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# Selective Deprivation of Sleep in Pycnoleptic Children

## Effects of Deprivation of Slow-Wave Sleep and REM Sleep on the Frequency and Duration of Petit Mal Attacks

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**Summary.** 1. *Selective deprivation of slow-wave and paradoxical sleep* was performed in 10 children with pycnoleptic attacks (8 of them before anticonvulsive treatment, 2 of them while under medication). The *frequency and duration of petit mal attacks* were intraindividually compared during night sleep and after waking for a 5-h period.

2. After *deprivation of slow-wave sleep* with reduction of EEG stages 3 and 4 to about one-third of the baseline but *normal duration of sleep,* petit mal attacks are more frequent and long-lasting than after normal sleep or selective deprivation of REM sleep.

3. Although total sleep time is significantly diminished after *selective deprivation of paradoxical sleep* the frequency of attacks during the waking state was lower than after normal sleep and deprivation of slow wave sleep. This observation shows a clear influence of the *quality of sleep* on the frequency of epileptic attacks.

4. During sleep petit mal seizures were mainly found during *stages 2* and *paradoxical sleep.* Single spike and irrregular spike wave discharges, however, occurred more frequently during slow-wave sleep. Their frequency was not significantly different in the deprivation conditions.

5. In contrast to experimental data in animals, REM deprivation is less provoking to epileptic attacks outside sleep than deprivation of stages 3 and 4 sleep. Therefore a sufficient amount of slow-wave sleep should be preserved for pycnoleptic children.

**Key words:** Selective Sleep Deprivation – Slow-Wave Sleep – REM Sleep – Pycnoleptic Attacks – Children.

**Zusammenfassung.** 1. *Selektiver Entzug von Tiefschlaf* (Stadien 3 und 4) *und REM-Schlaf* (paradoxer Traumschlaf) wurde bei *10 Kindern mit Pyknolepsie* durchgeführt (8 vor Medikament-Einstellung, 2 während Antiepileptikatherapie). Die *Frequenz und Dauer spontaner Absencen* wurden intraindividuell

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während des Nachtschlafes und nach dem Aufwachen für die Dauer von 5 Stunden bestimmt und mit normalem Schlaf verglichen.

2. Bei *Entzug von Tiefschlaf* (Verminderung der EEG-Stadien 3 und 4 auf etwa ein Drittel der Basisnacht) aber *normaler Gesamtschlafdauer* sind in der *folgenden Wachperiode und auch während des Schlafes selbst Petits maux häufiger* und länger andauernd als bei normalem Schlaf oder nach selektivem REM-Entzug.

3. Trotz signifikant verminderter Gesamtschlafzeit nach *selektivem REM-Entzug* war die Anfallsfrequenz im Wachzustand geringer als nach normalem Schlaf und Entzug der Stadien 3 und 4. Auch dieser Befund zeigt, daß die *Schlafqualität* einen Einfluß auf die Häufigkeit epileptischer Anfälle hat.

4. *Kleine Anfälle* fanden sich während des Schlafes vorwiegend in den *Stadien 2* und im *REM-Schlaf.* Dagegen wurden *einzelne* spikes und irreguläre spike wave-Abläufe meist während der *tiefen* Schlafstadien 3 und 4 beobachtet, aber ihre Häufigkeit war in den einzelnen Versuchsbedingungen nicht signifikant unterschieden.

5. Im Gegensatz zu bisherigen Befunden zeigte sich, daß *REM-Entzug weniger anfallsprovozierend wirkt als Entzug von Tiefschlaf.* Bei Kindern mit Pyknolepsie sollte deshalb auf ausreichenden Tiefschlaf geachtet werden.

**Schlüsselwörter:** Selektiver Schlafentzug – Tiefschlaf – REM-Schlaf – Pyknoleptische Anfälle – Kinder.

## Introduction

Sleep deprivation is used as a method of provoking epileptic discharges in the EEG as well as true clinical seizures [3, 8, 9, 12]. It is still a matter of discussion whether this effect is due to a general lack of sleep and to subsequent drowsiness or to a deficit of REM sleep. Cohen et al. [5] found a decreased threshold and prolonged duration of electrically induced seizures after selective deprivation of paradoxical sleep. From findings in human epileptics Bergonzi [4] claimed that REM deprivation causes increased brain excitability in contrast to selective deprivation of slow-wave sleep. Therefore the theory of a "seizure preventing function" of paradoxical sleep was discussed.

We have approached this problem again in humans since it has clinical importance, particularly if effects of antiepileptic drugs on sleep are considered. In patients an investigation of seizures induced by pharmacologic or electroconvulsive treatment after selective deprivation of sleep is impossible for ethical reasons. Therefore we decided to investigate a group of patients with frequently occurring epileptic seizures, i.e., pycnoleptic children, and to compare the number and duration of petit mal attacks after selective deprivation of slow-wave and REM sleep.

## Patients and Methods

We investigated 10 children with petit mal epilepsy. Their mean age was $8 \pm 3$ years. They all presented typical petit mal attacks with 3/s spike-wave discharges in the EEG. Two of them were treated with antiepileptic drugs such as DPA and Mysoline. All interictal EEG recordings

were normal except for a slight generalized slowing of basic rhythm in two cases who suffered from grand mal, as well. Neurological signs of brain damage could not be found. The night preceding the experimental night provided baseline data. We started to record at 2100 h and the children were awoken at 0700 h. Recording of EEG with a paper speed of 1.5 cm/s was continued until 12:00 h, which means that 5 h of recordings during wakefulness were obtained. The children remained in bed and were kept awake. The deprivation nights were administered in randomized order and there was an interval of at least two nights between them. The deprivation of slow-wave sleep or paradoxical sleep was performed by means of painful electrical stimuli applied to the forehead. Sometimes, supplementary acoustical stimulation was necessary. The scoring of EEG night sleep records was based on the international fixed criteria. All EEG regular 3/s spike wave discharges lasting at least for 3 s were considered as petit mal attacks in this study. Spikes and irregular spike-wave complexes with a duration less than 3 s were counted only during night sleep. During wakefulness they could not always be distinguished from movement artifacts and were not considered. The frequency and duration of petit mal attacks were intraindividually compared for the baseline and deprivation nights for the wakefulness periods (0700 h until 1200 h) as well as for the sleep periods. Statistical significance evaluated by means of Student's t-test for paired observations was used.

## Results

*Characteristics of Sleep in the Different Conditions* (See Fig. 1)

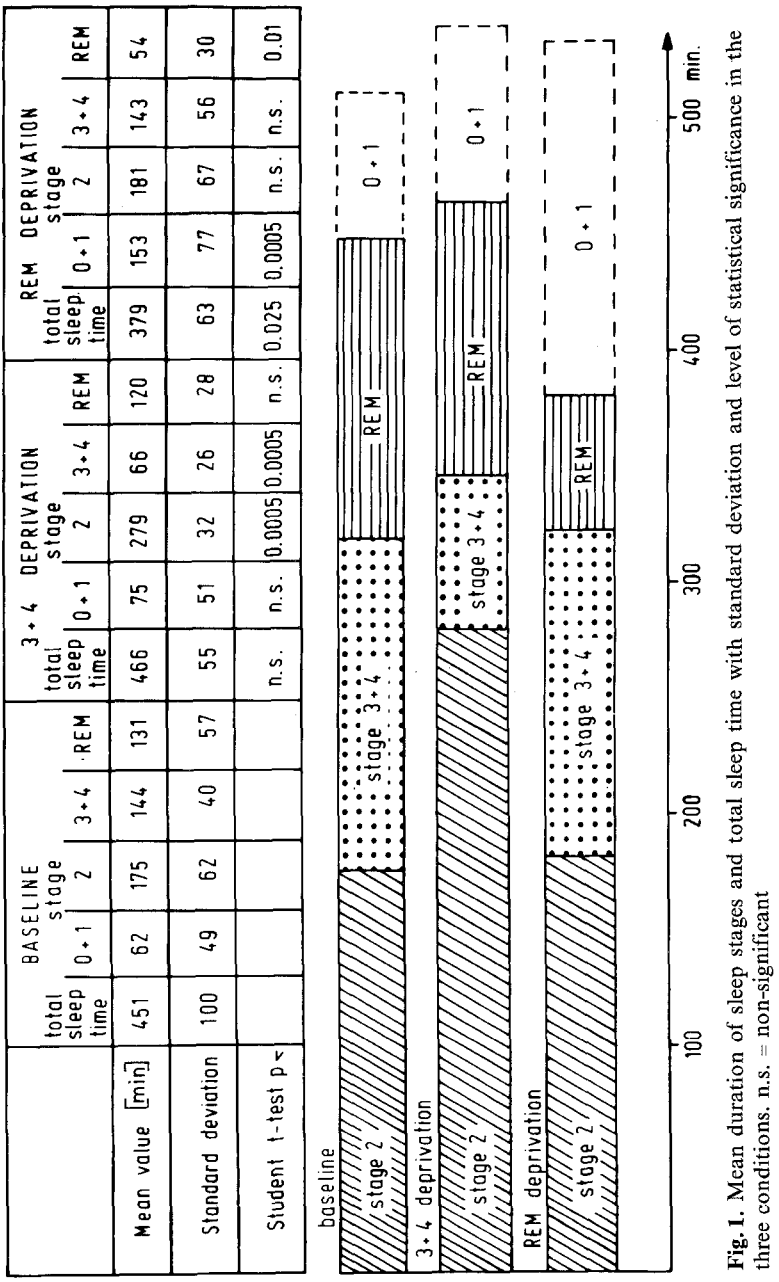
*1. Total Sleep Time (Stages 0 and 1 Excluded).* Total duration of sleep was not significantly different when baseline night and nights with deprivation of sleep stages 3 and 4 were compared. In the REM deprivation condition total sleep time was 379 ± 63 min. A comparison to baseline nights with 452 ± 100 min showed a statistically significant difference ($t = 2.54$, $P<0.025$).

*2. Stage 0 and 1.* Stages 0 and 1 were remarkably increased in nights with selective deprivation of paradoxical sleep with 153 ± 77 min in comparison to 62 ± 49 min in baseline nights ($t = 4.87$, $P<0.0005$). There is no difference if baseline nights and nights with deprivation of slow wave sleep are compared.

*3. Stage 2 Sleep.* Stage 2 sleep is increased in nights with deprivation of slow-wave sleep. The amount of 279 ± 32 min in deprivation nights is significantly higher than in the baseline condition ($t = 5.65$, $P<0.0005$). There is no difference when baseline nights and nights with selective deprivation of paradoxical sleep are compared.

*4. Slow Wave Sleep (Stages 3 and 4).* The total amount of slow-wave sleep was 66 ± 26 min in deprivation nights compared to 144 ± 40 min in baseline nights ($t = 5.81$, $P<0.0005$). The total duration of sleep stages 3 and 4 was not diminished by REM deprivation procedures.

*5. Paradoxical Sleep.* Duration of REM sleep was 54 ± 30 min in nights with deprivation of paradoxical sleep and 131 ± 57 min in the baseline conditions ($t = 2.99$, $P<0.01$). There was no significant difference when the amount of REM sleep in nights with deprivation of sleep stages 3 and 4 and in baseline nights was compared.



| | BASELINE total sleep time | BASELINE stage 0+1 | BASELINE stage 2 | BASELINE stage 3+4 | BASELINE REM | 3+4 DEPRIVATION total sleep time | 3+4 DEPRIVATION stage 0+1 | 3+4 DEPRIVATION stage 2 | 3+4 DEPRIVATION stage 3+4 | 3+4 DEPRIVATION REM | REM DEPRIVATION total sleep time | REM DEPRIVATION stage 0+1 | REM DEPRIVATION stage 2 | REM DEPRIVATION stage 3+4 | REM DEPRIVATION REM |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mean value [min] | 451 | 62 | 175 | 144 | 131 | 466 | 75 | 279 | 66 | 120 | 379 | 153 | 181 | 143 | 54 |
| Standard deviation | 100 | 49 | 62 | 40 | 57 | 55 | 51 | 32 | 26 | 28 | 63 | 77 | 67 | 56 | 30 |
| Student t-test p < | | | | | | n.s. | n.s. | 0.0005 | 0.0005 | n.s. | 0.025 | 0.0005 | n.s. | n.s. | 0.01 |



**Fig. 1.** Mean duration of sleep stages and total sleep time with standard deviation and level of statistical significance in the three conditions. n.s. = non-significant

*Epileptic EEG Activity during Night Sleep* (Figs. 2 and 3)

*1. Petit Mal Seizures during Different Sleep Stages.* In baseline nights petit mal seizures were almost equally frequent during stages 0 and 1 and REM sleep. They occurred more rarely during stage 2 sleep and were very rare during slow wave

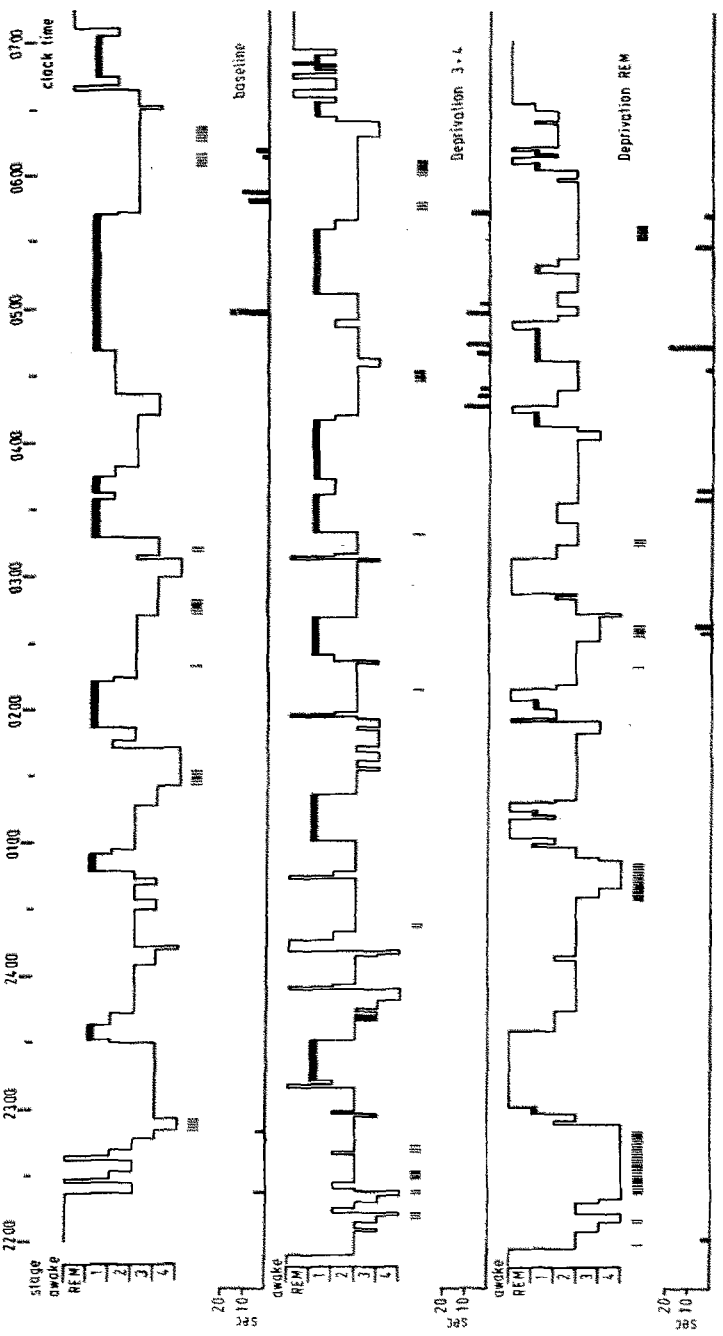



**Fig. 2.** Loomis schema of night sleep of a 9-year-old child suffering from pycnoleptic attacks. Baseline sleep is seen in the upper part, selective deprivation of slow-wave sleep in the middle row and deprivation of paradoxical sleep in the lowest row. Single spikes and irregular spike-wave discharges are symbolized by vertical bars; petit mal attacks are represented by thick bars with their individual duration

sleep. A similar distribution was found during the deprivation nights, the seizure frequency being higher during stages 0 and 1. These stages were more frequent than in baseline conditions, particularly in nights with selective deprivation of paradoxical sleep.

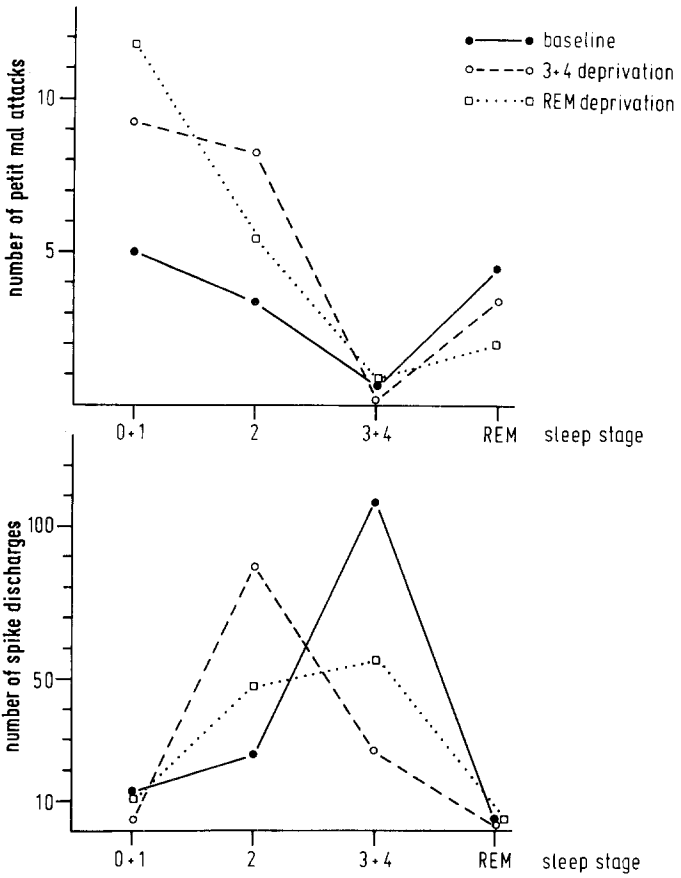



**Fig. 3.** Relation of petit mal attacks and single-spike discharges in the different sleep stages. There is an inverse relationship between frequency of petit mal attacks and single-spike discharges if slow-wave and REM sleep are considered. A very low frequency of seizures is found during slow-wave sleep, while the amount of single-spike discharges is highest during slow-wave sleep in the baseline condition

In nights with selective deprivation of paradoxical sleep or slow-wave sleep the frequency of petit mal attacks during the night was significantly higher in both conditions than in normal night sleep. During normal night sleep a mean number of 13.6 ± 11.2 petit mal attacks with a total duration of 77.6 ± 50.7 seconds was found. After selective deprivation of slow-wave sleep a mean frequency of 21.0 ± 24.8 attacks was counted. This difference in attack frequency was not statistically significant ($t$ = 0.87).

In the REM deprivation condition an average of 20.0 ± 15.5 attacks with a duration of 158.0 ± 179.2 seconds was observed. This difference in attack frequency was also not statistically significant ($t$ = 1.28).

*2. Spike and Irregular Spike Wave Discharges.* Single-spike discharges and groups of irregular spike waves shorter than 3 s were found to be most frequent during slow-wave stages 3 and 4 and least frequent during REM sleep in the

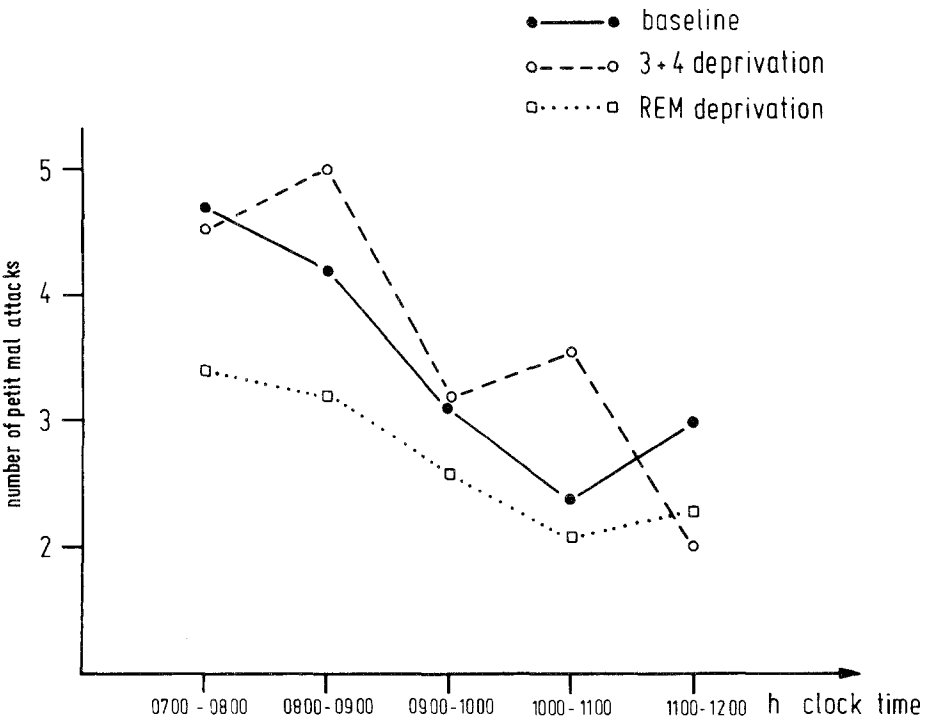



**Fig. 4.** Mean frequency of petit mal attacks from 07:00 until 12:00 h during wakefulness after selective deprivation of slow-wave and REM sleep compared to normal sleep. Difference between the total number of petit mal attacks after REM and slow-wave sleep deprivation is statistically significant

baseline conditions. This type of epileptic EEG activity was increased during stage 2 sleep in the deprivation nights. During normal night sleep we found a number of 149.3 ± 295.6 spike or irregular spike-wave discharges compared to 118.7 ± 180.6 in sleep with selective deprivation of slow-wave stages ($t = 0.71$) and 118.7 ± 186.3 during nights with deprivation of paradoxical sleep ($t = 0.81$).

*Daytime Petit Mal Attacks after Selective Deprivation of Sleep* (Fig. 4)

In the baseline condition we observed 17.4 ± 22.1 petit mal attacks with a mean duration of 148.3 ± 119.5 s. As can be seen from Figure 4, petit mal attacks were most frequent during the period of time from 07 00 until 08 00 h, which is after the transition from sleep to wakefulness. The same observation was made in the deprivation conditions. After selective deprivation of slow-wave sleep, the mean number of recorded petit mal attacks was 18.5 ± 16.1 with a total average duration of 172.1 ± 144.2 s. The difference between these parameters and the corresponding baseline values is not statistically significant if the whole period of time is considered. However, from 10 00 h until 11 00 h after deprivation of slow-wave sleep the difference of 2.4 ± 4.02 in baseline and 3.6 ± 3.7 in deprivation condition is statistically significant ($t = 2.09$, $P<0.05$). The mean duration of

attacks was found to be 13.9 ± 19.1 s after normal sleep compared to 34 ± 26.9 s after deprivation of slow-wave sleep ($t$ = 2.39, $P<0.025$).

The lowest total frequency of 13.6 ± 12.6 attacks during wakefulness was found after nights with selective deprivation of paradoxical sleep and a mean duration of 137.0 ± 107 s. The difference between these values and the corresponding ones for the baseline condition is not statistically significant. If a statistical comparison is made between the frequency of petit mal attacks after slow-wave sleep and REM deprivation, a significantly higher number of attacks is found after slow-wave sleep deprivation ($P<0.05$).

## Discussion

During a 5-h period of wakefulness in the morning the frequency and duration of spontaneous epileptic petit mal attacks were found to be significantly increased after selective deprivation of slow-wave sleep above the baseline frequency and their number decreased after selective REM deprivation. This is an unexpected result, as it was hypothesized from the findings in animal experiments [5, 6, 7] and human epileptics [4] that only selective deprivation of paradoxical sleep but not reduction of slow-wave sleep would increase epileptic activity. In a previous study of Bergonzi et al. [4] the observation of an increased number of spike discharges within recovery sleep after selective deprivation of paradoxical sleep was interpreted as higher nervous excitability in a variety of epileptics. In this investigation [4], however, only night sleep was considered and we found no report about an influence of selective sleep deprivation on the frequency of epileptic seizures during the day. During deprivation nights we observed that the number of attacks and spike discharges were increased when compared to normal sleep. This was found both during deprivation of slow-wave sleep and of REM sleep, as well.

It must be particularly emphasized that in our investigation total duration of sleep was nearly equal to values of baseline nights when a selective deprivation of sleep stages 3 and 4 was performed. The total sleep time was, however, significantly reduced in the REM deprivation condition. In spite of this abbreviation of night sleep the frequency of petit mal attacks during the following period of wakefulness was diminished compared to the conditions with normal total sleep time. This shows that not only sleep duration but also changes of the quality of sleep can activate epileptic activity. Effects of antiepileptic drugs upon the quality of sleep have not been systematically investigated. They most probably will reduce the amount of paradoxical sleep and not influence slow-wave sleep.

The fact that the difference between seizure frequency after slow-wave sleep deprivation and normal sleep was not more striking could be explained by the difficulty of reducing EEG stages 3 and 4 to very low amounts. This is apparently easier to perform in adults than in children [1, 2].

Finally we wish to point out fluctuations of vigilance which obviously play a role in generating petit mal attacks [11]; they may be particularly favoured by the deprivation of slow-wave sleep. Therefore, any condition reducing slow-wave sleep in pycnoleptic children should be avoided.

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