

Distribution of REM Sleep on a 90 Minute Sleep-Wake Schedule

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Summary: Ten young adult (ages 17-21) volunteers lived on a 90 min sleep-wake schedule for 86 ($n = 5$) or 96 ($n = 5$) consecutive 90 min periods. Subjects were permitted to sleep in 30 min episodes separated by 60 min of enforced wakefulness. These bedrest episodes were monitored by electroencephalogram, electro-oculogram, and electromyogram and were scored in 30 sec epochs using standard criteria. REM sleep episodes on this schedule occurred with a clear daily cycle; 74% of REM sleep time occurred during the rising phase of the body temperature cycle. REM showed a marked tendency to recur during alternate bedrest episodes. Finally, REM and slow wave sleep occurred together during only 27 of 910 bedrest episodes. The findings support a circadian influence on the temporal distribution of REM sleep. Neither the sleep-independent nor the sleep-dependent models of REM sleep were supported by findings on the 90 min schedule. **Key Words:** REM sleep—Body temperature cycle—Circadian rhythm—Sleep-wake schedule.

REM sleep is a unique behavioral state that occurs briefly in humans in discrete episodes during a night's sleep. The REM state is characterized polygraphically by a desynchronized electroencephalogram (EEG), bursts of rapid eye movements, and tonically suppressed chin electromyogram (EMG). REM sleep during the night occurs in a cyclic fashion, alternating with NREM sleep. The average period of this cycle is about 90 min. Although this cyclicity is consistent throughout a night's sleep, the absolute duration of the REM portion of the NREM-REM cycle tends to increase later in the night.

Kleitman (1963) has postulated that the NREM-REM cycle represents an extension into sleep of the basic rest-activity cycle (BRAC) that is evident in a number of waking behaviors. Other investigators have presented evidence that the cyclic recurrence of REM sleep is a sleep-dependent process (Brezinova, 1974; Moses et al., 1978), in which the timing of REM sleep is regulated by the distribution of the intervening NREM sleep and relatively insensitive to waking intervals.

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Others have found circadian factors to be of primary importance in the distribution of REM sleep (Weitzman et al., 1974; Schulz et al., 1975; Czeisler, 1978).

The data described below represent a reanalysis of a study using a 90 min schedule of sleep and wakefulness (Carskadon and Dement, 1975, 1977). This analysis is specifically aimed at describing the temporal distribution of REM sleep as it occurred on this unusual sleep schedule.

SUBJECTS AND METHODS

The study was performed in three runs of one, four, and five subjects. The subjects were 17–21 years of age. Five subjects were recorded on each of two slightly different experimental protocols. Each protocol began with three consecutive days in the laboratory, during which sleep was permitted between midnight and 0800. Five subjects (3 men, 2 women) began the 90 min schedule at midnight following 16 hr of wakefulness after the third night and lived on the schedule for 86 consecutive 90 min periods. The second group of subjects (3 men, 2 women) began the 90 min schedule at 0900 following the third night and continued on the schedule for 96 consecutive 90 min periods. In both protocols, the 90 min schedule was ended at 0800, and subjects were not permitted to sleep until midnight. Recovery sleep was *ad lib* for two consecutive nights.

On the 90 min schedule, bedrest was scheduled for sixteen 30 min portions per 24 hr day. The bedrest episodes were separated by 60 min of enforced wakefulness. All sleep was recorded in individual, sound-attenuated, dark bedrooms. The recordings included monopolar C3 or C4 EEG, monopolar electro-oculogram from left and right outer canthi, and EMG from chin placements. All records were scored in 30 sec epochs according to the criteria of Rechtschaffen and Kales (1968).

As illustrated in Fig. 1, activities were strictly scheduled on the 90 min periods. Standard calibrations preceded each bedrest episode. Oral temperature was taken immediately following bedrest, before subjects got out of bed. Temperature was taken using either a glass thermometer (left in place for 5 min) or an IVAC digital electronic thermometer. Subjects were monitored polygraphically during performance tests given for 20 min on alternate activity episodes. Free time was used for showers, electrode replacement, meals, and card or board games. Subjects were attended continuously to prevent sleep at unscheduled times. Alcohol and caffeinated beverages were unavailable to subjects throughout the study.

RESULTS

One of the most striking factors in the timing of REM sleep on the 90 min schedule was the recurrence of REM sleep on alternating bedrest episodes. Figure 2 illustrates this dominant pattern of recurrence. Of the 240 REM episodes (minus the first one for each subject), only 19 (8.3%) occurred on consecutive bedrest episodes; 128 (55.7%) occurred with one intervening bedrest episode; 15 (6.5%) occurred after two NREM sleep episodes had intervened. REM occurring with three or more intervening NREM sleep episodes accounted for 29.6% (68) of the total. Figure 2 also shows the distribution of slow wave (stages 3 + 4) sleep for

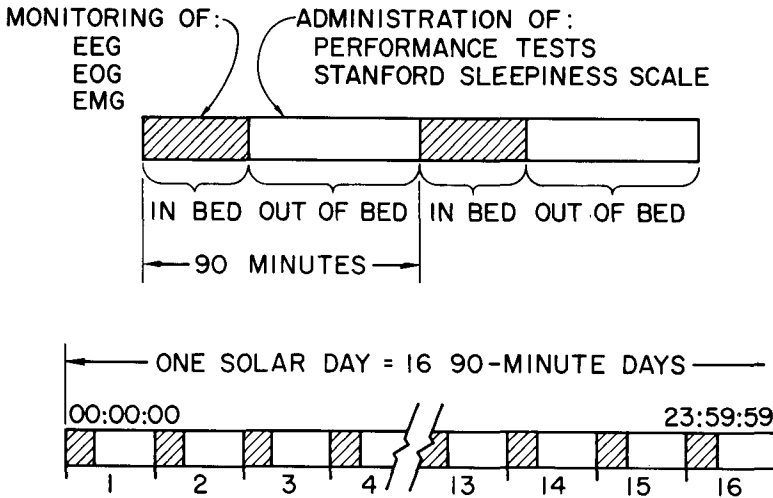


FIG. 1. Schedule of activities on two consecutive 90 min periods and for one solar day. Stanford Sleepiness Scales were given at 30 min intervals. Performance tests were given on alternate activity (out-of-bed) episodes.

comparison. Slightly more than 40% of slow wave sleep recurred on alternate bedrest episodes, and 35% recurred on consecutive episodes. Very few slow wave sleep episodes were separated by three or more bedrest episodes, indicating that slow wave sleep was more widely distributed than REM sleep.

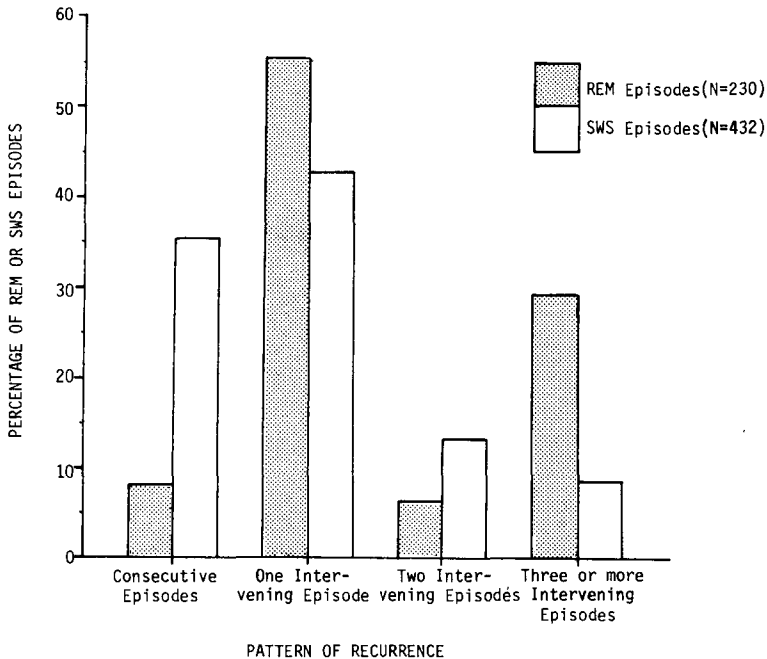


FIG. 2. Pattern of recurrence of REM and slow wave sleep (SWS) during bedrest episodes on a 90 min schedule. Shaded areas represent REM sleep; unshaded areas, SWS.

REM sleeps recurring with three or more intervening NREM sleep episodes were virtually always those that appeared to be the first of the next day's REM sleep quota. To evaluate the possible influence of a daily fluctuation, as suggested by these data, the relationship of REM sleep to body temperature was evaluated as Czeisler (1978) has done for the Weitzman et al. (1974) data from a 180 min sleep-wake schedule. Figure 3 illustrates these findings, showing that REM sleep tended to occur during the rising phase of the temperature cycle. In a further analysis, bedrest episodes were aligned with the lowest daily body temperature of each subject. In this analysis, 74% of REM sleep time occurred on the eight bedrest episodes occurring after the lowest body temperature of each day.

Figure 3 also illustrates the average amounts of slow wave sleep and total sleep time. It is evident that total sleep time also demonstrated a daily pattern, with lowest sleep times occurring at 2230 and the highest sleep time at 0900. The timing of slow wave sleep also reflected a daily fluctuation, although with a lower amplitude. The highest average slow wave sleep time occurred at 0600 and the lowest at 2230. When analyzed by alignment with the individual's lowest daily body temperature, 60% of total sleep and 59% of slow wave sleep occurred in the eight bedrest episodes following the low body temperature.

On the 90 min schedule, REM and slow wave sleep rarely occurred during the same bedrest episode. Of 910 total bedrest episodes, REM sleep occurred on 240 occasions and slow wave sleep occurred on 442 occasions. Both states appeared together on only 27 of the bedrest episodes. Figure 4 illustrates for one subject this mutually exclusive relationship between REM and slow wave sleep across two days on the schedule (90 min periods 33 through 64). This figure also illustrates the characteristic pattern of recurrence for REM sleep.

Moses et al. (1978) have analyzed these sleep data in a somewhat different manner, by determining REM-NREM cycle lengths after excluding all periods of wakefulness. REM-NREM cycle lengths computed by these investigators varied significantly from the base line to the 90 min schedule. The basal REM-NREM cycle period with all waking episodes excluded was 105 min versus 60 min on the 90 min schedule.

DISCUSSION

In confirmation of the Czeisler (1978) report, our analysis showed a clear daily pattern of occurrence of REM sleep, apparently related to the underlying body temperature fluctuation. These findings on short sleep-wake schedules, along with Czeisler's (1978) findings in free-running human subjects, weigh heavily in favor of a circadian influence on the temporal distribution of REM sleep in humans.

The sleep-independent and sleep-dependent theories of the recurrence of REM sleep were neither supported nor rejected by these results. The Moses et al. (1978) analysis of REM-NREM cycles on a 90 min schedule argue against a sleep-dependent model. As these authors state, however, the broken sleep with relatively brief REM episodes suggest that "each REM episode was not completed, and to compensate, the next REM onset appeared earlier than expected" (Moses et al., 1978). On the other hand, the pattern of REM episodes recurring on alter-

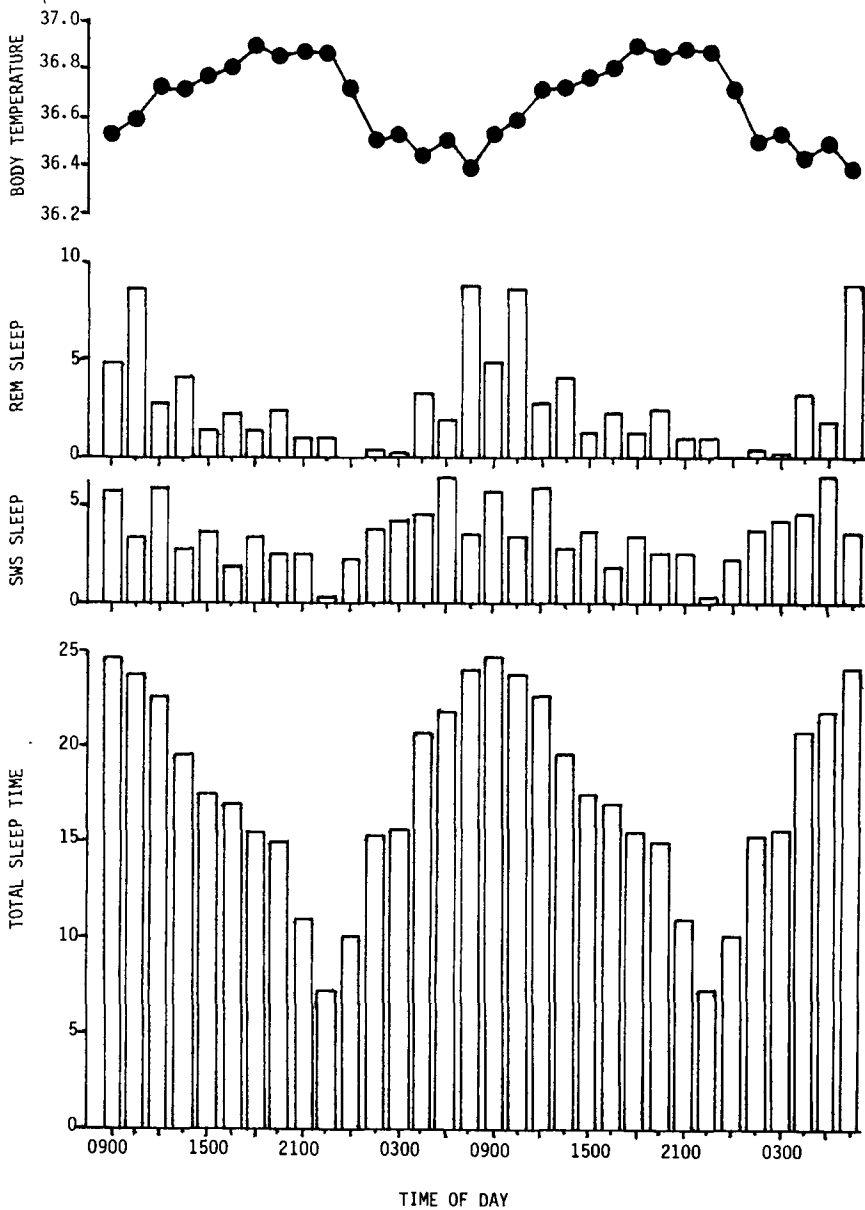
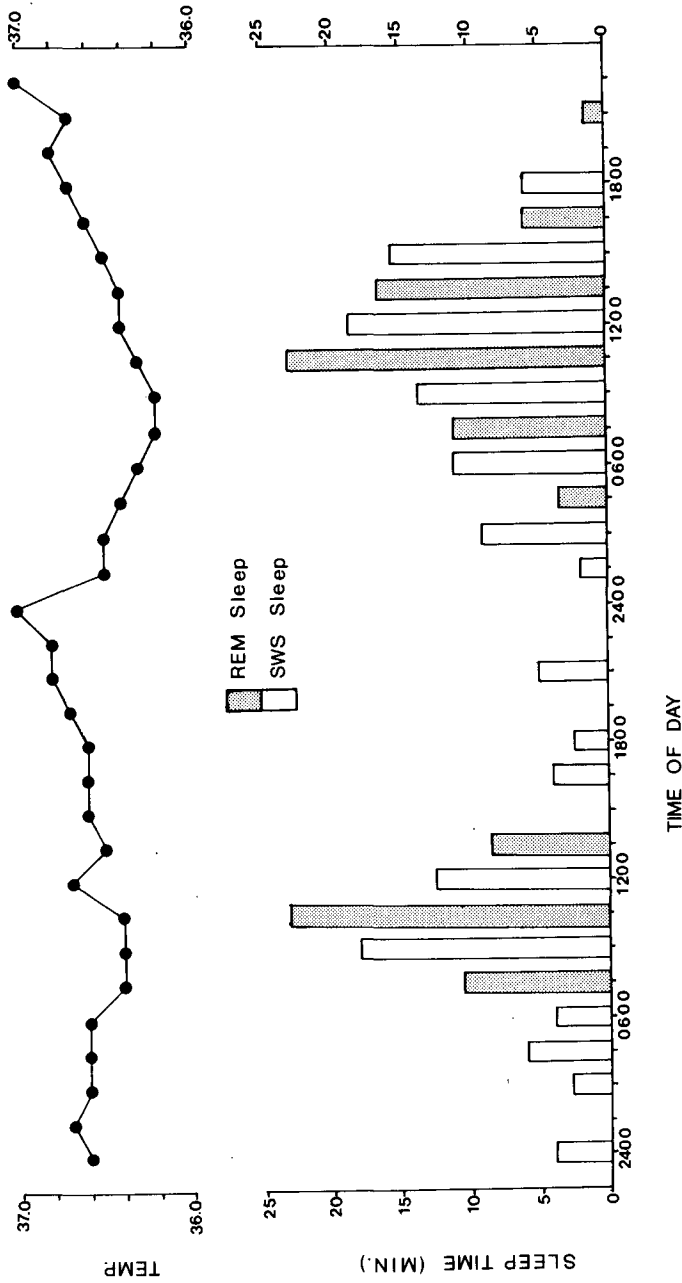


FIG. 3. Average distribution (per time of day) of body temperature, REM sleep, slow wave sleep (SWS), and total sleep time for 10 subjects recorded on the 90 min schedule. The averaged data are duplicated for two days so that the daily fluctuations may be more easily visualized.

nate bedrest episodes was a marked feature and extremely predictable result on the 90 min schedule. In addition, there appeared to be a mutually exclusive relationship between REM and slow wave sleep during the time of peak sleep tendency. This persistent, repetitive pattern of REM during alternate sleep episodes suggests that REM is not simply programmed to occur at 90 min inter-



vals; on the 90 min schedule other factors that may or may not involve a sleep-independent ultradian rhythm (BRAC) appear to influence the recurrence of REM sleep.

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DISCUSSION

Dr. Dement pointed out that REM sleep deprivation has a profound effect on the temporal distribution of REM sleep. He cited a dramatic example of the effect of REM sleep deprivation shown in Fig. 1. He stated that the amount and duration of REM episodes appear to be a lawful function of prior deprivation (Fig. 2).

The effect of increasing sleep pressure on the occurrence of REM sleep or slow wave sleep as a consequence of increasing sleep deprivation was raised by Dr. Borbély. When all sleep across the five experimental days was averaged, the subjects appeared to be slightly less sleepy while on the 90 min day (i.e., increasing average sleep latency). The long recovery sleep, however, contradicted that conclusion. The short, frequent sleep episodes

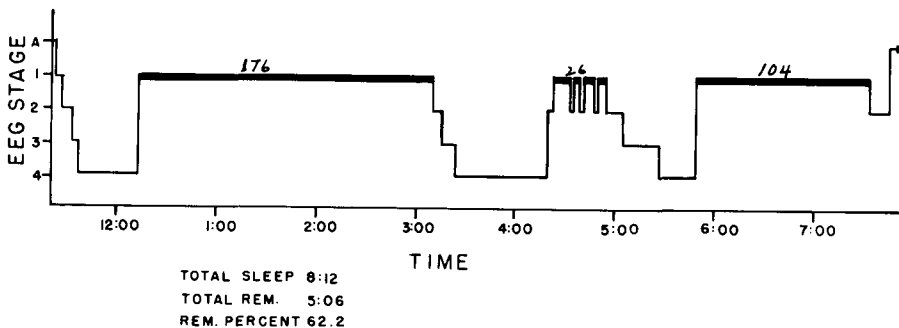


FIG. 1. Continuous plot of sleep stages on the first recovery night in a subject after 15 nights of REM sleep deprivation. The thick horizontal bar represents REM sleep. The small numbers atop the bar are the duration (in minutes) of each REM sleep episode. (Reprinted from Dement, 1965, with permission.)

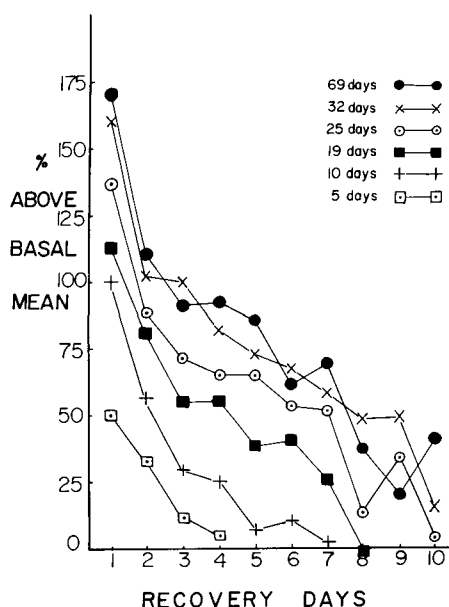


FIG. 2. Recovery curves for varying durations of REM sleep deprivation. Percentage increases were computed by comparing the mean daily base-line REM sleep time (in minutes) with the total minutes of REM sleep on each successive recovery day. Note the essential equality of the 32- and 69-day curves. (Reprinted from Dement et al., 1967, with permission.)

appeared sufficient to offset the sleep loss effect, although the subjects still became very sleepy in the trough of the 24 hour cycle.

Dr. Wirz-Justice raised a neurochemical issue related to the timing of REM sleep. Prior work has shown that cholinergic agonists can phase advance the circadian rhythm of REM sleep in man and in cats. A study of the circadian rhythm of muscarinic cholinergic receptor binding in entrained rats was performed. As seen in Fig. 3, the circadian rhythm of receptor binding is similar to that of REM sleep, with a maximum at the end of the inactive (light) phase and a minimum in the middle of the active (dark) phase. She stated that these data were similar to the results reported by Perry et al. (1977) in a postmortem human brain study.

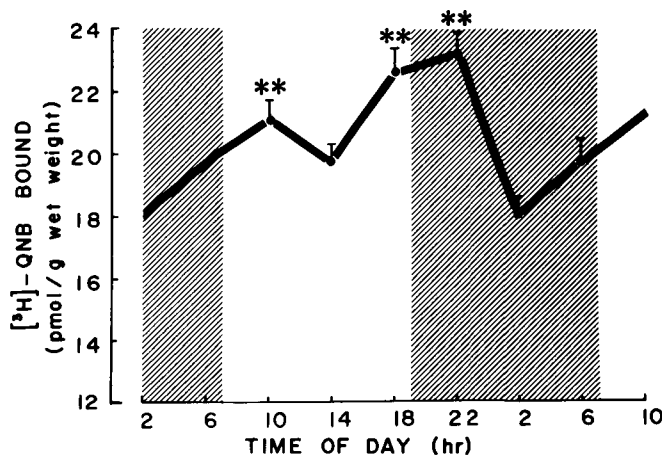


FIG. 3. Circadian rhythm of muscarinic cholinergic receptor binding. ** $p < 0.01$. (Data from A. Wirz-Justice.)

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