Deprivation of Dreaming Sleep by Two Methods

I. Compensatory REM Time

HAROLD SAMPSON, PhD, SAN FRANCISCO

THE rapid-eye-movement (REM) period of sleep has been identified as a distinctive and regularly recurring psychophysiological state within the nightly sleep cycle.[2-4,8,20-22] REM periods occur during sleep at an interval of about 90 minutes from the onset of one period to the onset of the next in the human adult, and constitute about 20% of total sleep time. The psychologic phenomenon of dreaming occurs during REM periods,[2,3,8-10] and this finding allows investigation of the function of dreaming sleep by reducing REM time.

Dement drastically reduced the typical amount of nightly dreaming of experimental subjects by arousing them just after the beginning of each REM period.[6] Subjects showed a progressive increase in dream attempts during a series of deprivation nights and, on recovery nights, a marked elevation in dream time that grossly compensated for the prior REM deficit. Several of Dement's subjects reported an increase in appetite and most seemed tense and irritable. Comparable awakenings *outside* of REM periods over a series of control nights did not produce any of the effects observed during the deprivation series. Thus, the effects could not be attributed simply to the multiple awakenings.

Dement proposed that there is a need for a certain amount of nightly dreaming. Fisher and Dement [11] elaborated the possible relationship of these findings to Freud's conception of the safety-valve function of dreaming: dreaming may provide a necessary discharge for instinctual tensions which cannot be gratified in reality. Dement's study reported no control, however, for multiple interruptions *of the REM process itself*. Two interventions of potentially distinctive import—a reduction of REM time and an interruption of the ongoing REM (and dream) process—remained confounded.

To test the hypothesis that REM interruption rather than deprivation was responsible for the observed effects, a study was undertaken to compare the results of two methods of reducing dream time. The first method was the dream interruption technique used by Dement in his original study; the second method involved successive nights of *partial sleep* deprivation. Within the first 2½ hours of sleep, there is typically only about 10-15 minutes of the night's total 90-100 minutes of REM time. A subject allowed only 2½ hours of sleep would have a reduction in REM time comparable to that achieved by dream interruption techniques. The reduction could occur without any interruptions of ongoing REM periods.

The experimental questions were, first, whether these alternative methods of reducing REM time would produce comparable REM elevations on recovery nights; and second, whether the two methods would produce comparable psychological effects. An affirmative answer to these questions would exclude the REM interruption hypothesis and lend further support to the REM deprivation hypothesis. The present report will be confined to the first question; the psychological data require a distinctive analysis and will be presented separately.

Before the experiment got underway, Berger and Oswald [5] reported the results of a study with similar aims and methods. They used six subjects in a series composed of two or three adaptation nights of sleep, four baseline nights of sleep, four nights of total sleep deprivation, and four recovery nights of sleep. They found a significant *decrease* in the mean percentage of total sleep time spent in dreaming on the first recovery night compared to baseline nights; but on the second recovery night, dream time was significantly *increased* over baseline levels. The

Submitted for publication Feb 5, 1965.
From the Mt. Zion Hospital.
Reprint requests to 1600 Divisadero St, San Francisco, Calif 94115.

results demonstrated a compensatory effect in the absence of any dream interruption, as well as an apparent priority of nonREM sleep over REM compensation.

The present experiments were continued as planned not only to provide independent verification or modification of the findings of Berger and Oswald, but also to provide a systematic comparison of two methods of REM deprivation using the *same* subjects as their own control. This experimental design permitted direct comparison of two conditions on both the magnitude and method of compensation. The self-control design also permitted assessment of psychological effects of deprivation of dreaming sleep with and without multiple awakenings.

The partial sleep deprivation (PSD) condition also could provide distinctive information. Dream interruption selectively interferes with REM sleep alone, and extended sleep loss eliminates all stages of sleep. PSD is somewhat intermediate as it involves some deprivation of both REM and nonREM sleep, but a proportionally greater deprivation of the REM stage. How, then, would the priorities of compensation take place? Would the subject held to a limited period of sleep over successive nights tend to squeeze more dreaming into the short sleep period, or would he tend instead to eliminate dreaming entirely on behalf of more nondreaming sleep? Many people live under conditions of partial sleep deprivation for extended periods of time, but the effect of these patterns of living on the sleep cycle and on daytime behavior are almost unknown.

Methods

The subjects were six healthy male college students, age 20 to 29, who volunteered for the experiment and were paid for participation. The experiment was outlined to the subjects in a preliminary screening interview. They were informed that the investigation concerned the effects of different kinds of sleep disruption on the subsequent sleep cycle and on behavior. Subjects D and F knew, however, that the multiple awakenings procedure would interrupt physiologically indicated dreaming.

Subjects were screened for apparent good health, absence of gross psychiatric disorder, and anticipated reliability in conscientiously following the experimental regimen. Subjects were instructed not to sleep outside of the laboratory during experimental runs, to abstain from drugs on any day preceding a laboratory night, and to consume no alcoholic beverages within four hours of retiring, and no more than a single beer or cocktail previous to that. The importance of these conditions for the experiment was carefully explained.

Subjects reported to the laboratory shortly before their usual bedtime. Electrodes were attached to the scalp in the occipital and parietal areas, with the ears as reference, to provide monopolar EEG tracings. Eye movements were recorded with bilateral electrodes placed near the external canthi, and in most instances additional supraorbital and infraorbital placements were used. The subject went to sleep in a quiet, dark room adjacent to the monitoring room. The EEG and eye-movement tracings were recorded continuously throughout the night.

Dream time (REM time) was scored in accordance with procedures detailed elsewhere,[7] except that the onset and termination of each REM period was defined by the first and last rapid-eye-movements of a REM period. Sleep time was scored from the first spindle of the night, with periods of waking record of one minute or longer deleted from total sleep time. All waking periods during which recording was stopped were also noted and deleted from sleep time. The scoring of each night's protocol was verified by a second scorer.

The basic experimental design consisted of four phases, each separated by about a week intermission during which the subject slept at home:

1. *Habituation (H).*—This phase consisted of two undisturbed sleep nights in the laboratory to permit some adaptation to the electrodes, the strange surroundings, and the experience of being an experimental subject. Electrical potentials of eye movements and brain waves were monitored and later analyzed.

2. *Baseline (B).*—The second phase of the experiment consisted of four nights of undisturbed sleep intended to provide benchmark measures of each subject's sleep and dream pattern. Brief psychological tests were administered following the fourth baseline night.

3. *Partial Sleep Deprivation (PSD) and Recovery (PSD-R).*—In the next phase of the experiment, the subject was awakened on three consecutive nights after only about 2½ hours sleep, and then permitted a normal amount of sleep on three or more succeeding nights. The subject was never awoken during a REM period in order to avoid any REM interruption during this experimental condition. After the subject was awoken, he remained under direct experimental supervision until his daytime activities began. There was no direct observation of the subjects during the day, but each subject indicated compliance with the instructions not to nap.

4. *Dream Interruption (DI) and Recovery (DI-R).* In the final phase of the experiment, the subject was aroused shortly after the inception of each rapid-eye-movement period, kept awake for a minimum of three-four minutes, and then allowed to return to sleep until the next REM period. The dream interruption procedure was continued for three consecutive nights, then followed by three consecutive nights of undisturbed sleep.

TABLE 1.—*REM% for Baseline Nights and for Each Recovery Night*

Condition			Subject						All Subjects	
Baseline (24 Nights)	Mean REM %		A 22.5	B 18.6	C 23.1	D 21.4	E 22.6	F 24.4	Mean REM % 22.1	σ 3.81
	Range		(21.3-24.1)	(17.6-20.8)	(16.1-28.7)	(19.2-25.9)	(19.3-26.9)	(19.2-30.5)		
PSD *	Night	1	32.3	22.9	22.0	24.2	19.8	24.1		
Recovery		2	25.9	26.7	22.8	16.5	25.0	33.3		
		3	29.6	23.0	21.5	18.7	27.1	31.2		
		4					25.7	27.1		
		5					23.9	29.8		
	Mean		29.3	24.2	22.1	19.8	24.3	29.1	24.8 †	4.50 †
DI *	Night	1	36.1	26.7	29.4	29.2	26.7	33.6		
Recovery		2	33.7	24.8	25.0	21.2	23.3	27.2		
		3	27.6	24.0	31.0	23.8	27.3	24.8		
	Mean		32.5	25.1	28.6	25.2	25.8	28.5	27.5	3.90

* PSD is Partial Sleep Deprivation, DI is Dream Interruption.
† Mean and sigma based on 18 recovery nights. Nights 4 and 5 omitted for subjects E and F to equalize contribution by each subject.

The PSD and DI conditions were administered in reverse order (DI prior to PSD) to subjects B and D. All subjects were given brief psychological tests again following the third PSD night and the third DI night.

The first subject, A, was also run through a one night total sleep deprivation procedure (TSD) followed by three recovery nights. A seventh subject, G, was recruited late in the study, and run through the three PSD nights only.

Results

Compensatory Dreaming Sleep.—The reduction of REM time by *both* methods—dream interruption and partial sleep deprivation—resulted in a significant overall increase in dreaming sleep on recovery nights (Table 1). This result disconfirmed expectations based on the investigator's REM interruption hypothesis. Recovery REM percents were above the baseline mean at the 0.05 and 0.01 levels respectively by small sample one-tailed *t*-test. Subjects slept longer on the average during baseline nights (Table 2), but the increased REM percent on recovery nights was not due to the longer sleep period. When results were computed for only the first 360 minutes of sleep time, eliminating nights on which subjects slept less than 360 minutes, the mean REM time was 74.4 minutes for 19 baseline nights ($\sigma=15.5$), 88.3 minutes for 19 PSD recovery nights ($\sigma=17.0$), and 93.9 minutes for 18 DI recovery nights ($\sigma=12.1$). The REM percents for both recovery series were above the baseline mean at a probability level better than 0.01 by small sample one-tailed *t*-test for this constant sleep period. Recovery nights were generally characterized by a reduced latency between sleep onset and the first REM period, slightly reduced intervals between REM periods, and slightly longer REM periods. Relatively more dreaming sleep took place in the early part of the night on recovery nights than on baseline nights (Table 3) except for PSD recovery night one.

TABLE 2.—*Selected Results for All Subjects Combined for Each Condition*

Condition		Total Sleep Time, * Mean	REM Time, Mean	%REM Time, Mean	Time to 1st REMP, Median	Interval Between REMPs, Median	REMP Duration, Mean	Time to Fall Asleep, Median
Habituation	Night 1 (*N* 6)	340.3	70.4	20.3	142	81 †	22.8	16.5
	Night 2 (*N*-6)	407.0	82.7	20.4	108	85	20.5	6.5
Baseline	(*N*-24)	394.0	87.4	22.1	80	88	20.7	7.0
PSD	(*N*-18)	152.6	20.0	13.1	72	—	16.7	4.0
PSD recovery	(*N*-18)	450.6	112.7	24.8	70	85	22.2	3.0
DI	(*N*-18)	278.3	15.3	5.5	66	—	—	6.5
DI recovery	(*N*-18)	422.1	116.5	27.5	60	78	24.3	4.5

* All entries are in minutes except % REM time. PSD is Partial Sleep Deprivation, DI is Dream Interruption.
† Based on five subjects. C's sleep pattern was erratic and no interval was computed.

Subjects were slightly more deprived of REM sleep in the DI than in the PSD series. The difference in overall recovery REM percents was not, however, statistically significant when sleep time was held constant at 360 minutes.

The relationship between REM deprivation and subsequent compensatory dreaming sleep did not result from a few extreme cases (Table 1). Every subject showed a striking elevation in REM percent, ranging from about 18% to 60% above his own baseline level, on the first recovery night of undisturbed sleep following the DI series, and subsequent recovery nights usually showed continued elevation. Four of the six subjects also showed a substantial elevation in REM percent during the recovery series following PSD nights. A fifth subject, D, had an elevated REM percent on his first PSD recovery night, but came to the laboratory very late on both the second and third recovery nights and was unable to get a full six hours sleep on either occasion. D's results appear to reflect partial REM compensation on the first recovery night, with further sleep and REM deprivation on the second and third. There was no indication of compensatory REM time during subject C's PSD recovery series, and there was no obvious reason for the absence of a compensatory effect.

Delay in REM Compensation.—Overall, REM compensation took place more rapidly following the DI series with its three nights of selective REM deprivation than following the PSD series with its three nights of (partial) nonREM as well as REM deprivation. Table 3 shows that the amount of dreaming sleep throughout the first PSD recovery night only approximated baseline levels, while dreaming sleep on the first DI recovery night was distinctly elevated. REM compensation following the PSD condition tended to be delayed until a certain amount of nonREM sleep occurred. However, some individual subjects (eg, subject A) did show substantial REM compensation on the first recovery night (Table 1). Subject A also participated in a one night total sleep deprivation experiment followed by three recovery nights, and in this condition as well as in the PSD condition his percent of dreaming sleep on the first recovery night was substantially elevated above his baseline level. There may be individual differences in the hierarchy of compensation between REM and nonREM sleep when both are in deficit.

PSD Series.—On successive nights of partial sleep deprivation there was some tendency for more dreaming sleep to be squeezed into the limited sleep time. Data were available for this condition for seven subjects. For all subjects combined, about twice as much dreaming sleep took place in an equivalent total sleep period on the second and third PSD nights as on the first, or as on baseline nights (Table 4). However, individual patterns were quite variable, which may reflect differences in the hierarchy of compensation between REM and nonREM sleep when both are in deficit.

REM Period Latency.—The latency of the first REM period (ie, the interval between sleep onset and the first REMP) tended to decline with increased REM deprivation, even when there was concurrent deprivation of nonREM sleep. Table 2 shows that median time to the first REM period for all subjects combined was below the baseline median for the PSD, PSD recovery, DI, and DI recovery conditions. During each of these deprivation conditions, latencies of less than 60 minutes were more than twice as frequent as during the baseline condition. REM periods right after sleep onset are relatively rare under normal conditions; during 24 baseline nights only one subject on one occasion had an initial REM period latency of less than ten minutes. Four of six subjects—plus the seventh, for whom no baseline data were available—had one or more REM periods within ten minutes of sleep onset under deprivation conditions. B had *seven* latencies of less than ten minutes in 12 nights of deprivation and deprivation recovery.

Every subject made repeated "dream attempts" on DI nights. The mean number of REM periods during undisturbed baseline nights was 4.2; the mean number of REM period awakenings required on the first DI night was 11.5; and subsequent interruption nights showed a further mean rise. On each DI night, however, the pressure toward REM sleep became insistent only after several hours of nonREM sleep (Figure). The number of awakenings increased but slightly from night to night in the first third of the sleep period in spite of the build-up of REM deficit. In contrast, there

was a dramatic increase in the number of REM awakenings from night to night in the last third of the sleep period. There was also a marked increase in the number of REM attempts from the first to the last third of the sleep period of each interruption night.

Instability of Stage.—Deprivation tended to increase the number of interruptions of ongoing REM periods by nonREM sleep, and also the number of brief REM intrusions into stretches of nonREM sleep. These indications of instability of stage were greatest when both REM and nonREM sleep were in deficit. An instability index was computed by adding together the number of scoreable (lasting one minute or longer) nonREM sleep interruptions of REM periods and the number of isolated rapid-eye-movements for each night. The mean nightly rate of the instability index for all subjects combined across all baseline nights was 5.3. The mean rate for the first DI recovery night was 7.5; and for the first PSD recovery night 11.7. Both increases are statistically significant beyond the 0.01 level by one-tailed *t*-test, as is the difference between the two recovery conditions. All six subjects had a higher instability rate on the first PSD recovery night than on the first DI night or than their own baseline mean. The instability index rate of five subjects was above their baseline mean on the first DI recovery night.

Deliberate Avoidance of Dreaming.—Two subjects, D and F, knew that they would be awoken at the inception of each physiologically indicated dream during the DI series, and also knew of the association between rapid-eye-movements and dreaming. Both made deliberate efforts to prevent themselves from dreaming so as to avoid arousal by a noxious buzzer stimulus. Neither was able to prevent himself from repeated REM attempts; however, both had instances of apparent success in *interrupting the REM process themselves prior to awakening by the experimenter*. Their EEG protocols indicated numerous instances of transient arousal at the very inception of a REM period, before the experimenter could sound the buzzer. The REM process would typically resume minutes after the self-arousal. Following buzzer awakenings, the subjects usually reported the earlier attempt to stop dreaming. Both characterized the attempt as effortful and unpleasant. D described "an anxious feeling in the pit of my stomach" after a self-arousal, and

TABLE 3.—*Cumulative Minutes of REM Time for Varying Lengths of Sleep—All Subjects Combined*

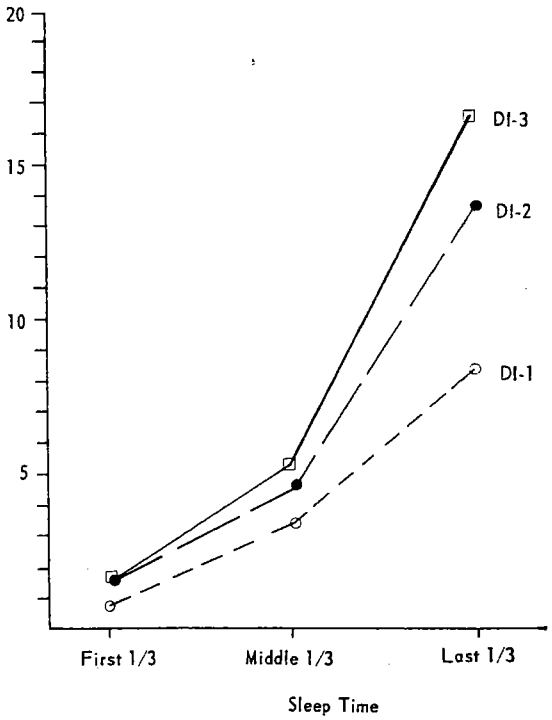
		Sleep Time, Min			
Condition		60	120	240	360
Baseline		1.2	9.2	34.6	74.4[19]
PSD	1	1.5	10.3	34.4	72.8
Recovery	2	6.3	15.9	40.5	95.8[4]
	3	1.7	12.7	48.0	100.9[5]
	4	0	11.9	44.5	89.7
	5	0.3	13.6	39.3	87.2
	Mean	2.6	12.9	41.1	88.3[18]
DI	1	2.8	13.3	49.0	95.4
Recovery	2	3.2	16.1	48.2	91.8
	3	4.5	21.3	56.9	94.5
	Mean	3.5	16.9	51.4	93.9

Baseline entries based on 25 nights—five for F and four for all other subjects; PSD recovery on 22 nights; and DI recovery on 18 nights. Nights were omitted whenever there was less than 360 minutes of sleep time—*N*'s less than the total number are shown by the raised numbers.

TABLE 4.—*Minutes of Sleep Time and REM Time During Three Partial Sleep Deprivation Nights Compared to Baseline*

	Night							
	Baseline		PSD 1		PSD 2		PSD 3	
Subject	ST *	Mean REMT †	ST	REMT	ST	REMT	ST	REMT
A	153	5.4	147	1.1	152	14.6	160	38.9
B	170	18.5	169	39.8	171	36.9	170	39.3
C	149	8.2	150	3.8	150	56.7	146	10.3
D	158	12.1	151	11.8	150	23.1	172	18.0
E	139	15.5	138	19.6	139	21.8	140	15.2
F	151	17.0	150	7.8	152	2.5	150	13.8
G ‡	—	—	150	1.5	146	13.8	151	29.9
Mean	153.0	12.8	150.7	12.2	151.4	24.2	155.6	23.6

* Equated to mean ST allowed on the three PSD nights.
† Based on four baseline nights for each subject.
‡ No baseline data obtained.



Mean number of awakenings for *DI-1, DI-2,* and *DI-3* in the first, third, middle third, and last third of sleep based on means of six subjects.

soon abandoned efforts to prevent dreaming. F continued the effort, and reported that when "his thoughts would get too illogical" he would try to rouse himself and stare straight ahead to avoid eye-movements. These observations suggest that a psychic factor—here, a conscious wish to avoid dreaming—can interfere with the REM process.

Habituation.—As expected, it generally took longer to fall asleep on the first night in the laboratory than on most subsequent nights (Table 2). Also, the first REM period was delayed or "skipped" more frequently on habituation nights than during the baseline condition. No REM period occurred within two hours of sleep onset on 7 of 12 habituation nights compared to 5 of 25 baseline nights. Subject A frequently omitted his "first" REM period. When his data were excluded, nonREM periods occurred within two hours of sleep onset on five of ten habituation nights compared to only 2 of 21 baseline nights.

Comment

The hypothesis that compensatory dreaming sleep is caused by multiple interruptions of the REM process rather than by deprivation is disconfirmed in the present experiment. Deprivation by the PSD as well as the interruption method resulted in compensatory dreaming sleep on recovery nights. Other investigators have reduced nightly REM time without REM interruption by nights of total sleep deprivation,[5,13] and by drugs.[16] These procedures also resulted in gross compensation on recovery nights for the prior REM deficit. The REM interruption hypothesis may be unequivocally rejected. However, none of the reported findings bear on the separate issue of whether or not there is any particular psychologic effect of interrupting ongoing dreams.

Sleep is not homogenous, and we require periods of nonREM as well as periods of REM sleep. The results of the present study are consistent with findings of Berger and Oswald, and of Williams, Hammack, Daly, Dement, and Lubin, that nonREM sleep tends to take precedence over REM sleep when there has been deprivation of all stages. Perhaps nonREM sleep has some particular role in relation to immediate relief of "fatigue" with its physiologic concomitants. In any event, nonREM sleep precedes REM sleep on an ordinary (nondeprivation) night, and most of the night's dreaming sleep takes place only after several hours of nonREM sleep. The interval between sleep onset and the first REM period of the night tends to be longer than usual when the subject is very tired or has stayed up especially late. Roffwarg et al [19] have noted that children have an unusually long latency to the first observed REM period from about the time that they discontinue afternoon naps. Maron et al [15] have reported a mean latency of only 64 minutes to the first REM period for young adult subjects who took an afternoon nap, while subjects who napped in the early evening, when presumably they were more tired, had a mean latency of 84 minutes, approximating that expected at normal bedtime.

The sharp rise in the instability index following the PSD condition suggests a "competition" between REM and nonREM sleep when both are in deficit, rather than a simple hierarchical scheduling of compensation. It is consistent with this view that the latency of the first REM period generally declined with increased REM deprivation even when there was concurrent

nonREM deprivation. The possibility of prominent individual differences in the hierarchy of compensation between REM and nonREM sleep has been mentioned. These differences could reflect differential capacities to delay or suppress dreaming sleep. Rechtschaffen et al [18] have observed the frequent occurrence of REM periods at sleep onset in narcoleptics, and have suggested that this may be due to an (unknown) neurophysiological disorder of the REM triggering process. Subject B in the present study had no complaints of narcolepsy, but had several relatively brief REM latencies on habituation and baseline nights, had a REM period within two minutes of sleep onset on the third PSD night, and had latencies of four, six, and eight minutes respectively on the three PSD recovery nights. His REM period latencies during the DI series dropped to two minutes on the second deprivation night, four minutes on the third, and three minutes on the first recovery night. B reported that he occasionally woke up at home right after sleep onset from a frightening dream that someone had broken into his room and was attacking him. B was a very irritable young man much preoccupied with the difficulty of controlling aggressive feelings. He was aroused at dawn of his second laboratory night by the clatter of garbage cans in the hospital courtyard below, and exploded, "I want to wage cold war on all garbage men. If I ever get my hands on one of those guys I'm going to kill him." The relationship between B's unstable control of rage, his sleep onset nightmares, and his sleep onset REM periods in the laboratory may be fortuitous, but such observations warrant notice at this stage of limited knowledge about the triggering of REM periods.

Neither the present experiment nor other reported REM deprivation studies have demonstrated a psychologic "need to dream." The main line of clearly replicable findings concern compensatory REM sleep following prior deprivation. The experimental subject is deprived of the entire REM stage rather than only the dreaming component. After reviewing various lines of evidence, including Jouvet's demonstration of compensatory phenomena following deprivation of the analogous "rapid sleep" stage in decorticate cats, Snyder recently concluded [20] that present data are more compatible with a physiological explanation than with a hypothetical need to dream in the psychodynamic framework.

The influence of psychodynamic factors on REM sleep is barely explored as yet. Antrobus et al [1] have found that persons who rarely recall dreams have significantly less total REM time than persons who frequently recall dreams. Nonrecallers tended to have as frequent, but shorter, REM periods. These investigators suggested that repressive personality trends might tend to reduce the amount of dreaming as well as to limit dream recall. The tendency for the first REM period to be delayed on habituation nights, when subjects are particularly uneasy about the laboratory situation, might be a related instance of a psychic inhibition of dreaming sleep. We also observed that an intention—the wish to avoid the experimenter's buzzer and full arousal—can lead to voluntary, transient interruption of the REM process. This phenomenon may be analogous to the way in which psychic factors of other kinds—for example, intentions arising from defenses of the ego—may control the emergence of distressing dream content by transiently interrupting the dream or awakening the sleeper.

The absolute difference in REM per cent between the "recall" and "nonrecall" groups in the Antrobus et al study was of the order of magnitude of about 5%. Reported habituation effects on REM sleep have been of generally similar or lesser magnitude. The degree to which psychologic stress can influence the amount of dreaming sleep is not yet known. Psychologic factors may turn out to have an important effect on the sheer amount of REM sleep. On the other hand, the REM stage is a universal aspect of the human sleep cycle, a fundamental biologic process with apparent counterpart in the "rapid sleep" of other mammals, and it would not be surprising if psychodynamic factors turn out to have only a relatively small direct effect on the *amount* of dreaming sleep. The question awaits further research. It will also be important to investigate whether any effects of psychodynamic factors on the amount of REM sleep are mediated primarily through disturbances of falling asleep, disruptions of ongoing sleep, and interruptions of REM periods, or operate more directly on the development or triggering of the REM stage.

Summary

The nightly amount of dreaming sleep of six experimental subjects was reduced by two methods—dream interruption and partial sleep deprivation—to determine whether compensatory dreaming sleep could be attributed to dream interruption rather than deprivation. Both methods of reducing dreaming sleep resulted in compensatory REM time on recovery nights, disconfirming the dream interruption hypothesis. The results of the experiment were also consistent with other findings that nonREM sleep tends to take precedence over REM sleep when there has been deprivation of all stages; and a possible special relation of nonREM sleep to the relief of fatigue with its physiologic concomitants was suggested. Individual differences were found in the hierarchy of compensation between REM and nonREM sleep when both were in deficit.

The functional significance of REM sleep is still unclear. REM deprivation studies have not demonstrated a "need to dream" in the psychodynamic sense, but there is some evidence that psychologic factors may inhibit, interrupt, and perhaps shorten the REM stage. The degree to which psychodynamic factors influence the amount of REM sleep, and the mechanisms of this influence, require further investigation.

This study was supported by grant No. MH-05723 National Institute of Mental Health, United States Public Health Service.

Satoshi Hibi assisted in data collection, scoring protocols, and data analysis. Stanley Fong and Jocelyn Cerul also participated in the work.

REFERENCES

1. Antrobus, J. S.; Dement, W.; and Fisher, C.: Patterns of Dreaming and Dream Recall: EEG Study, J Abnorm Soc Psychol 69:341-344, 1964.

2. Aserinsky, E., and Kleitman, N.: Regularly Occurring Periods of Eye Motility and Concomitant Phenomena During Sleep, Science 118:273-274, 1953.

3. Aserinsky, E., and Kleitman, N.: Two Types of Ocular Motility Occurring in Sleep, J Appl Physiol 8:1-10, 1955.

4. Berger, R.: Tonus of Extrinsic Laryngeal Muscles During Sleep and Dreaming, Science 134:840, 1961.

5. Berger, R. J., and Oswald, I.: Effects of Sleep Deprivation on Behavior, Subsequent Sleep, and Dreaming, J Ment Sci 108:457-465, 1962.

6. Dement, W.: Effect of Dream Deprivation, Science 131:1705-1707, 1960.

7. Dement, W.: Manual for Scoring EEG Records for REM Periods (Dream Time), unpublished data.

8. Dement, W., and Kleitman, N.: Cyclic Variations in EEG During Sleep and Their Relation to Eye Movements, Body Motility, and Dreaming, Electroenceph Clin Neurophysiol 9:673-690, 1957.

9. Dement, W., and Kleitman, N.: Relation of Eye Movements During Sleep to Dream Activity: Objective Method for Study of Dreaming, J Exp Psychol 53:339-346, 1957.

10. Dement, W., and Wolpert, E. A.: Relation of Eye Movements, Body Motility, and External Stimuli to Dream Content, J Exp Psychol 55:543-553, 1958.

11. Fisher, C., and Dement, W. C.: Studies on Psychopathology of Sleep and Dreams, Amer J Psychiat 119:1160-1168, 1963.

12. Foulkes, W. D.: Dream Reports From Different Stages of Sleep, J Abnorm Soc Psychol 65:14-25, 1962.

13. Goodenough, D. R., et al: Comparison of "Dreamers" and "Nondreamers": Eye Movements, Electroencephalograms, and the Recall of Dreams, J Abnorm Soc Psychol 59:295-302, 1959.

14. Kamiya, J.: "Behavioral, Subjective, and Physiological Aspects of Sleep and Drowsiness," in Fiske, D. W., and Maddi, S. (eds.): Functions of Varied Experience, Homewood, Ill: Dorsey Press, 1961, pp 145-174.

15. Maron, L.; Rechtschaffen, A.; and Wolpert, E. A.: Sleep Cycle During Napping, Arch Gen Psychiat 11:503-508, 1964.

16. Rechtschaffen, A., and Maron, L.: Effect of Amphetamine on Sleep Cycle, Electroenceph Clin Neurophysiol 16:438-445, 1964.

17. Rechtschaffen, A.; Verdone, P.; and Wheaton, J.: Reports of Mental Activity During Sleep, Canad Psychiat Assoc J 8:409-414, 1963.

18. Rechtschaffen, A., et al: Nocturnal Sleep of Narcoleptics, Electroenceph Clin Neurophysiol 15: 599-609, 1963.

19. Roffwarg, H. P.; Dement, W. C.; and Fisher, C.: "Preliminary Observations of Sleep-Dream Pattern in Neonates, Infants, Children and Adults," in Harms, E. (ed.): Monographs on Child Psychiatry No. 2, New York: Pergamon Press, 1964, pp 60-72.

20. Snyder, F.: New Biology of Dreaming, Arch Gen Psychiat 8:381-391, 1963.

21. Snyder, F.; Hobson, J. A.; and Goldfrank, F.: Blood Pressure Changes During Human Sleep, Science 142:1313-1314, 1963.

22. Snyder, F., et al: Changes in Respiration, Heart Rate, and Systolic Blood Pressure in Human Sleep, J Appl Physiol 19:417-422, 1964.

23. Williams, H. L., et al: Responses to Auditory Stimulation, Sleep Loss and EEG Stages of Sleep, Electroenceph Clin Neurophysiol 16:269-279, 1964.