially mediated by the density of spindles following acquisition in the high-skill group.

DISCUSSION

The present study examined the effect of initial skill level on the relationship between sleep microarchitecture and procedural memory consolidation. There were eight specific predictions—four for each group. Each of the four predictions for the high-skill group was confirmed. Spindle density in the high-skill group increased significantly after learning the pursuit rotor task, whereas there was no significant change in REM density in this group. Spindle density on acquisition night was also highly correlated with performance on the pursuit rotor at retest 1 week later, but the change in REM density was not. A post hoc hierarchical regression analysis revealed that the spindle density on the acquisition night partially mediated the correlation observed between pursuit rotor performance during acquisition and retest: After controlling for postlearning spindle density, the predictive ability of TOT during acquisition was attenuated by almost 50%. Three out of the four predictions regarding the low-skill group were supported. The change in REM density observed across nights and after learning, although of moderate size, failed to reach significance. As predicted, the change in spindle density was not significant in this group. The two predictions regarding the pattern of correlations in the low-skill group were both supported. There was a significant relationship between pursuit rotor performance at retest and the change in REM density but not spindle density. In fact, the correlation between TOT during acquisition and TOT during retest in low-skill individuals was largely mediated by changes in REM density that occurred after learning: The predictive ability of TOT during acquisition was attenuated by over 80% after controlling for changes in REM density. Taken together, these results suggest the occurrence of a double dissociation in the neural processes involved in the consolidation of a procedural memory task in low- and high-skill individuals. Spindles and REMs appear to be important and dissociable electrophysiological markers of procedural memory consolidation processes occurring during postacquisition sleep in high-skill and low-skill individuals, respectively.

There are two particularly interesting and relevant points that are worth mentioning about the performance on the pursuit rotor task in this study.[2] First, the overall magnitude of improvement, especially in the high-skill group, is not as pronounced as the improvements on other motor tasks following a period of sleep (Huber, Ghilardi, Massimini, & Tononi, 2004; Fischer, Hallschmid, Elsner, & Born, 2002; Walker et al., 2002). One very plausible explanation for this finding is that there is a ceiling effect on the pursuit rotor task. In other words, participants are able to improve on this task up to a certain point, after which they are not physically able to improve any more. This ceiling effect is especially apparent in the retest data shown in Figure 1 where the lines of both groups are essentially flat for most of the retest phase. The second point is that although the performance of the high-skill group was significantly better than the low-skill group at the end of the initial acquisition session, the two groups did not differ at the beginning of the retest session 1 week later. These results suggest that consolidation processes that occur across one or several nights of sleep seem to have effectively normalized the performance of the two groups. We must also point out, however, that the high-skill group ended the retest session with significantly better performance than the low-skill group. Thus, although sleep has helped to normalize performance between the two groups, there is still some degree of difference between them that remains. It may very well be the case that sleep-related memory consolidation processes are capable of improving performance only up to a certain point, after which other factors related to overall skill level and/or learning potential constrain further improvement. Future research should address the issue of how much sleep is able to facilitate task performance and role of individual differences in this regard (i.e., do some people benefit more than others from sleep-related memory consolidation processes?). In addition, in light of the first point mentioned above, researchers in the future should also consider choosing tasks that limit or avoid potential ceiling effects.

The present study found evidence that spindles and REMs are differentially related to procedural memory consolidation in high- and low-skill groups, respectively. An important question to address regarding these results is *how* spindles and REMs are related to memory consolidation. Previous research has suggested that each of these phasic events is an important electophysiological indicator of synaptic plasticity. During sleep spindles, an interesting scenario takes place between the thalamus and the cortex (Destexhe & Sejnowski, 2001; Steriade, 1999; Steriade & Amzica, 1998). Cortical neurons receive both excitatory input (at the level of the dendrites) and inhibitory input (at the level of the cell body) from the thalamus. The result is an influx of calcium into the cortical neurons without excessive cell firing. It has been suggested that the influx of calcium

that occurs during sleep spindles may trigger the cellular process (e.g., changes in gene expression) that are involved in synaptic plasticity. Thus, synapses may be undergoing chemical and morphological changes that make them more efficient during sleep spindles, and these synaptic changes may support the consolidation of memories. In our model, we hypothesize that Stage 2 sleep spindles reflect plasticity-related changes that occur in the corticostriatal motor system following motor learning. Sleep spindles are generated via interactions between the thalamus and the cortex (Destexhe & Sejnowski, 2001; Steriade, 1999; Steriade & Amzica, 1998). The corticostriatal system involves connections among the frontal cortex, striatum (caudate and putamen), globus pallidus, substantia nigra, and thalamus (Alexander, DeLong, & Strick, 1986). Because sleep spindles have been hypothesized to represent an ideal time for synaptic plasticity and because the thalamus is part of the corticostriatal motor system, we have suggested that sleep spindles are important for motor consolidation processes involving the corticostriatal system (for more detailed discussion, see Smith, Aubrey, et al., 2004).

Regarding REMs and memory consolidation, there has been some very impressive work by Datta and Patterson (2003) that link REM sleep and synaptic plasticity. Using a model of memory consolidation in the rat, these researchers have shown that the P-wave-generating cells, which are located in the brainstem, are involved in learning and memory. Like the ponto–geniculo–occipital (PGO) waves recorded in cats, the P waves observed in rats precede the REMs that occur during REM sleep. The cells that generate P waves are controlled by the same pontine areas that are essential for initiating and sustaining REM sleep. Augmenting cholinergic levels in the brainstem area that contains P-wave-generating cells enhances memory consolidation (Mavanji & Datta, 2003) and destruction of this area impairs memory consolidation (Mavanji, Ulloor, Saha, & Datta, 2004). In addition, the brainstem areas that contain P-wave-generating cells are also connected to many of the brain areas that are involved in memory processing (Datta & Patterson, 2003). In this context, REMs may actually be electrophysiological indicators of neural processes (e.g., brainstem activation) that are involved in memory consolidation. Due to ethical considerations, it has not been possible to clearly demonstrate PGO wave activity in humans to the same extent as it has in nonhumans because this would require the implantation of intracerebral recording electrodes. However, a recent positron emission tomography imaging study has shown that activation within the primary occipital cortex and the lateral geniculate nuclei were strongly related to spontaneous eye movements during REM sleep but not to spontaneous eye movements during wakefulness (Peigneux et al., 2003). These brain areas correspond to two of the three brain areas that researchers have consistently used to record PGO activity in cats. The authors of this study concluded that the mechanisms that produce PGO wave activity during REM sleep in nonhumans are likely to be quite similar to the mechanisms that produce eye movements during REM sleep in humans.

Previously, we have attempted to link REM sleep and REMs to consolidation processes occurring within the corticocerebellar motor system (Smith, Nixon, et al., 2004). REM sleep deprivation has been associated with cellular changes within the cerebellum, including reductions in brain-derived neurotrophic factor (BDNF; Sei, Saitoh, Yamamoto, Morita, & Morita, 2000) and intracellular calcium (Mallick & Gulyani, 1996), both of which have been linked to synaptic plasticity. The influx of calcium has been shown to regulate the expression of the BDNF gene (Finkbeiner, 2000). Furthermore, REM deprivation in humans impairs the acquisition of eyeblink conditioning, a task that heavily involves the cerebellum (Ohno, Unishihara, Sei, & Morita, 2002). Converging evidence at the cellular and behavioral level therefore suggests that REM sleep is an ideal time for consolidation processes with the cerebellum to occur.

The main effect of night for spindle density indicated that the increase in spindle density overall (i.e., for both groups combined) was significant. This result is consistent with a previous study (Fogel & Smith, 2006) that also reported a significant increase in spindle densities after learning the pursuit rotor task. Note that in this previous study the authors did not divide the sample into groups—analyses were performed on only one sample. The results of the present study suggest that the increase in spindle density in the high-skill individuals was large and robust enough to drive the overall increase in spindle densities for the entire sample (i.e., both low- and high-skill groups combined). Accordingly, researchers interested in the relationship between sleep and procedural memory consolidation should take the initial skill level of their participants into account when designing their studies.

Although the correlation between change in REM density and pursuit rotor performance at retest was significant in the low-skill group, the actual increase in REM density across nights was not significant. It should be pointed out that although the increase was not statistically significant, the magnitude of the increase was almost one half of a standard deviation, suggesting that the increase was of moderate size. There are at least two possible reasons why this increase in REM density failed to reach significance. First, there were only 12 participants in each group. Perhaps the Group × Night interaction would have been significant if we had used larger sample sizes. Second, it is possible that the pursuit rotor task was not difficult or novel enough to fully engage the REM mechanism in the low-skill group. According to our model (Smith, Aubrey, et al., 2004), REM sleep should be important for tasks that require novel motor sequences to be programmed (e.g., tasks

that have never been done before or tasks in which individuals do not have much initial skill). According to this explanation, the magnitude of error correction or programming of motor sequences needed to perform the pursuit rotor task was only strong enough to marginally engage the REM system during postacquisition sleep. In retrospect, it is likely that all of our participants, even those in the low-skill group, had some kind of previous experience performing tasks that would be similar to the pursuit rotor (e.g., tracking a target or moving one's hand in a clockwise fashion). Perhaps our low-skill participants would have shown significant increases in REM densities had we used a more novel or difficult task. Future studies are needed to address this possibility.

One potential limitation of the present study is that we did not have a proper control group (i.e., one that did not perform the pursuit rotor task). There are several reasons why we feel that the lack of true control group does not limit the results of this study. First, we used a between- and within-subjects design. We recorded a baseline night of sleep for each subject and then compared the change in sleep architecture following learning to this baseline night. In this way, each subject served as his or her own control. Second, the low- and high-skill groups showed different patterns of sleep-related changes following learning. For example, the fact that spindle density increased significantly in the high-skill group but not in the low-skill group strongly suggests that this increase was not due to simple night-to-night variations in sleep architecture or to some other nonspecific aspect about participating in this particular study. Third, there is evidence that spindle and REM densities are very consistent across regular nights of sleep (Darchia, Campbell, Palagini, & Feinberg, 2004; Gaillard & Blois, 1981). In addition, previous studies on the relationship between sleep and memory that have used ''no-learning'' control groups have reported no significant changes in spindle density (Fogel & Smith, 2006) or REM density (Smith, Aubrey, et al., 2004). We therefore believe that our results are robust and that our conclusions can be interpreted in a meaningful manner.

A second limitation of this study is that we only monitored the sleep for one night before and one night following acquisition (in addition to the first night that was used to acclimatize participants to the sleep-recording apparatus). It would be very useful in the future to sleep record participants for several nights following learning to obtain a better picture of the nature and time course of the sleep-related changes that occur after learning. For example, according to our model (Smith, Nixon, et al., 2004) novel tasks should initially lead to increases in REM densities followed by increases in spindle densities. Thus, one would predict that spindle density should increase at some point (e.g., the next night) in our low-skill group. Future studies are needed to investigate this possibility.

A third limitation of our study concerns sample size. As mentioned above, the fact that we had only 12 participants in each skill group may explain the failure to find a significant increase in REM densities after learning in the low-skill group despite the moderate effect size of this increase. Although our sample size can be considered adequate in terms of other sleep studies that obtain polysomnographic recordings before and after learning, it would nonetheless be useful to employ larger sample sizes in future studies. This point becomes particularly relevant when one considers the results of our correlation analyses. Despite the fact that the correlations between performance at retest and the phasic activity following learning were quite large (.65 in the low-skill group and .64 in the high-skill group), these correlations would no longer be considered statistically significant if we were to apply a conservative control (such as the Bonferroni correction) for the number of correlations that we performed in Table 3. Larger samples in the future would overcome this limitation.

In conclusion, we investigated the role of initial skill in the relationship between sleep and procedural memory consolidation in young adults. We divided participants into low- and high-skill groups based on their initial performance of the pursuit rotor task. The results of the present study suggest the presence of a double dissociation in the sleep-related processes that are involved in procedural memory consolidation in low- and high-skill individuals. The increase in REM density, but not spindle density, which occurred during postacquisition sleep in low-skill participants was significantly related to their performance on the pursuit rotor at retest 1 week later. In contrast, the increase in spindle density, but not REM density, which was observed during postacquisition sleep in high-skill participants was significantly related to their performance on the pursuit rotor task 1 week later. These results indicate that the changes in sleep microarchitecture that take place after learning depend on the initial skill level of the individual and therefore provide validation of the model proposed by Smith, Aubrey, et al. (2004) that links sleep stages to motor learning. Accordingly, skill level is an important variable that needs to be considered in future research on sleep and memory consolidation.

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Notes

1. All data were scored by one person who was blind to group membership (i.e., she did not know whether a participant was in the low- or high-skill group). Although it is very unlikely that there was any systematic bias in this person's scoring that would have influenced our results, we had a second scorer randomly select 10 nights of data and score them for sleep stage and phasic events (i.e., spindles and REMs). This second scorer was also blind to group membership. The correlation between the two raters was greater than or equal to .95 for the total number of sleep spindles, the total number of rapid eye movements, as well as the number of minutes in Stage 2, SWS, and REM. The correlation was slightly lower (.93) for the number of minutes in Stage 1. We are therefore quite confident in our data and the results generated from them.
2. We specifically thank two anonymous reviewers for bringing these two points to our attention.

REFERENCES

Ackerman, P. L. (1987). Individual differences in skill learning: An integration of psychometric and information processing perspectives. *Psychological Bulletin, 102,* 3–27.

Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. *Annual Review of Neuroscience, 9,* 357–381.

Buchegger, J., Fritsch, R., Meier-Koll, A., & Riehle, H. (1991). Does trampolining and anaerobic physical fitness affect sleep. *Perceptual and Motor Skills, 73,* 243–253.

Cohen, J., & Cohen, P. (1983). *Applied multiple regression/correlation analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Darchia, N., Campbell, I. G., Palagini, L., & Feinberg, I. (2004). Rapid eye movement density shows trends across REM periods but is uncorrelated with NREM delta in young and elderly human subjects. *Brain Research Bulletin, 63,* 433–438.

Datta, S., & Patterson, E. H. (2003). Activation of phasic pontine wave (p-wave): A mechanism of learning and memory processing. In P. Maquet, C. Smith, & R. Stickgold (Eds.), *Sleep and brain plasticity* (pp. 135–156). Oxford: Oxford University Press.

Destexhe, A., & Sejnowski, T. J. (2001). *Thalamocortical assemblies: How ion channels, single neurons and large-scale networks organize sleep oscillations* (Vol. 49). Oxford: Oxford University Press.

Doyon, J., & Ungerleider, L. G. (2002). Functional anatomy of motor skill learning. In L. R. Squire & D. L. Schacter (Eds.), *Neuropsychology of memory* (pp. 225–238). New York: Guilford Press.

Finkbeiner, S. (2000). Calcium regulation of the brain-derived neurotrophic factor gene. *Cellular and Molecular Life Sciences, 57,* 394–401.

Fischer, S., Hallschmid, M., Elsner, A. L., & Born, J. (2002). Sleep forms memory for finger skills. *Proceedings of the National Academy of Sciences, U.S.A., 99,* 11987–11991.

Fogel, S. M., & Smith, C. T. (2006). Learning-dependent changes in sleep spindles and Stage 2 sleep. *Journal of Sleep Research, 15,* 250–255.

Frutiger, S. A., Strother, S. C., Anderson, J. R., Sidtis, J. J., Arnold, J. B., & Rottenberg, D. A. (2000). Multivariate predictive relationship between kinematic and functional activation patterns in a PET study of visuomotor learning. *Neuroimage, 12,* 515–527.

Gaillard, J. M., & Blois, R. (1981). Spindle density in sleep of normal subjects. *Sleep, 4,* 385–391.

Gauthier, I., Tarr, M. J., Anderson, A. W., Skudlarksi, P., & Gore, J. C. (1999). Activation of the middle fusiform ''face area'' increases with expertise in recognizing novel objects. *Nature Neuroscience, 2,* 568–573.

Grafton, S. T., Mazziotta, J. C., Presty, S., Friston, K. J., Frackowiak, R. S. J., & Phelps, M. E. (1992). Functional anatomy of human procedural learning determined with regional cerebral blood flow and PET. *Journal of Neuroscience, 12,* 2542–2548.

Huber, R., Ghilardi, M. F., Massimini, M., & Tononi, G. (2004). Local sleep and learning. *Nature, 430,* 78–81.

Jackson, D. N. (1998). *Multidimensional aptitude battery-II.* Port Huron, MI: Sigma Assessment Systems.

Jenkins, I. H., Brooks, D. J., Nixon, P. D., Frackowiak, R. S. J., & Passingham, R. E. (1994). Motor sequence learning: A study with positron emission tomography. *Journal of Neuroscience, 14,* 3775–3790.

Mallick, B. N., & Gulyani, S. (1996). Alterations in synaptosomal calcium concentrations after rapid eye movement sleep deprivation in rats. *Neuroscience, 75,* 729–736.

Maquet, P., Laureys, S., Peigneux, P., Fuchs, S., Petiau, C., Phillips, C., et al. (2000). Experience-dependent changes in cerebral activation during human REM sleep. *Nature Neuroscience, 3,* 831–836.

Mavanji, V., & Datta, S. (2003). Activation of the phasic pontine-wave generator enhances improvement of learning performance: A mechanism for sleep-dependent plasticity. *European Journal of Neuroscience, 17,* 359–370.

Mavanji, V., Ulloor, J., Saha, S., & Datta, S. (2004). Neurotoxic lesions of phasic pontine-wave generator cells impair retention of 2-way active avoidance memory. *Sleep, 27,* 1282–1292.

Nader, R., & Smith, C. (2003). A role for stage 2 sleep in memory processing. In P. Maquet, C. Smith, & R. Stickgold (Eds.), *Sleep and brain plasticity* (pp. 87–98). Oxford: Oxford University Press.

Ohno, H., Unushihara, R., Sei, H., & Morita, Y. (2002). REM sleep deprivation suppresses acquisition of classical eyeblink conditioning. *Sleep, 25,* 877–881.

Peigneux, P., Laureys, S., Fuchs, S., Collette, F., Perrin, F., Reggers, J., et al. (2004). Are spatial memories strengthened in the human hippocampus during slow wave sleep? *Neuron, 44,* 535–545.

Peigneux, P., Laureys, S., Fuchs, S., Delbeuck, X., Degueldre, C., Aerts, J., et al. (2003). Generation of rapid eye movements during paradoxical sleep in humans. *Neuroimage, 14,* 701–708.

Plihal, W., & Born, J. (1997). Effects of early and late nocturnal sleep on declarative and procedural memory. *Journal of Cognitive Neuroscience, 9,* 534–547.

Plihal, W., & Born, J. (1999). Effects of early and late nocturnal sleep on priming and spatial memory. *Psychophysiology, 36,* 571–582.

Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized terminology, techniques and scoring systems of human subjects.* Los Angeles: Brain Information Service/Brain Research Institute.

Sakai, K., Hikosaka, O., Miyauchi, S., Takino, R., Sasaki, Y., & Putz, B. (1998). Transition of brain activation from frontal to parietal areas in visuomotor sequence learning. *Journal of Neuroscience, 18,* 1827–1840.

Sei, H., Saitoh, D., Yamamoto, K., Morita, K., & Morita, Y. (2000). Differential effect of short term REM sleep deprivation on NGF and BDNF protein levels in the rat brain. *Brain Research, 877,* 387–390.

Smith, C. (1993). REM sleep and learning. In A. Moffitt, M.

Kramer, & R. Hoffmann (Eds.), *The functions of dreaming* (pp. 341–361). New York: SUNY Press.
Smith, C. (2001). Sleep states and memory processes in humans: Procedural versus declarative memory systems. *Sleep Medicine Reviews, 5,* 491–506.
Smith, C. (2003). The REM sleep window and memory processing. In P. Maquet, C. Smith, & R. Stickgold (Eds.), *Sleep and brain plasticity* (pp. 117–133). Oxford: Oxford University Press.
Smith, C., & MacNeill, C. (1994). Impaired motor memory for a pursuit rotor task following Stage 2 sleep loss in college students. *Journal of Sleep Research, 3,* 206–213.
Smith, C. T., Aubrey, J. B., & Peters, K. R. (2004). Different roles for REM and Stage 2 sleep in motor learning. *Psychologica Belgica, 44,* 79–102.
Smith, C. T., Nixon, M. R., & Nader, R. S. (2004). Posttraining increases in REM sleep intensity implicate REM sleep in memory processing and provide a biological marker of learning potential. *Learning and Memory, 11,* 714–719.
Steriade, M. (1999). Coherent oscillations and short-term plasticity in corticothalamic networks. *Trends in Neuroscience, 22,* 337–345.
Steriade, M., & Amzica, F. (1998). Coalescence of sleep rhythms and their chronology in corticothalamic networks. *Sleep Research Online, 1,* 1–10.
Stickgold, R., LaTanya, J., & Hobson, J. A. (2000). Visual discrimination learning requires sleep after training. *Nature Neuroscience, 3,* 1237–1238.
Wagner, U., Gais, S., & Born, J. (2001). Emotional memory formation is enhanced across sleep intervals with high amounts of rapid eye movement sleep. *Learning and Memory, 8,* 112–119.
Wagner, U., Hallschmid, M., Verleger, R., & Born, J. (2003). Signs of REM sleep dependent enhancement of implicit face memory: A repetition priming study. *Biological Psychology, 62,* 197–210.
Walker, M. P., Brakefield, T., Morgan, A., Hobson, J. A., & Stickgold, R. (2002). Practice with sleep makes perfect: Sleep-dependent motor skill learning. *Neuron, 35,* 205–211.
Walker, M. P., & Stickgold, R. (2006). Sleep, memory, and plasticity. *Annual Review of Psychology, 57,* 139–166.