**Background:** Patients with brief, complex partial seizures frequently suffer from tiredness and decreased productivity that continue well beyond the postictal period. A possible explanation is that seizures, even when occurring during the day, disrupt sleep the following night.

**Objective:** To determine the effect of temporal lobe complex partial seizures on sleep structure and daytime drowsiness.

**Methods:** Patients with temporal lobe epilepsy were admitted for video-electroencephalography monitoring. All-night polysomnography was recorded under the following 3 conditions: seizure free, seizure during the day before the recording, and seizure during the recording. Percentage of time in each sleep stage, sleep efficiency, and time to first and second rapid eye movement (REM) period were compared for seizure vs control conditions. Daytime drowsiness was also measured, using a modified maintenance of wakefulness test and 2 subjective drowsiness tests.

**Results:** Daytime seizures reduced REM from 18% ± 1% to 12% ± 2% (P = .003). Night seizures reduced REM from 16% ± 1% to 6.8% ± 2% (P < .001). Night seizures also significantly reduced stages 2 and 4 while increasing stage 1 sleep. Night seizures, but not day seizures, significantly reduced sleep efficiency, increased time to first REM period, and increased drowsiness as measured by the maintenance of wakefulness test.

**Conclusions:** Temporal lobe complex partial seizures decrease REM sleep, particularly when occurring during sleep but also when occurring on the previous day. This may, in part, be responsible for the prolonged impairment of functioning that some patients report following seizures.

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PATIENTS AND METHODS

To examine a relatively homogeneous population, only patients with temporal lobe partial epilepsy (based on interictal and ictal recordings) were included. Patients with known or suspected sleep disorders were excluded, although no specific testing for sleep apnea or periodic limb movements was performed. No patient had exclusively diurnal or nocturnal seizures. Patients were admitted consecutively to the Epilepsy Monitoring Unit at Columbia-Presbyterian Medical Center, New York, NY, for diagnosis or surgical evaluation. Temporal onset seizures were verified by video-electroencephalographic (EEG) monitoring, and computerized seizure detection ensured that un-witnessed seizures were detected. Most patients were receiving maintenance anticonvulsant therapy, although this was typically decreased and sometimes discontinued during the admission. Patients taking or withdrawing from barbiturate or benzodiazepine therapy were excluded (including patients who received a single dose of a benzodiazepine following a seizure cluster). Beverages containing caffeine were not allowed.

In addition to the usual 10-20 array of scalp electrodes, patients had bilateral outer canthus electrodes and chin electromyograph electrodes plus a subtentorial chain of electrodes (total, 29 scalp electrodes). Seizures were diagnosed using video-EEG with the full scalp array. The subjects were not sleep deprived on the nights before recording or during recording and were not allowed daytime naps. Nurses instructed the patients to sleep at 11 PM and awakened them at 7 AM; however, precise “lights-off” times were not recorded. Although the patients were in shared rooms on a hospital ward, efforts were made to optimize sleeping conditions. During recordings, the doors of the room remained closed, the lights off, and the patients undisturbed unless a seizure occurred. Following seizures, patients were encouraged to return to sleep. If a benzodiazepine was given after prolonged or repeated seizures, the night was not included in the analysis.

Polysomnography was scored in 30-second epochs, according to standard technique, by reformatting digital EEG to polysomnographic channels and settings. Because there was not always a precise lights-off time, scoring began at sleep onset (defined by 3 consecutive epochs of stage 1 or 1 epoch of stage 2 sleep) and continued until awakening in the morning by the staff. Therefore, sleep latency could not be determined, and total recording time varied somewhat. Sleep efficiency is generally defined as time asleep as a percentage of time in bed (lights off to lights on). For this study, sleep efficiency was calculated somewhat differently from the standard, as percentage of time asleep from sleep onset until awakening. This would likely result in higher numbers, but comparisons between groups should still be valid. Sleep following a seizure was scored as postictal sleep until the first epoch of normal stage 2 sleep or wakefulness was seen; after that point, the usual criteria for sleep staging were used. Seizure and postictal epochs were not included in total sleep for our purposes. This modification of the original Rechtschaffen and Kales criteria was essential, as diffuse delta typically seen after seizures is clearly not synonymous with any of the accepted sleep stages. Using our method, stages 3 and 4 sleep possibly could not be underestimated; however, it would be uncommon for patients to pass directly into these stages without first entering stage 2. Similarly, mild slowing and attenuation seen postictally would not be scored as stage 1 sleep, and this stage could be underestimated.

The initial night of recording was not used in the analysis (to control for first-night effects); however, patients adhered to the sleep schedule on that night.

Control polysomnograms were defined as no seizure for at least 24 hours before sleep onset and during the recording. Polysomnograms following daytime seizures (DAYSZ condition) were defined as a seizure between 7 AM and 11 PM on the day the recording began. Polysomnograms with night seizures (NTSZ condition) were defined as a seizure during the recording (after sleep onset). Patients were identified who had at least 1 control and 1 DAYSZ and/or NTSZ polysomnogram; these were used in the subsequent analysis. Sleep efficiency, percentage of time in each sleep stage, time to first and second rapid eye movement (REM) period, and total sleep time were calculated. For nights with seizures, a subanalysis was made when seizures occurred before or after the first REM period. All results were compared using a paired t test vs controls. When more than 1 suitable recording was obtained, the results were averaged before comparisons using the paired t test.

Between 1 and 3 PM daily, patients were administered 3 tests of drowsiness. Subjective measures included the Stanford Sleepiness Scale (SSS), where the patient chooses a description giving a numerical result from 1 to 7, and a linear analog scale of drowsiness (LASD). For the latter, patients were asked to mark their drowsiness on a 100-mm line, from alert to sleepy, yielding a result from 0 to 100. In addition, a more objective nap test was performed that was a modification of the maintenance of wakefulness test (MOW). For our MOW, patients were placed in a quiet, dark room in the supine position and instructed to stay awake. The time to sleep was determined by 3 consecutive stage 1 epochs or a single epoch of another sleep stage, giving a number from 0 to 20. The standard MOW includes 3 scheduled recordings in a day. The SSS and LASD were administered immediately before each MOW. All tests were performed on days meeting the inclusion criteria for the polysomnogram portion of the study, and therefore were not performed on the day of admission or on the following day. The SSS, LASD, and MOW all give numerical results that were compared using a paired t test. When more than 1 suitable test was obtained, the results were averaged before comparisons using the paired t test. When data were not normally distributed, nonparametric statistics were used.

SLEEP STAGES

Daytime seizures did not result in any significant changes in sleep stages 1 through 4; however, REM period was

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significantly reduced (12% ± 2% vs 18% ± 1%; P = .003). Night seizures caused an increase in stage 1 sleep (15% ± 2% vs 11% ± 2%; P = .002) and decreases in stage 2 sleep (47% ± 3% vs 56% ± 2%; P = .004), stage 4 sleep (0.9% ± 0.5% vs 2.4% ± 0.9%; P = .046), and REM sleep (6.8% ± 2% vs 16% ± 1%; P < .001) (Figure 1). For nights when the seizure occurred before the first REM period (n = 7), REM sleep was further reduced (3% ± 1% vs 16% ± 2%; P = .002).

TIME TO FIRST REM

Daytime seizures increased the time from sleep onset to the first REM period (180 ± 38 vs 116 ± 17 minutes), although this difference was not significant (P = .09). Night seizures significantly increased the time to the first REM period (214 ± 43 vs 116 ± 37 minutes; P = .009). When seizures occurred before the first REM period, the difference was more striking (358 ± 30 vs 114 ± 20 minutes; P < .001) (Figure 2). The time from the beginning of the first REM period to the beginning of the second was not different for any group.

There were no significant differences in total recording duration for NTSZ (416 ± 12 vs 432 ± 12 minutes) or DAYSZ conditions (443 ± 16 vs 439 ± 9 minutes) compared with controls for each group.

SLEEP EFFICIENCY

Day seizures did not significantly decrease sleep efficiency (87% ± 4% for DAYSZ vs 91% ± 2% for control conditions; P = .20). Night seizures significantly decreased sleep efficiency (74% ± 4% for NTSZ vs 91% ± 2% for control conditions; P < .001), and this effect was further enhanced when seizures occurred before the first REM period (65% ± 3% vs 90% ± 2%; P < .001) (Figure 3).

DROWSINESS MEASURES

Three groups were compared with controls using paired t test. Because nocturnal and diurnal seizures disrupt sleep, controls for the purposes of drowsiness measures were defined as recordings with no seizures during the previous 32 hours (the previous day, the previous night, or the morning of the test). These were compared with recordings when a seizure occurred the previous day (DAYSZ condition), the previous night (NTSZ condition), or the morning before the test (SAMESZ condition).

The results of the MOW are shown in Figure 4. Lower numbers represent shorter time to sleep onset and, therefore, increased drowsiness. For DAYSZ condition, MOW was 15 ± 2 for controls vs 17 ± 2 for seizure (n = 6; P = .56). For NTSZ condition, MOW was 16 ± 2 for controls vs 7 ± 3 for seizure (n = 8; P = .01). For SAMESZ condition, MOW was 18 ± 2 for controls vs 12 ± 2 for seizure (n = 7; P = .11). Therefore, nocturnal seizures were the only type that significantly increased drowsiness by this measure, although the number of suitable patients was small.

There were no significant differences in the more subjective SSS and LASD for any of these groups.

Using nonparametric statistics (the Sign Test10), stage 4 sleep was not significantly different between control and NTSZ conditions, and the MOW was not significantly different following nocturnal seizures. All other differences noted above remained significant (P < .05) using this stricter test.

These data suggest that complex partial seizures profoundly disrupt nocturnal sleep, even when they occur during the daytime. Diurnal and nocturnal seizures decreased REM sleep. Nocturnal seizures increased stage

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**Characteristics of Study Recordings**

<table>
<thead>
<tr>
<th>Seizure Condition</th>
<th>DAYSZ</th>
<th>NTSZ</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of recordings</td>
<td>15</td>
<td>13</td>
</tr>
<tr>
<td>No. of male-female subjects</td>
<td>6:9</td>
<td>6:7</td>
</tr>
<tr>
<td>Age range (mean), y</td>
<td>26-56 (41)</td>
<td>26-64 (45)</td>
</tr>
</tbody>
</table>

* DAYSZ indicates a seizure occurred between 7 AM and 11 PM on the day of the recording; NTSZ, a seizure occurred during the nocturnal recording.

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1 sleep and decreased stages 2 and 4 sleep and sleep efficiency.

There are several possible reasons for decreased REM sleep in patients with seizures. The first is simply that sleep is more disrupted, with more frequent awakenings and less time asleep. This is supported by decreased sleep efficiency with nocturnal seizures; however, sleep efficiency was not changed with diurnal seizures. A second explanation for decreased REM sleep is that seizures affect the circadian pattern responsible for REM, thus delaying its onset. This is supported by the increased time to first REM period with all seizures (although this was not statistically significant for diurnal). Third, patients knew that they had had seizures; therefore, psychological factors could have affected sleep. Finally, seizures may have a direct REM suppressant effect without disruption of circadian rhythms.

Previous investigations of the effects of seizures on sleep have given variable results, largely due to differences in methods. Baldy-Moulinier reported decreased REM sleep only on nights with generalized or multiple partial seizures when compared with healthy controls. Besset reported decreases in total sleep time and REM sleep when patients with seizures were compared with patients with no seizures. Studies of single-night recordings performed on patients with epilepsy and compared with healthy controls, with no mention of the proximity of seizures, showed no change or decreases in REM sleep in patients with epilepsy, but did not report whether seizures occurred.

In all of these studies, patients with epilepsy continued to receive anticonvulsants, which can alter sleep. This is particularly significant when comparisons were made with healthy controls who took no anticonvulsants. All but 1 study did not control for the first-night effect, which could affect patients with epilepsy differently than controls. Finally, all of these studies failed to control for the presence of daytime seizures, which (in our investigation) also affect sleep.

Our study controlled for any effects of the underlying disease process by using patients as their own controls. This method also controls for anticonvulsant effects to some extent. Although anticonvulsant therapy typically was tapered during admissions, in general, patients took less drug when seizures occurred than on control nights. Although this was not specifically recorded, the number of recordings with no anticonvulsant at therapeutic level in our study was similar for the 3 study conditions. Therefore, any sleep disruption due to anticonvulsant treatment would be expected to affect control nights at least as much as seizure nights. Our study specifically addresses seizures with temporal lobe onset; other seizure types (such as frontal onset or primary generalized epilepsy) could affect sleep differently.

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29% (5/17) for NTSZ conditions. Precise lights-off times were not recorded, so the important measure of sleep latency could not be calculated and sleep efficiency calculations were modified. As respiratory measures were not included, it is possible that undiagnosed sleep apnea was present in some patients, which could induce seizures and exacerbate baseline sleep disruption. As our study looked only for differences with seizure occurrence in the same patients, this confounding factor would only be important if seizures induced or exacerbated sleep apnea. This possibility could be investigated with formal sleep studies.

As the precise function of REM sleep is unclear, the importance of the observed selective REM deprivation is unknown. Studies in animals show that prolonged REM sleep deprivation causes death, supporting an essential (although as yet undiscovered) function. REM sleep has also been implicated in learning and memory consolidation. Therefore, REM sleep deprivation could be responsible for the dullness and decreased productivity that many patients experience following seizures. Deprivation of REM sleep is not, however, known to cause drowsiness; decreased sleep efficiency observed in patients with nocturnal seizures is more likely responsible for this. It is also possible that changes in REM sleep are part of an overall disruption in circadian rhythms, which include temperature and melatonin secretion. Disruption of these patterns clearly causes decreased functioning, as demonstrated in the disorders of jet lag and shift work. The temperature curve is difficult to displace, and this is unlikely to occur with isolated seizures. However, evidence suggests that melatonin secretion is disrupted by seizures.

Overall, our study shows that temporal lobe complex partial seizures disrupt sleep (especially REM), particularly when occurring early in a night’s sleep but even when they happen many hours before sleep onset. Further studies will be needed to determine whether similarly altered sleep patterns are seen in patients with other types of epileptic seizures.

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REFERENCES


Figure 4. Abbreviated maintenance of wakefulness test (MOW) following seizures. Top, MOW is shown for CONT vs DAYSZ conditions. No difference was seen. Bottom, MOW for CONT vs NTSZ conditions or when a seizure was seen on the morning before the test (SAMESZ condition). The MOW was significantly less (and thus the patients more drowsy) for the NTSZ condition only. Other abbreviations are given in the legend to Figure 1. Asterisk indicates a significant difference (P < .05) compared with CONT.

MOW (although this was not significant by the Sign Test). This finding is clearly not simply a postictal effect, as patients who had seizures during the day just before the test (SAMESZ condition) were not significantly drowsier than under control conditions. Multiple measures, as described in the standard MOW, would be more accurate; however, this was not deemed practical in patients with frequent seizures in an epilepsy monitoring unit. Although suggestive, our results must be interpreted with caution and need to be confirmed by standard testing.

There are limitations to our study. The environment was not a sleep laboratory, so conditions were not optimal for sleep. Although this limits the significance of baseline data, the differences between seizure and seizure-free studies should be valid. There were no baseline sleep diaries, so it is possible that patients were relatively sleep deprived on admission. If this were true, most patients had control recordings first, since REM rebound may have contributed to relative increases in REM on control nights. It is therefore possible that patients with temporal lobe epilepsy chronically cycle between REM deprivation and rebound. Anticonvulsants also affect sleep. Therapy was typically tapered during admissions, and specific doses were not recorded. However, the percentage of recordings with no anticonvulsant at therapeutic level in this study was 26% (13/50) for control, 30% (6/20) for DAYSZ, and 29% (5/17) for NTSZ conditions. Precise lights-off


Typographical Error. In the December Archives a typographical error appeared in the first paragraph of the editorial “Proton Magnetic Resonance Spectroscopy: An In Vivo Window to Study Neurodegenerative Disorders” by Linfante and Ashizawa (Arch Neurol. 1999;56:1446-1447). The sentence reading “The pulse sequence produces a signal decay in which the spins are percussing at a frequency determined by the local magnetic field and ultimately by their chemical relationship with different compounds” should have read “The pulse sequence produces a signal decay in which the spins are precessing at a frequency determined by the local magnetic field and ultimately by their chemical relationship with different compounds.” (Bold typeface added.) We regret the error.