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SLEEP STUDIES ON A 90-MINUTE DAY[1]

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In adult humans non-rapid eye movement (NREM) and rapid eye movement (REM) sleep recur with a period of 80–120 min (Williams *et al.* 1974). In normals, a night of sleep invariably begins with 70–90 min of NREM sleep prior to the first episode of REM sleep. Following the first nocturnal cycle, NREM and REM sleep alternate with a somewhat longer period that averages approximately 90 min, although there is considerable variation during the night.

Kelley *et al.* (1973) (see also Dement *et al.* 1972) observed one subject on a 90-min schedule of sleep and wakefulness for nearly 6 days. The original intention of the Kelley *et al.* study was to determine whether the subject could adjust to a schedule that was comparable to the NREM–REM cycle or the putative basic rest–activity cycle (BRAC—see Kleitman 1963, 1969). During the course of the study, Kelley *et al.* (1973) found that REM sleep showed a very unusual pattern of occurrence in their subject. That is, REM sleep occurred in close proximity to the onset of sleep. The aim of the current study was to determine whether this unusual occurrence of REM sleep was replicable.

SUBJECTS AND METHODS

Subjects for this study were 5 healthy, normal undergraduate students (3 men: MA, RL and GR, and 2 women: PH and AR), aged 17–21, who usually slept 7.5–8 h a night. Four of the volunteers had taken a course on sleep and dreams in which the Kelley *et al.* (1973) study was discussed; thus they were not completely naive to the general purposes of the experiment. Specific aims, however, were not discussed with the volunteers, nor were any results revealed during the course of the study.

For one week prior to the start of the experiment, subjects were asked to maintain a regular schedule of sleep and wakefulness, obtaining approximately 8 h of sleep each night and taking no naps. The subjects also agreed to refrain from ingesting alcohol, marijuana, sleeping pills, or alerting compounds during this time.

At the start of the study, each subject was assigned an individual bedroom in the Stanford University Sleep Laboratory. One subject (AR) was recorded 5 months after the others. All sleep recordings were obtained in these darkened, sound-attenuated bedrooms. Polygraphic monitoring equipment was located centrally in a separate room. Sleep periods were monitored with scalp and facial leads recording electroencephalogram (EEG), electro-oculogram (EOG), and electromyogram (EMG) in the standard manner (Rechtschaffen and Kales 1968). During the time spent on the 90-min schedule, the subjects were confined to the vicinity of the laboratory. No attempt was made to isolate the subjects from environmental time cues, although they were undoubtedly reduced by virtue of the confinement.

The experiment began with 2 consecutive all-night baseline sleep recordings following 2 laboratory adaptation nights. The baseline sleep recordings were obtained for 8 h each night from midnight to 08.00. The 90-min schedule began with a 30-min sleep period at midnight following

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the second baseline night. A precise schedule was strictly maintained for 86 consecutive 90-min periods ending at 08.00, 5 1/3 days after it was begun. Subjects were awakened promptly at the end of each 30-min dark period, and temperature and pulse were taken while they remained in bed with polygraphic monitoring. After these readings were obtained (approximately 4 min after lights on), subjects were permitted to get out of bed. At intervals during the waking periods various mood, sleepiness, and performance measures were taken.

Meals were served 4 times per 24-h period on the 90-min schedule, at 05.00, 11.00, 17.00 and 23.00. If the subjects were hungry at other times of the day, they were permitted to have a piece a of fruit or a beverage, except during the last 24 h on the 90-min schedule as explained below. Caffeinated beverages were forbidden during the entire experiment.

On every day of the study, except the last 24 h on the 90-min schedule, 24-h urine samples were collected in all subjects but one (AR). On the last 24 h of the 90-min portion of the study, urine was collected from 4 of the subjects during each waking period, and 120 ml of fluids were ingested during each 90 min. In one subject (GR), continuous blood sampling was conducted throughout the final 24 h on the 90-min schedule. The subject fasted during this time.

As a pilot study, two additional subjects were run at another time. After 5 months on a normal schedule, subject MA repeated the identical protocol with the exception that he slept only 15 min during each of the 90-min periods. (In discussing results, he will be designated as MA_{15} for these data and as MA_{30} for the original data.) That is, he was aroused 15 min early from each scheduled sleep period. The second pilot subject (RP) was a 47-year-old male narcoleptic patient, in whom the narcolepsy–cataplexy syndrome had been diagnosed by complaint (he reported sleep attacks, cataplexy, hypnagogic hallucinations, and sleep paralysis) and by recording a sleep onset REM period during a daytime nap. This patient had been free of medication for more than 6 months at the time of the study. After one baseline all-night sleep recording from 00.00 to 08.00, RP was placed on the 90-min schedule at 09.00 with a ratio of 30 min of sleep to 60 min of wakefulness as in the 5 normal subjects. He remained on this schedule for 48 h, after which he took a 1 h nap before leaving the laboratory to meet a family obligation.

All sleep records were scored according to the Rechtschaffen and Kales manual (1968) in 30-sec epochs. For the most part results were analyzed graphically and by tabulation. Contrast *t* tests and chi square tests were computed where appropriate. This paper will deal chiefly with the sleep stage results; results on mood,

TABLE I

Description of sleep periods on the 90-min schedule.

Subject	Number of periods	Less than 5 min sleep	Over 15 min sleep	Number of REMPs*	Number of SOREMPs**	Over 15 min REM	Number of 3&4***	Over 15 min 3&4
MA_{30}	86	20	54	24	20	6	39	1
RL	86	2	67	24	21	9	43	0
PH	86	6	74	20	15	11	48	6
GR	86	15	56	26	17	6	39	5
AR	86	17	57	16	6	6	56	7
Group total	430	60	308	110	79	38	225	19

* Number of REMPs refers to the number of sleep periods in which REM sleep was recorded.
** Number of SOREMPs refers to the number of occasions on which REM sleep occurred within 10 min of sleep onset.
*** Number of 3&4 refers to the number of sleep periods in which stage 3 and/or 4 NREM sleep occurred.

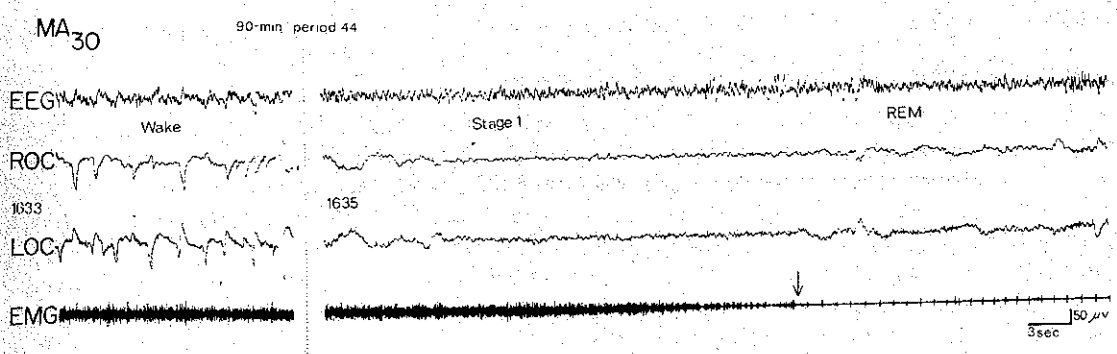

Fig. 1. This figure shows a sleep onset REM period in subject MA_{30} on the 44th sleep period on the 90-min schedule. In the left portion of the figure, the subject was awake 3 min after lights out at 16.33. In the succeeding 2 min (not shown) the subject alternated between wakefulness and stage 1 sleep. At 16.35, the subject was in stage 1. At the arrow (↓), approximately 30 sec later, the EMG was suppressed, signaling the appearance of REM sleep, confirmed by rapid eye movements and EEG activation. EEG was recorded from C_3; eye movements were recorded from the right (ROC) and left (LOC) outer canthi; EMG was recorded from chin placements.

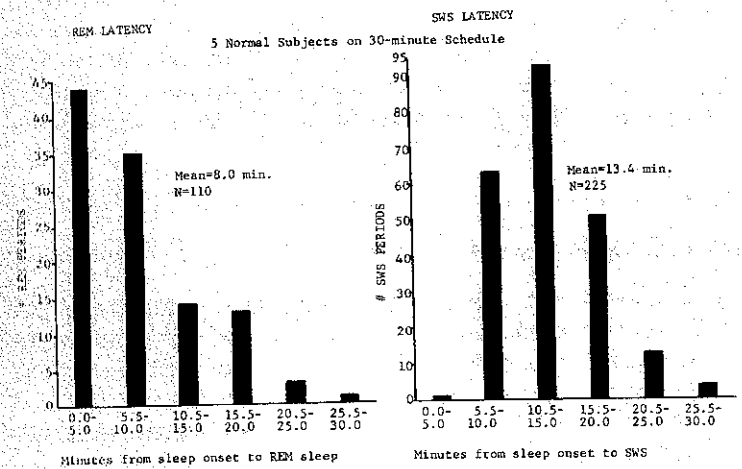

Fig. 2. Distributions of latency from sleep onset to the first epoch scored as REM sleep on the 90-min protocols are given in the bar graph on the left of the figure. Similar distributions are given for latency to the first epoch of stage 3 or 4 in the right of the figure. The two graphs are from the group of 5 normal subjects on the 30-min protocol. The mean SWS latency is higher than mean REM latency ($P < 0.001$).

performance, sleepiness, temperature, pulse, urine, and blood samples will be reported elsewhere.

RESULTS

A. *Normal subjects on 30–60 min sleep–wakefulness schedule*

Table I summarizes salient features of sleep stages on the 90-min schedule of sleep and wakefulness. On this schedule, REM sleep occurred within 10 min of sleep onset on 79 of 110 occasions in the 5 subjects. (Sleep onset was judged by 3 consecutive 30-sec epochs of stage 1 sleep or by stage 2 or REM sleep for 30 sec.) This unusual appearance of REM sleep is designated as sleep onset REM periods (SOREMPs), and an example is given in Fig. 1. The first REM sleep on the 90-min schedule occurred at 06.00 of the first day in GR and AR, and the first SOREMPs appeared in PH and RL at 07.30 on Day 1. The mean REM latency for the 110 sleep periods in which REM appeared during the 90-min schedule was 8.0 min, ranging from 0 to 26.5 min. By contrast, the shortest REM latency on the baseline and

TABLE II

Frequency of recurrence of REM sleep and SWS on 90-min schedule.

Subject	Consecutive sleep periods	1 intervening sleep period	2 intervening sleep periods	3 or more intervening sleep periods
Number of REM sleep periods				
MA_{30}	4	11	1	8
RL	1	11	2	10
PH	0	10	2	8
GR	1	18	1	6
AR	0	8	1	7
Group total	6	58	7	39
Number of slow wave sleep periods				
MA_{30}	12	16	4	7
RL	13	21	4	5
PH	21	18	7	2
GR	8	24	3	4
AR	36	11	8	1
Group total	90	90	26	19

Chi-square test was performed on Group REM and Group SWS $P < 0.001$.

adaptation nights was 45.5 min. Fig. 2 shows the distribution of latencies to REM sleep during the experimental period and contrasts them with the latencies to slow wave sleep (SWS—stages 3 and 4). The REM and SWS latency distributions were obviously different, with a significantly ($P < 0.001$) higher mean SWS latency than REM latency.

Table II demonstrates another peculiarity of the REM sleep periods that occurred on the 90-min schedule. REM sleep very rarely (6 of 110) occurred on consecutive sleep periods. For the most part, REM sleep recurred on alternate 90-min periods; that is, with one NREM sleep period intervening. Most of the REM sleep episodes separated by more than two 90-min periods were the first to appear on each 24-h day. No REM periods were ever recorded on the midnight sleep period. Thus, in general, the first daily REM episode occurred in the sleep period at 01.30 or later, and then REM appeared on alternate sleep periods until 4 or 5 REM episodes had occurred. REM sleep did not appear again until after midnight. Data tabulated for SWS showed a significantly ($P < 0.001$) different pattern of recurrence from REM sleep, with an equal tendency overall for SWS to recur on consecutive or on alternate sleep periods.

While REM sleep occurred primarily between 03.00 and 08.00 on baseline nights, as would be expected on a normal schedule, REM tended to occur chiefly from 07.30 to 14.00 during the 90-min schedule. On recovery nights, REM occurred with a normal cycle during the first 8 h of sleep and continued its cyclic recurrence for as long as the subjects remained sleeping.

Slow wave sleep occurred more frequently than REM sleep. SWS was present in the first sleep period of the 90-min schedule in MA_{30} and AR, and it appeared in the other subjects by the second sleep period. On only 11 occasions of the 225 SWS episodes was SWS present when REM sleep occurred or when total sleep time (TST) was less than 5 min. The latency from sleep onset to the first epoch scored as SWS did not change

from the baseline period to the experimental period. The mean baseline SWS latency was 14.5 min (range = 11.0–20.0 min) and the mean SWS latency for the 225 experimental periods on which SWS occurred was 13.4 min (range = 3.5–29.0 min). SWS seldom gained predominance within a 30-min sleep episode, as is clearly evidenced by the occurrence of only 19 periods in which SWS attained 15 min or more (see Table I). On the baseline days SWS was limited primarily to the first 3 h of sleep; during the experimental period SWS occurred at least once on each of of the 16 daily sleep periods. The concentration of SWS in the first 3 h of sleep on the recovery nights was similar to baseline.

Referring to Table I, it appeared that RL and PH adjusted their sleep–wakefulness pattern most easily, obtaining more than 5 min of sleep on all but 2 and 6 periods and sleeping more than half the allotted time on 67 and 74 of the periods respectively. All subjects obtained over 15 min of sleep on at least 60% of the sleep periods. Of interest was the finding that only 10 of the 60 periods with less than 5 min of sleep occurred between 07.30 and 20.00. The peak of TST tended to occur in the sleep periods between 09.00 and 12.30, and the least sleep occurred most frequently from 21.00 to 02.00.

Table III summarizes the mean daily sleep stage characteristics of the 5 normal subjects. Baseline sleep stage values, averaged over the 2 baseline all-night recordings for each subject, were comparable to normal age group parameters (Williams *et al.* 1974). During the 90-min day portion of the study, the subjects showed a significant overall reduction from baseline of TST per 24 h. This reduction was associated with significantly lower amounts of REM ($P < 0.001$ on Day 1; $P < 0.01$ on Days 2–5) and stage 2 ($P < 0.05$ on Days 1 and 3; $P < 0.01$ on Days 2, 4 and 5) sleep on all experimental days. Stage 4 was also reduced during the experimental period, significantly ($P < 0.01$ on Day 1; $P < 0.05$ on Days 3–5) lower than baseline on

TABLE III

Pooled sleep data per 24 h of 5 subjects on 90-min sleep–wake schedule in minutes.

Sleep stage	Baseline mean	Mean Day 1	Mean Day 2	Mean Day 3	Mean Day 4	Mean Day 5	Mean Day 6*	Mean Recovery Day 1	Mean Recovery Day 1**	Mean Recovery Day 2
Stage 1	28.95	55.60^{+}	51.40	48.20	44.40	39.90	11.80	45.20	7.40^{+}	16.60
S.D.	13.98	12.97	20.21	9.29	10.52	5.66	4.94	32.39	3.96	7.09
Stage 2	244.55	183.70^{+}	146.30^{++}	140.20^{+}	124.20^{++}	135.20^{++}	48.50	484.20^{++}	283.70	193.00
S.D.	19.09	37.91	41.71	41.30	25.58	25.57	16.21	118.48	29.12	107.26
Stage 3	29.15	28.50	28.20	28.30	23.30	29.00	9.20	45.40	39.90	24.20
S.D.	7.26	9.49	7.41	6.95	6.62	4.65	4.22	17.25	14.46	13.22
Stage 4	55.05	29.10^{++}	38.60	35.70^{+}	32.10^{+}	37.30^{+}	16.80	105.70^{++}	93.30^{++}	42.00
S.D.	24.79	20.05	24.61	22.15	18.97	22.39	18.38	41.53	33.35	42.42
REM	108.20	22.90^{+++}	65.80^{++}	51.70^{++}	48.50^{++}	50.10^{++}	11.60	235.40^{++}	96.90	79.00
S.D.	14.01	13.98	14.96	10.20	21.01	12.19	11.20	34.58	12.86	50.09
TST	465.80	316.60^{++}	325.80^{++}	297.20^{++}	273.00^{+++}	291.40^{+++}	93.10	914.80^{++}	475.40	354.80
S.D.	8.18	56.69	53.21	55.66	38.23	21.11	41.21	163.85	2.41	199.78

*Day 6 included only six 30-min sleep periods. **Includes only the first 8 h of dark time. Differences between the Baseline Means and the 5 daily experimental means (except Day 6) and between the Baseline Means and the Recovery 1 means were computed using *t* tests. "$^{+}$" indicates significant difference from the Baseline Mean at $P < 0.05$; "$^{++}$" indicates significant difference from the Baseline Mean at $P < 0.01$; and "$^{+++}$" indicates significant difference from the Baseline Mean at $P < 0.001$.

TABLE IV

Mean sleep stage percentages.

Subject	% Stage 1		% Stage 2		% Stage 3		% Stage 4		%SWS		%REM		%TST	
	BSLN	90-Min	BSLN	90-Min	BSLN	90-Min	BSLN	90-Min	BSLN	90-Min	BSLN	90-Min	BSLN	90-Min
MA_{30}	5.8	13.1	55.9	52.0	7.8	11.5	5.8	5.3	13.6	16.8	20.7	18.1	96.2	57.7
RL	6.4	16.5	50.6	55.9	6.0	7.7	9.0	4.3	15.0	12.0	28.0	15.5	95.3	67.7
PH	3.4	15.7	49.9	46.0	6.8	7.8	14.8	15.2	21.6	23.1	25.1	15.6	97.5	73.1
GR	11.0	19.9	51.3	40.2	6.9	9.3	10.1	11.7	17.0	21.0	20.7	19.3	98.6	55.9
AR	4.5	13.8	50.7	45.1	3.8	9.5	19.4	17.6	23.2	27.1	21.7	10.6	97.0	56.2
Group mean	6.2	15.8**	52.2	47.8	6.3	9.2*	11.8	10.8	18.1	20.0	23.2	15.8*	96.9	62.1**

Differences between the baseline (BSLN) and experimental (90-Min) group means were computed using t tests.

* $P < 0.05$.

** $P < 0.001$.

all but Day 2. Stage 1 was generally higher than baseline ($P < 0.05$ on Day 1) while stage 3 was essentially unchanged.

Mean sleep stage percentages for the baseline and experimental period in each subject are given in Table IV. There was an increase of stage 1 percentage ($P < 0.001$) during the experimental period. Percentage of stage 3 sleep was also significantly higher ($P < 0.05$), whereas REM percentage ($P < 0.05$) and percentage of TST ($P < 0.001$) were significantly reduced.

Sleep time was quite extended on the first recovery night. The lowest (PH) TST on Recovery 1 was nearly 11.5 h, and the highest (GR) was over 18.5 h. The other subjects slept approximately 13.5 h (AR), slightly under 16 h (RL), and slightly over 16 h (MA_{30}). Overall (see Table III), this represented a significant increase in TST ($P < 0.01$) from the baseline mean. The amount of stages 2 and 4 and REM sleep were also significantly higher ($P < 0.01$) than the baseline means. Considering only the first 8 h of Recovery 1, however, the only significant differences from baseline were a decrease of stage 1 ($P < 0.05$) and an increase of stage 4 ($P < 0.01$).

The sleep of 2 of the subjects (MA_{30} and GR) was greatly disturbed on the second recovery night, as evidenced primarily by a long (271 min) wake period after sleep onset in MA_{30} and by a long (373.5 min) latency to sleep onset in GR. On the other hand, the sleep of RL, PH, and AR appeared nearly normal on Recovery 2. Unfortunately, we were unable to record sleep on subsequent nights in 4 of the subjects due to their academic obligations. AR was recorded for 2 additional nights on which dark time was limited to 8 h as on baseline; on these nights there were no striking differences from the baseline recordings. All subjects reported that they returned to a normal diurnal sleep–wakefulness cycle within one week of the end of the 90–min schedule.

Sleepiness and mood, which were measured throughout the study, showed initial discomfort on the 90-min schedule. Daily, 24-h fluctuations were present on both measures. Overall, the sleepiness and mood of the subjects tended to show improvement during the experimental period, approaching baseline levels by the 4th or 5th experimental day. Recovery levels achieved the baseline, or slightly higher, values on both sleepiness and mood.

B. *Pilot subjects MA_{15} and RP*

In general, MA_{15} showed sleep stage changes similar to the subjects on the 30-min schedule. Baseline values were consistent for both experiments. MA_{15}, however, tended to have REM sleep more frequently (29 REM periods in 86 sleep periods) and with a shorter latency from sleep onset (mean = 4.1 min for the 29 REM periods). The REM sleep periods in MA_{15} also tended to occur on alternate sleep periods. The amount of SWS was much lower in MA_{15} than in the normals on the 30-min schedule, while REM sleep was only approximately 10 min less per 24 h. Thus REM % during the experimental period in MA_{15} was virtually unchanged from baseline (baseline mean = 21.7%; experimental mean = 20.2%).

The daily TST for MA_{15} was approximately 90 min less than the mean for the other subjects. MA_{15} slept over half the allotted time on all but 7 of the 86 sleep periods. On many occasions it was extremely difficult to keep him awake when he was lying in bed prior to lights out time. It was also very difficult to arouse him at the end of the sleep periods. On all sleep periods except the 22.30 and 00.00 periods, sleep occupied virtually 100% of the 15-min allotment. MA_{15} slept 16 h 32 min on Recovery 1 and 10 h 40 min on Recovery night 2. Measures of sleepiness and mood showed severe discomfort throughout the experimental period, with diurnal fluctuations.

The narcoleptic patient (RP) showed several striking differences from the normal subjects. REM sleep time and REM percent were much higher in RP than in the normals (mean REM time Day 1 = 89.5 min and Day 2 = 120.0 min; mean experimental REM percent = 30.8%). REM sleep also occurred with greater frequency (17 REM episodes in 32 sleep periods) and usually (11 times) on consecutive sleep periods. SWS appeared on only 8 of the sleep periods. TST, while less than the normals on baseline, was 100 min greater on Day 1 and 150 min greater on Day 2 than the mean values for the same days in the normal subjects.

Finally, it should be noted that, with the exception of SOREMPs, none of the normal subjects

placed on either of the 90-min day protocols evidenced signs of the narcolepsy–cataplexy syndrome. Specifically, no sleep paralysis, cataplexy, or hypnagogic hallucinations were reported by the normals. In addition, the narcoleptic patient reported no exacerbation of his symptoms during the experiment. He reported 4 partial cataplectic attacks, all involving only the face and neck muscles, lasting for several seconds, and in response to laughter. Two cataplectic attacks occurred at approximately 05.30 on consecutive days, and one each at 09.30 and 22.30 of the second day. Vague, non-threatening hypnagogic hallucinations of short duration were reported by RP on 2 occasions, at 08.00 after waking from the baseline recording, and at 20.00 on the first experimental day. RP also reported one brief episode of sleep paralysis when falling asleep at 18.00 on the second day. Two short sleep attacks were observed by the experimenter in the hour following the patient's last sleep recording on the 90-min schedule.

DISCUSSION

It should be re-emphasized that the first REM sleep period normally occurs only after 70–90 min of NREM sleep. Until recently, it was felt that this sequence had some causal attribute, *i.e.*, that NREM sleep served a necessary "priming" function for the occurrence of REM sleep. Furthermore, it was felt that reversal of this pattern—the occurrence of a REM period at the onset of sleep or after a very short amount of NREM sleep—in normal adults could only be elicited by extended periods of selective REM sleep deprivation by physical or pharmacologic manipulations that resulted in a greatly increased "REM pressure". In cats, for example, Dement *et al.* (1967) found sleep onset REM periods only following 22 days of selective REM deprivation. In humans, Dement and Rechtschaffen (1968) summarized the results of prolonged selective REM sleep deprivation in 38 subjects in whom the deprivation period was 5–16 consecutive nights. Only 2 subjects showed sleep onset REM periods, one after 2 nights and one after 5 nights of REM deprivation. The other subjects, who were deprived of REM sleep for 7, 8, and as long as 16 nights, never showed this state reversal. Because such extreme measures seemed necessary to elicit a SOREMP in normal adult volunteers, the pathological significance attributed to the acknowledged occurrence of SOREMPs in narcolepsy–cataplexy syndrome was greatly enhanced (Rechtschaffen *et al.* 1963; Dement *et al.* 1966a). Indeed, it has led to the use of the SOREMP as an important diagnostic criterion (Wilson *et al.* 1973; Guilleminault and Dement 1974; Kessler *et al.* 1974).

In the Kelley *et al.* (1973) study, however, it was shown that REM periods beginning at sleep onset or after only a few minutes of NREM sleep occurred after only 30 h on the 90-min schedule. More recently, SOREMPs have been found by Weitzman *et al.* (1974) in a study of a 180-min sleep–wakefulness schedule, and by L. C. Johnson and his colleagues (personal communication) in a study of napping on a 240-min schedule. (A 1966 report by Webb *et al.* showed SOREMPs in normal subjects in "early morning sleep". It is difficult to interpret these data in light of present findings, however, because details, such as the interval between the end of nocturnal sleep and the early morning sleep period, are not given, and because it is difficult to determine which group had SOREMPs in the study.)

The present study confirmed that SOREMPs can be easily induced in normal subjects. REM sleep consistently appeared immediately following wakefulness or following short periods of stage 1 or stages 1 and 2 sleep. In less than 10% of the REM periods did the usual progression through stages 1, 2, 3 and 4 occur.

The reversal of what had heretofore been an inviolate progression of sleep states in normal individuals raises some doubt about the significance of the diagnostic nap recording of a SOREMP in narcolepsy–cataplexy. At the very least, the apparent ease of producing SOREMPs in normals would indicate that careful attention must be given to the whole day's schedule and the time of the diagnostic nap.

The 90-min day protocol demonstrated several additional peculiarities of REM sleep. In several ways REM sleep appeared to be more sensitive to the schedule change than did the other sleep stages in the normal subjects. REM time per 24 h was significantly reduced throughout the experimental period, although this effect was much

more severe on the first experimental day than later in the study. REM sleep also seemed more sensitive to time of day. As was noted, most REM sleep tended to occur during the morning and early afternoon hours. This time-locked aspect of REM is comparable to findings reported by Webb and Agnew (1967) from a study of naps taken at several times during the day, although in the present study SOREMPs were more likely to occur later during the day than in the Webb and Agnew study. In addition, REM sleep during the day appeared to be rigidly programmed, with a clear tendency to recur on alternate 90-min periods. This pattern of recurrence may reflect a refractory period for REM sleep of longer than 60 min but shorter than 180 min in normal subjects. Finally, it should be noted that the recovery sleep did not appear typical of subjects given REM sleep deprivation, even though REM appeared to be significantly deprived during the experimental period. Usually, selective REM deprivation or even partial REM deprivation leads to a rebound of REM occurring early during nocturnal recovery sleep (Dement 1965; Dement *et al.* 1966b). It is difficult to determine whether the increase of REM sleep seen on Recovery 1 reflected a deprivation-rebound effect or was due to the extended sleep in which a normal NREM–REM cycle was maintained.

The extremely long sleep times on the first recovery night may be explained as the result of an accruing sleep deprivation, although there was a relative lack of complaints usually associated with sleep loss. The latter consideration notwithstanding, recovery sleep for the subjects on the 30-min protocol seemed too extreme to be accounted for solely by the relatively modest amount of sleep deprivation. Considering only TST, the sleep loss was equivalent to approximately 3 nights of total sleep deprivation during 6 experimental days. For comparison, a young man who was totally sleep deprived for 264 h (11 consecutive days) slept only 14 h and 40 min on his first recovery night (Gulevich *et al.* 1966). In addition, the amount of sleep obtained on Recovery 1 in this study did not appear to be directly related across subjects to the amount of deprivation each subject experienced. In the one within subject comparison available, MA_{15} who was sleeping an average of 60 min less per 24 h than on the 30-min protocol, slept only 20 min more on Recovery 1 than he did in the 30-min study. It should be noted, however, that MA_{15} was able to sleep much better on the second recovery night than he did in the earlier study. Alternative explanations for the lengthy recovery sleep times include a possible sleep satiation effect that has been seen to vary among individuals (Aserinsky 1969) or a disruption of the 24-h cycle. Evidence supporting the latter alternative was the apparent shift in peak TST on the 90-min schedule to the hours between 09.00 and 12.30. This shift may have carried over to the recovery period, influencing the subjects' prolonged sleep during these hours on Recovery 1.

The differences between baseline and experimental amounts of stages 1, 2, 3, and 4 sleep may be interpreted on the basis of the usual progression of sleep stages. In this study, sleep stages progressed in the normal manner, except for most occasions when REM sleep was present. Hence, given the 16-fold increase in opportunities for sleep onset daily, it is readily apparent that stage 1 would increase precipitately. Stage 2 sleep, which normally recurs with great regularity throughout normal nocturnal sleep, was limited by the short sleep periods. Because the latency to SWS did not change from the baseline to the experimental period, SWS usually occurred late during the sleep period and was frequently interrupted by the end of the period, often before stage 4 sleep occurred. Hence the amount of SWS was lower than baseline, while the percentage of stage 3 sleep was higher. On the first recovery night SWS was significantly higher than on baseline, which would indicate that the significant reduction of stage 4 per 24 h represented a substantial deprivation.

A final word should be said about the 90-min day protocol itself. In addition to its possible usefulness in studying the narcolepsy–cataplexy syndrome, it also has potential experimental utility in studies of the function of sleep because of its different effects on the sleep states. We have demonstrated that REM sleep occurring prior to SWS can be reliably elicited in normal subjects with a technique requiring only a minimum of 8 h of monitoring. Furthermore,

SWS occurred in the same sleep period with REM sleep on less than 10% of the periods of REM sleep. Thus, the dichotomy between REM and NREM sleep seemed to be sharpened. Finally, because REM sleep tended to occur on alternate sleep periods on this schedule, it became quite predictable. These three factors would seem to make the use of the 90-min day paradigm very attractive in studies of the differential functions of REM and SWS.

SUMMARY

After 2 adaptation and 2 baseline all-night sleep recordings, 5 normal young adult subjects (3 males) were placed on a schedule alternating 60 min of wakefulness and 30 min of sleep for 5 1/3 24-h periods. A 2-day recovery period followed. One male subject (MA_{15}) was later placed on the identical protocol with the exception that he was allotted periods of 75 min of wakefulness and 15 min of sleep during the experimental period. One male narcolepsy–cataplexy patient was placed on the 60–30 schedule for 48 h.

All subjects showed REM sleep during the schedule manipulation. REM sleep occurred within 10 min of sleep onset (SOREMP) on 79 of 110 REM sleep occasions in the 5 normals, on all 29 REM episodes in MA_{15}, and on 16 of 17 REM periods in the narcoleptic. In the normals, REM sleep showed a tendency to recur on alternate 90-min cycles, while in the narcoleptic REM recurred on consecutive periods. Compared to baseline, REM sleep 24 h was decreased in the normals and increased in the narcoleptic.

Time spent in slow wave sleep and stage 2 was also reduced in the normal subjects on the 90-min schedule, and stage 1 sleep time was increased. Peak sleep times for the 5 normals occurred between 09.00 and 12.30 and lowest sleep times from 21.00 to 02.00. During the first recovery night, sleep times ranged from 11.5 to 18.5 h, including significant increases of slow wave sleep and REM sleep. Except for SOREMPs, no signs of the narcolepsy-cataplexy syndrome were seen in any of the normal subjects.

RESUME

ETUDE DU SOMMEIL AU COURS D'UN JOUR DE 90 MINUTES

Cinq jeunes adultes normaux (3 hommes et 2 femmes) furent tout d'abord enregistrés pendant 4 nuits successives. Les deux premières furent traîtées comme nuits d'adaptation aux conditions d'enregistrement, les deux dernières servirent de nuits de référence. Puis les sujets furent soumis à un horaire artificiel. Pendant 5 et 1/3 journées successives, ils furent placés sous un protocole alternant continuellement 60 min d'éveil et 30 min de sommeil. Enfir leur sommeil fut enregistré pendant la période de récupération de 2 jours à la fin de l'expérience. Un jeune homme (MA_{15}) fut ultérieurement soumis à un protocole pratiquement identique. Seul l'horaire artificiel fut modifié; il fut soumis à une alternance de 75 min d'éveil et 15 min de sommeil. Un malade présentant un Syndrome Narcolepsie–Cataplexie fut aussi placé sous l'horaire artificiel 60 min d'éveil–30 min de sommeil pendant 48 h.

Tous les sujets présentèrent des périodes de PMO (phase de mouvement occulaire rapide) au cours de la période avec horaire artificiel. Le sommeil en PMO apparut au cours des 10 premières minutes de sommeil, 79 fois sur 110 chez les 5 normaux; toutes les PMO (29) apparurent au cours des 10 premières minutes de sommeil chez le sujet MA_{15}, et 16 des 17 PMO enregistrées chez le Narcoleptique furent aussi notées au cours des premières 10 min de sommeil. Chez les normaux, le sommeil en PMO a tendance à alterner tous les 2 "cycles de 90 min", alors que chez le Narcoleptique, le sommeil en PMO est réenregistré lors de chaque cycle. Par rapport aux nuits de référence, la durée totale du sommeil en PMO par 24 h diminua chez les normaux et augmenta chez le Narcoleptique.

La durée totale du sommeil lent (stades 3 et 4 d'une part et stade 2 d'autre part) diminua aussi chez les sujets normaux placés sous un horaire de 90 min. Chez les 5 normaux, c'est entre 9 h et 12.30 h que le temps de sommeil fut le plus long, tandis qu'il fut minimum entre 21 h et 2 h.

Au cours des premières 24 h de "récupération", les sujets dormirent entre 11.5 h et 18.5 h. Une augmentation significative de stade 3–4 et de sommeil en PMO fut enregistrée. En dehors des périodes d'endormissement en phase de PMO (PMO apparaissant moins de 10 min après l'endormissement), aucun symptôme du Syndrome Narcolepsie–Cataplexie ne fut observé chez les sujets normaux soumis à l'expérimentation.

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