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The Role of Sleep in Forgetting in Temporal Lobe Epilepsy: A Pilot Study

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Abstract

The purpose of this study is to examine how sleep impacts memory function in temporal lobe epilepsy (TLE). TLE patients (n=7) and control subjects (n=9) underwent training and overnight testing on (1) a motor sequence task (MST) known to undergo sleep-dependent enhancement in healthy subjects, and (2) the selective reminding test (SRT), a verbal memory task on which TLE patients have shown impaired performance 24 hours after training. Sleep data was collected by polysomnography. Results indicate that TLE patients display greater forgetting on the SRT compared to controls over 12 hours of daytime wake, but not over a similar period including a night of sleep. Slow wave sleep is correlated with overnight performance change on the SRT. TLE patients show no deficit in sleep-dependent MST improvement. The findings provide potential insight into the pattern and pathophysiology of forgetting in TLE.

Keywords

Sleep; Temporal Lobe Epilepsy; Cognition; Memory

Introduction

Subjective memory impairment and objective memory abnormalities on neuropsychological testing are common in patients with temporal lobe epilepsy (TLE) ^{1,2}. In addition, several types of memory dysfunction have been described in TLE that may not be apparent on standard testing ³. Accelerated long-term forgetting (ALF) has been described in several case series and case reports of TLE patients ^{3–5}. Patients who experience this phenomenon learn normally but forget information more quickly than expected, a process that can be observed over days or weeks. While the etiology of ALF is unclear, one hypothesis that has not hitherto been explored is the role of sleep in ALF and other cognitive deficits seen in TLE. Over the past decade, an increasing amount of evidence has suggested that sleep plays an important role in consolidation of different types of declarative and non-declarative

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memory^{6,7}. Sleep disturbance and subjective sleep complaints are common in TLE^{8,9}, although they have not been explored as potential contributors to memory dysfunction in this population. The purpose of this study is to evaluate the role of sleep in cognitive performance in TLE by (i) determining whether TLE patients demonstrate overnight improvement on a procedural motor sequence learning task on which healthy subjects show sleep-dependent improvement¹⁰ and (ii) evaluating whether TLE patients perform differently when tested after a period of wake versus a period of sleep on a verbal memory test on which such patients have previously been found to perform more poorly than controls at 24-hour retest⁵.

Methods

Participants

Outpatients (n=7) with probable or definite (n=6) temporal lobe epilepsy (TLE) were recruited from a tertiary care epilepsy referral clinic. Definite TLE was defined by documentation of temporal lobe focus on continuous EEG/video monitoring, and probable TLE was defined by seizure semiology, in combination with interictal EEG, neuroimaging and clinical history¹¹. Patients were on stable anti-epileptic drug (AED) regimens, with no new AEDs added in the 2 months prior to enrollment, and no change in dose of existing AEDs in the 1 month prior to enrollment. Two TLE patients did undergo medication alterations as part of a scheduled admission for continuous video-EEG monitoring within the study period. In the TLE patients who underwent continuous video-EEG monitoring, medications were stable for approximately one week prior to any individual testing block. None of the patients had a history of epilepsy surgery. Healthy control subjects (n=9) were recruited from the community by newspaper and internet advertisements. Both healthy and TLE subjects were screened to exclude known sleep or memory disorders. In order to estimate overall intellectual ability, subjects were administered the Wechsler Abbreviated Scale of Intelligence (WASI). None of the participants were regularly taking medication to enhance sleep. The Institutional Review Board of Brigham and Women's Hospital and Beth Israel Deaconess Medical Center approved the study, and informed written consent was obtained from each participant. See Table 1 for characteristics of patients and control subjects. See Supplementary Table 1 for further details on TLE patients.

Finger Tapping Motor Sequence Task

During training on the finger tapping motor sequence task (MST)¹⁰, and again at retest 24 hr later, subjects were shown a numeric sequence of five numbers on a computer screen (e.g., 4-1-3-2-4). Using their left hand, subjects performed twelve 30-second trials during which they repeatedly typed the displayed sequence as quickly and accurately as possible. There were 30-second pauses between trials. Each trial was scored for the number of correct sequences typed, as well as the number of errors. Training occurred between 8AM and 4PM, and testing took place 24 hours later.

Selective Reminding Test

During the selective reminding test (SRT)¹², subjects were read a list of 12 unrelated words at a rate of one word every 2 seconds, and were immediately asked to recall verbally as many words as possible, in any order. Patients were then selectively reminded verbally of those words they omitted on the previous trial, and were again asked to recall all 12 words. Selective reminding followed by recall continued until two consecutive trials yielded recall of all 12 words, or until twelve trials were completed. After a 30-minute delay period, during which subjects completed questionnaires and watched an episode of a television sitcom during any remaining time, they again recalled as many of the studied words as possible. Approximately 12 hours later, recall was again performed. After this final recall,

subjects were asked to identify the original 12 words from a list containing 20 additional new words, with all 32 words presented on a single sheet of paper.

All subjects underwent the SRT protocol twice using different word lists – once with baseline testing in the morning and retest approximately 12 hours later in the evening (Wake condition), and a second time with baseline testing in the evening and retest approximately 12 hours later the following morning (Sleep condition). Subjects were instructed not to nap during the day between morning baseline testing and evening retesting. Studied word lists were identical to Forms 2 and 3 of Hannay and Levin¹³. Use of the two word lists was counterbalanced across conditions.

To eliminate the possibility that poor performance at 12 hours in the TLE group was a function of inadequate learning at baseline, only TLE and control subjects who recalled 11 or 12 words by the end of training were included in the final analysis (7 of 9 controls, 6 of 7 TLE). However, even with all subjects included, there still were no significant differences in performance during the final trial at baseline ($p=0.65$ for the Wake condition; $p=0.74$ for the Sleep condition), and no differences at the 0-min test, either between groups or across conditions (mixed model, condition [AM, PM, repeated] $\times$ group [control, TLE] ANOVA: main effect of condition, $p=0.24$; main effect of group, $p=0.72$; interaction, $p=0.51$) suggesting that the two groups had comparable learning potential.

Overnight Sleep Recordings

Participants were wired for standard polysomnographic (PSG) sleep recordings the evening of the SRT Sleep condition. Recordings took place in the General Clinical Research Center of Brigham and Women's Hospital, Boston. EEG (C3, C4, Cz, O1, O2, F3, F4, A1, A2), EOG, submental EMG, nasal pressure, rib cage and abdominal movements, and lower extremity EMG were recorded on an Embla A10 ambulatory monitor (Medcare Systems, Buffalo NY). Temporal leads (T3 and T4) were added for TLE patients. EEG data was sampled at 200 Hz and band-pass filtered between .3 and 35 Hz for analysis. Two TLE patients underwent the overnight portion of the study during their first night of a planned admission for continuous video-EEG monitoring already scheduled for clinical purposes. For these two patients, EOG, submental EMG, nasal pressure, rib cage and abdominal movement channels, and lower extremity EMG were added to the standard LTM 22-lead scalp EEG leads for the first night of their LTM admission. PSG records were scored for sleep stages¹⁴ and for arousals, respiratory events, and periodic limb movements of sleep¹⁵.

Actigraphy

Participants wore a Mini-Mitter Actiwatch-64 actigraph (Philips Respironics, Bend, OR) on their wrist from the first visit of the first study block until study completion. The actigraph monitors and records wrist movement in 1-minute epochs, and provides an estimates of sleep and nap time based on periods of wrist immobility.

Experimental Design

After a screening visit, subjects were scheduled for three test blocks, with a minimum of 4 days between blocks. The MST protocol was run during the first block, and the SRT protocol during blocks 2 and 3 with the order of conditions (Wake, Sleep) counterbalanced. All participants maintained sleep diaries for the duration of the study (Block 1 through Block 3), and TLE patients additionally maintained seizure diaries. Subjective sleepiness was assessed prior to each testing session using the Stanford Sleepiness Scale. Subjects were asked to abstain from alcohol during testing blocks from 24 hours prior to each training session until the end of testing for that block. TLE patients continued their normal schedules of anti-epileptic drugs, taking them at approximately 12-hour intervals. Although caffeine

intake was not regulated, there was no difference in average daily caffeine intake between the groups.

Results

Participants

Table 1 compares characteristics of TLE and control subjects. There were no significant differences in age, education, sleep quality indexes, or IQ scores. TLE patients did have significantly higher scores on the Beck Depression Inventory, although the mean score in TLE subjects was less than 10, the threshold for clinically significant mood disturbance. Supplementary Table 1 provides additional details about each TLE participant.

Finger Tapping Motor Sequence Task (MST)

TLE patients did not differ from control subjects in performance speed on the motor sequence task (MST, Fig. 1) either during the training session (first trial, $p=0.55$; final 3 trials, $p=0.79$), or at retest 24 hr later (initial improvement [last 3 training trials vs. first 3 retest trials], $p=0.23$; plateau learning [last 3 training trials vs. last 3 retest trials], $p=0.63$; Fig. 1 inset). See Supplementary Table 2 for additional details regarding MST performance.

Selective Reminding Test (SRT)

In the Sleep condition, no significant difference in recall was seen between TLE and control subjects (Fig. 2A). A mixed model ANOVA of group (TLE, control) $\times$ time (0, 30 min, 12 hours; repeated) showed a main effect of time ($F=8.6$, $p=0.0007$), but no main effect of group ($F=0.4$, $p=0.54$) and no interaction between group and time ($F=0.6$, $p=0.50$). In addition, no difference between TLE and control subjects was seen at 30-minute recall (9.8 versus 11.0; $p=0.17$) or at 12-hour recall (9.3 versus 9.1; $p=0.90$).

In contrast, significant group differences in recall were observed in the Wake condition (Fig. 2B). A mixed model ANOVA of group (TLE, control) $\times$ time (0, 30 min, 12 hours; repeated) showed a main effect of time ($F=23.9$, $p<0.001$) as well as main effect of group ($F=11.7$, $p=0.006$) and a significant group by time interaction ($F=9.1$, $p=0.01$). Post-hoc *t*-tests revealed no significant group differences at 30-minute recall (10 vs. 11.4; $p=0.07$). However, at 12 hours, the TLE group recalled 30% fewer words than controls (7.2 vs. 10.3; $p=0.005$). Similar results were obtained when the percent change in recall between the final training trial and 12 hour retest session was analyzed (group $\times$ condition interaction, $F=4.9$, $p=0.05$; Fig. 3), with significantly greater forgetting in the TLE group in the wake condition (37% vs. 12%, $p=0.009$), but not in the sleep condition (19% vs. 23%, $p=0.72$). Adding Beck depression scores, which differed between groups, as a covariate also had no impact (main effect of group: $F=10.0$, $p=0.01$; main effect of Beck: $F=1.2$, $p=0.31$).

SRT Performance and SWS

Overnight changes in performance on declarative memory tasks have often been associated with the amount of slow wave sleep (SWS) during the intervening night^{16–18}, and especially with the SWS in the first half of the night¹⁹. In the current study, for all subjects combined, deterioration in performance between the zero time and 12 hr recall sessions correlated significantly with percent SWS in the entire night ($r=0.64$, ANCOVA – group: $F=1.1$, $p=0.31$, SWS%: $F=8.2$, $p=0.02$; Fig. 4), and approached significance in the first half of the night ($r=0.59$, ANCOVA – group: $F=0.6$, $p=0.46$, SWS%: $F=4.1$, $p=0.074$; not shown). Separate analyses of the correlation with total SWS for TLE patients and controls gave numerically stronger correlations, which failed to reach significance because of smaller sample sizes (controls: $r=0.69$, $p=0.088$; TLE: $r=0.70$, $p=0.190$; Fig 4).

Sleepiness and Overnight Sleep Recordings

There was no significant difference between TLE and control subjects in total sleep time or in the percentage of any individual stage of sleep either during the night as a whole or during any individual quartile of the night. No electrographic seizures were recorded during overnight polysomnography. Two subjects (one TLE and one control) met criteria for mild obstructive sleep apnea ($5 < \text{Apnea Hypopnea Index} < 15$) based on polysomnographic recordings. Repetition of all of the analyses without these subjects did not change the results substantively. See Supplementary Table 3 for additional details regarding sleep architecture.

Stanford Sleepiness Scale scores (SSS) did not significantly differ between the TLE and control groups either in the Wake condition at baseline (unpaired t-test: 2.7 versus 2.1; $p=0.39$) or at retest (2.8 versus 2.7; $p=0.86$) or in the Sleep condition at baseline (2.0 versus 2.3; $p=0.48$) or at retest (2.8 versus 2.6; $p=0.55$).

Actigraphy

Based on actigraphic data collected over an average of 3 weeks, there was no significant difference between the groups in either mean nocturnal sleep time (TLE: 394 min, controls: 392 min; $p=0.94$) or sleep efficiency (TLE: 87%, controls: 84%; $p=0.15$). There were also no differences in the frequency or length of naps.

Seizure Diaries

Based on subject self-report of seizure occurrence, one participant reported a simple partial seizure on the first day of the MST. Otherwise, subjects denied any clinical seizures within 5 days of initiating any of the individual testing blocks.

Discussion

In this study, TLE subjects, who were equivalent to controls in terms of sleep quality, sleep architecture, and total sleep time, as measured by both polysomnography and actigraphy, showed accelerated long term forgetting (ALF) on a declarative memory task across 12 hr of daytime wakefulness (7.2 vs. 10.3 words recalled, $p=0.005$), similar in magnitude to that previously reported across a 24-hour period (7.3 vs. 9.8 words recalled)⁵. In contrast, forgetting across 12 hours including a night of sleep was similar for both groups (9.1 vs. 9.3 words recalled, $p=0.90$). Both groups also showed similar sleep-dependent enhancement of procedural learning on a motor sequence task (10.8 vs. 14.0%, $p=0.63$). Although the results must be confirmed with further studies, this data provides potential insight into the pattern of forgetting in TLE patients and raises additional questions regarding the underlying pathophysiology. Most simply, sleep appears to protect the TLE brain against accelerated forgetting.

Accelerated long term forgetting implies a defect in memory consolidation. Previous suggestions for an underlying cause of ALF have included ongoing seizures, AED effects, structural lesions (hippocampal pathology), and psychiatric disorders or psychosocial factors such as self-esteem.³ Although the TLE subjects in our study had higher scores on the Beck Depression Inventory compared to controls, this did not explain the group difference on a measure of long-term memory. Also, depressed patients have not shown accelerated forgetting compared to controls on a picture recognition task when retested at 24 hours.²⁰

The cause of the increased forgetting (compared to normal controls) across the day is unclear. It could reflect chronic damage to the memory structures involved in TLE. Alternatively, it could be a real-time effect of interictal discharges or subclinical seizure activity during the day that either disrupts recently formed memories or impairs daytime

memory consolidation. Though there is some recent evidence relating interictal discharges to cognitive disruption in animal models ²¹, this has not been fully explored in humans. We were not able to note such an association in our study, as most of the TLE patients did not undergo continuous EEG monitoring during the day. Neither chronicity ($r=0.60$, $p=0.20$) nor partial seizure frequency ($r=0.39$, $p=0.45$) predicted the extent of daytime ALF seen.

An important question that cannot yet be answered is why ALF might occur differentially over a period of wake versus a period of sleep in TLE patients. One possible explanation is that TLE patients may be quicker to fatigue (related to TLE itself or to specific AEDs) resulting in a decrement in performance over the day. However Stanford Sleepiness Scores were similar at morning and evening SRT test (main effect of time: $p=0.74$, of group: $p=0.64$; interaction: $p=0.40$), as was training performance (main effect of time: $p=0.77$, of group: $p=0.92$; interaction: $p=0.47$). More interesting is the possibility that sleep provides protection against forgetting in TLE. This could result from passive protection against the impact of interference from daytime activities, active consolidation and stabilization of the memories, or a combination of both ^{6,7}. The finding that post-sleep performance on the SRT, a declarative memory task, correlated with the percentage of slow wave sleep in our study provides supporting evidence that active sleep-dependent memory consolidation is providing protection against forgetting in TLE. Consolidation of hippocampal-dependent memory has a known association with slow wave sleep, which has been demonstrated both in animal ¹⁶ and human subjects ^{17,19}. TLE and control subjects in this study exhibited equal percentages of slow wave sleep, which is likely contributing to their equivalent performance on this declarative memory task after a night of sleep.

Considering the small sample size and the preliminary nature of the findings, more definitive studies should be performed with a larger sample size. Future studies could look at forgetting over several time periods, such as 12 hours, 24 hours and a longer time interval of 2 to 6 weeks. This would permit a better understanding of the role of sleep as well as the role of other variables such as seizure frequency. Additionally, continuous EEG monitoring between memory tests during the day would be a means of exploring the possible role of interictal discharges or subclinical seizure activity in forgetting.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. Elger CE, Helmstaedter C, Kurthen M. Chronic epilepsy and cognition. *Lancet Neurol*. 2004 Nov; 3(11):663–72. [PubMed: 15488459]
2. Fisher RS, Vickrey BG, Gibson P, Hermann B, Penovich P, Scherer A, et al. The impact of epilepsy from the patient's perspective I. Descriptions and subjective perceptions. *Epilepsy Res*. 2000 Aug; 41(1):39–51. [PubMed: 10924867]
3. Butler CR, Zeman AZ. Recent insights into the impairment of memory in epilepsy: transient epileptic amnesia, accelerated long-term forgetting and remote memory impairment. *Brain*. 2008 Sep; 131(Pt 9):2243–63. [PubMed: 18669495]

4. Blake RV, Wroe SJ, Breen EK, McCarthy RA. Accelerated forgetting in patients with epilepsy: evidence for an impairment in memory consolidation. *Brain*. 2000 Mar; 123(Pt 3):472–83. [PubMed: 10686171]
5. Martin RC, Loring DW, Meador KJ, Lee GP, Thrash N, Arena JG. Impaired long-term retention despite normal verbal learning in patients with temporal lobe dysfunction. *Neuropsychology*. 1991; 5(1):3–12.
6. Stickgold R. Sleep-dependent memory consolidation. *Nature*. 2005; 437:1272–1278. [PubMed: 16251952]
7. Ellenbogen JM, Hulbert JC, Jiang Y, Stickgold R. The sleeping brain's influence on verbal memory: boosting resistance to interference. *PLoS One*. 2009; 4(1):e4117. [PubMed: 19127287]
8. Bazil CW. Epilepsy and sleep disturbance. *Epilepsy Behav*. 2003 Oct; 4(Suppl 2):S39–45. [PubMed: 14527482]
9. Bazil CW, Castro LH, Walczak TS. Reduction of rapid eye movement sleep by diurnal and nocturnal seizures in temporal lobe epilepsy. *Arch Neurol*. 2000 Mar; 57(3):363–8. [PubMed: 10714662]
10. Walker MP, Brakefield T, Morgan A, Hobson JA, Stickgold R. Practice with sleep makes perfect: sleep-dependent motor skill learning. *Neuron*. 2002 Jul 3; 35(1):205–11. [PubMed: 12123620]
11. Bell BD. WMS-III Logical Memory performance after a two-week delay in temporal lobe epilepsy and control groups. *J Clin Exp Neuropsychol*. 2006 Nov; 28(8):1435–43. [PubMed: 17050268]
12. Buschke H, Fuld PA. Evaluating storage, retention, and retrieval in disordered memory and learning. *Neurology*. 1974 Nov; 24(11):1019–25. [PubMed: 4473151]
13. Hannay HJ, Levin HS. Selective reminding test: an examination of the equivalence of four forms. *J Clin Exp Neuropsychol*. 1985 Jun; 7(3):251–63. [PubMed: 3998090]
14. Rechtschaffen, A.; Kales, A. A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects. Brain Information Service, University of California; Los Angeles: 1968.
15. Iber, C.; Ancoli-Israel, S.; Chesson, A.; Quan, SF., editors. Manual for the Scoring of Sleep and Associated Events: Rules, Terminology, and Technical Specifications. Westchester: American Academy of Sleep Medicine; 2007.
16. Lee AK, Wilson MA. Memory of sequential experience in the hippocampus during slow wave sleep. *Neuron*. 2002 Dec 19; 36(6):1183–94. [PubMed: 12495631]
17. Peigneux P, Laureys S, Fuchs S, Collette F, Perrin F, Reggers J, et al. Are spatial memories strengthened in the human hippocampus during slow wave sleep? *Neuron*. 2004 Oct 28; 44(3): 535–45.
18. Backhaus J, Born J, Hoeckesfeld R, Fokuhl S, Hohagen F, Junghanns K. Midlife decline in declarative memory consolidation is correlated with a decline in slow wave sleep. *Learn Mem*. 2007 May; 14(5):336–41. [PubMed: 17522024]
19. Plihal W, Born J. Effects of early and late nocturnal sleep on declarative and procedural memory. *J Cogn Neurosci*. 1997; 9(4):534–547.
20. Lewis P, Kopelman MD. Forgetting rates in neuropsychiatric disorders. *J Neurol Neurosurg Psychiatry*. 1998 Dec; 65(6):890–8. [PubMed: 9854966]
21. Kleen JK, Scott RC, Holmes GL, Lenck-Santini PP. Hippocampal interictal spikes disrupt cognition in rats. *Ann Neurol*. 2010 Feb; 67(2):250–7. [PubMed: 20225290]

Research Highlights

- TLE patients display greater forgetting on a verbal memory task compared to controls over 12 hours of daytime wake, but not over a similar period including a night of sleep.
- Slow wave sleep is correlated with overnight performance change on a verbal memory task.
- TLE patients show no deficit in sleep-dependent improvement on a motor sequence task compared to controls.
- Periods of sleep may provide protection against forgetting in TLE.

Figure 1. Motor Sequence Task (MST) performance, measured as the number of correct sequences typed during a 30-second trial

Control subjects (light grey circles) and TLE patients (dark grey circles) did not significantly differ either during baseline learning (left) or during retest after the passage of 24 hours, including a night of sleep (right). Insert chart displays plateau learning, i.e. the percent change in performance from the last 6 trials at training to the last 6 trials at retest in control subjects (light grey) and TLE patients (dark grey) ($p=0.63$).

Figure 2. Selective Reminding Test (SRT)

Mean free recall for healthy controls (light grey circles) and TLE patients (dark grey circles) across time. A: last evening trial, 30-min. delay, 12 hr (sleep) delay; B: last morning trial, 30-min. delay, 12 hr (wake) delay. TLE patients have significantly worse recall at 12 hrs, but only after a period of wake (B), and not after a period of sleep (A).

Figure 3. Percent change in mean free recall on the SRT between final baseline trial and retest 12 hrs later

Results for healthy controls (light grey bars) and TLE patients (dark grey bars) are illustrated over a period of wake (left) and a period of sleep (right). TLE subjects worsened significantly over wake, but not over sleep. Group $\times$ time interaction as measured by a mixed model ANOVA ($F=4.9$, $p=0.05$).

Figure 4. Correlation of the percent change in SRT performance (between the last trial at baseline and retest 12 hours later) and slow wave sleep as percent of total sleep time
For all subjects combined, percent change in SRT performance correlated significantly with percent SWS during the entire night ($r=0.64$, $p=0.02$). Correlation plots for TLE patients (dark grey) and control subjects (light grey) are also shown, although they did not reach significance.

Table 1

Subject Characteristics

	TLE	Control	p-value (unpaired t-test)
Age (yrs)	44.0	44.7	0.90
Education (yrs)	15.6	16.2	0.60
Beck Depression Inventory	9.9	1.3	0.002
Pittsburgh Sleep Quality Index	6.1	4.6	0.42
Epworth Sleepiness Score	6.4	5.0	0.44
Insomnia Severity Index	11.0	6.2	0.11
Edinburgh Handedness Scale	89.3	77.2	0.47
Verbal IQ	103.4	112.2	0.21
Performance IQ	108.4	113.1	0.59
Full Scale IQ	106.7	114.3	0.32

TLE=Temporal Lobe Epilepsy Patients