Sleep, 15(5):400–403

Precise Conservation of NREM Period 1 (NREMP1) Delta Across Naps and Nocturnal Sleep: Implications for REM Latency and NREM/REM Alternation

*I. Feinberg, *T. Maloney and †J. D. March

*Northern California System of Veterans Administration Clinics and the Department of Psychiatry, University of California at Davis, Davis, California, U.S.A.
†Delta Software, San Francisco, California, U.S.A.

Summary: The delta integrated amplitude (DIA) in nonrapid eye movement period 1 (NREMP1) of daytime naps was precisely subtracted from the NREMP1s of ensuing nocturnal sleep, indicating that the brain can retain a record of DIA expressed in sleep episodes initiated 12.5 and 8.5 hours before nocturnal sleep onset. The DIA subtraction was primarily accomplished by reduced NREMP1 duration [earlier rapid eye movement (REM) onset], suggesting that the timing of REM period 1 (REMP1) onset is controlled by delta need. This result is consistent with the hypothesis that REM sleep occurs when a stimulus for NREM has been partially depleted. **Key Words:** Sleep—EEG—Naps—Delta—Homeostasis—Period-amplitude—Computer.

In a previous report (1), we demonstrated precise homeostatic conservation of delta electroencephalogram (EEG) across a 5:00 p.m. nap and postnap sleep. In that study, conservation was the result of reduced delta in several nonrapid eye movement periods (NREMPs) of postnap sleep. In this study, we show that when naps are taken earlier in the day, delta conservation occurs entirely within NREM period 1 (NREMP1). A more extensive presentation of these results will appear in a future publication.

METHODS

Subjects were paid college student volunteers who gave informed consent. For the morning nap, n = 27, mean age = 21.3 years (SD = 1.7), females/males = 18/9. For the afternoon nap, n = 16, mean age = 21.6 years (SD = 2.1), females/males = 11/5. We chose a larger sample for the morning nap because a previous study (2) suggested that any effects of a morning nap on delta EEG might be quite small.

Subjects underwent sleep laboratory recording on an adaptation night, a baseline night (BN), a nap (either at 11:00 a.m. or 3:00 p.m.), and a postnap night (PNN). For the adaptation, BN and PNN, subjects were in bed with lights out from 11:30 p.m. to 7:00 a.m. During the naps, subjects were instructed to remain in bed for 2 hours and to sleep as much as possible.

EEG and electrooculogram (EOG) measures were amplified and recorded on a Grass Model 78 polygraph. Period and amplitude (PA) analysis algorithms (3) were applied to the C3-A2 EEG as described in an earlier study (4). The PA measure used here is delta (0.3–3 Hz) integrated amplitude (DIA), which is almost exactly proportional to power density in spectral analysis (5,6). Visual sleep stage scoring was carried out without knowledge of the computer measures.

RESULTS

Table 1 shows that sleep latency increased slightly above baseline (by 5–7 minutes) on the PNNs. Total sleep time (TST) was the same in the morning and afternoon naps (94.3 and 94.9 minutes). The naps did not significantly reduce TST on the postnap nights. One subject did not have REM sleep in the morning nap and two subjects had no REM sleep in the afternoon nap. For subjects with REM sleep, mean REMP1 duration in the 11:00 a.m. nap was significantly greater

Accepted for publication July 1992.
Address correspondence and reprint requests to I. Feinberg, M.D., Med:Psych TB 148, University of California at Davis, Davis, California 95616, U.S.A.

TABLE 1. *Mean (and SEM) sleep measures for baseline night (BN), nap and postnap night (PNN) for the 11:00 a.m. and 3:00 p.m. nap conditions*

	11:00 a.m. Nap (n = 27)[a]				3:00 p.m. Nap (n = 16)[a]			
	BN	Nap	PNN	p[b]	BN	Nap	PNN	p[b]
Sleep latency	16.8	15.8	22.0	0.051	22.0	14.0	28.2	
(Minutes)	(2.0)	(3.2)	(2.8)		(3.9)	(2.9)	(2.8)	
Total sleep	417.7	94.3	413.7		410.0	94.9	414.5	
(Minutes)	(6.0)	(3.7)	(6.3)		(6.4)	(6.1)	(3.8)	
NREMP1								
Duration	92.3	47.0	72.7	0.051	89.3	51.7	57.3	0.004
(Minutes)	(7.3)	(4.0)	(6.7)		(9.3)	(5.0)	(2.7)	
0.3–3 Hz EEG								
Total integrated amplitude (IA)	120.5	33.5	90.6	0.026	110.5	45.8	65.0	<0.001
(μV·second × 10^3)	(11.8)	(4.0)	(10.0)		(10.9)	(8.2)	(7.3)	
IA/epoch	433.0	223.0	406.0		422.0	270.0	368.0	0.016
(μV·second)	(21.5)	(15.5)	(24.6)		(27.8)	(22.2)	(34.6)	
Time/epoch	12.6	9.3	12.3		12.4	10.0	11.8	0.062
(Seconds)	(0.2)	(0.3)	(0.3)		(0.3)	(0.4)	(0.4)	
Average sample amplitude[c]	33.9	23.5	32.5		33.7	26.4	30.4	0.012
(μV)	(1.3)	(1.1)	(1.5)		(1.5)	(1.2)	(2.0)	
REMP1								
Duration	15.6	24.7[d]	14.4		22.9	27.7	20.4	
(Minutes)	(2.9)	(3.0)	(1.4)		(3.9)	(3.5)	(4.2)	

[a] For REMP1, n = 26 and n = 14 in the 11:00 a.m. and 3:00 p.m. studies; only those subjects with REM in the nap were included.
[b] Paired two-tailed *t* tests, BN vs. PNN.
[c] Average sample amplitude is equal to integrated amplitude (μV·sec) in 0.3–3 Hz divided by Time (sec) in the 0.3–3 Hz band.
[d] Significantly greater than BN (p = 0.011) and PNN (p = 0.003).

than in baseline and postnap nights. For the 3:00 p.m. nap, REMP1 duration did not differ significantly across conditions. REM occurring in the naps did not reduce the amount of REM in the PNNs, a result consistent with the general nonconservation of REM sleep (4).

Table 1 shows that NREMP1 duration was reduced below BN on both postnap nights. DIA in NREMP1 was significantly reduced below its baseline level on each postnap night. The amount of this reduction was almost exactly equal to the DIA expressed in the NREMP1 of each nap. Therefore, for NREMP1: $DIA_{NAP} + DIA_{PNN} = DIA_{BN}$ (see Fig. 1). The conservation was within 3% and 0.3% of baseline for the morning and afternoon naps, respectively. Examination of frequencies within 0.3–3 Hz showed that the conservation was most precise (within 1% for both naps) for the 1–2 Hz band, which contained about 53% of the total DIA in 0.3–3 Hz.

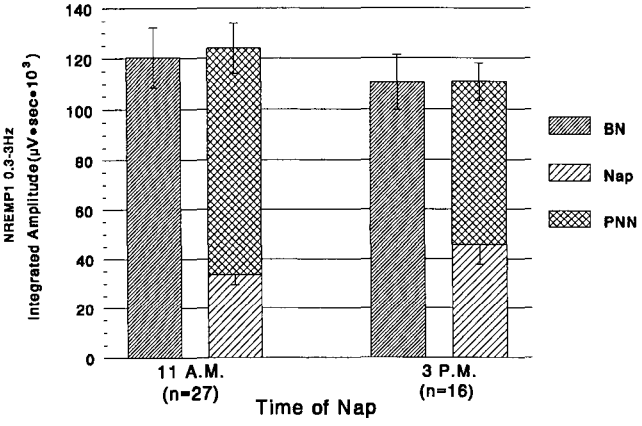

FIG. 1. Delta (0.3–3 Hz) integrated amplitude in NREMP1 on Baseline Night (BN), Nap and Postnap night (PNN) for naps initiated at 11:00 a.m. or 3:00 p.m. For both nap conditions, DIA in Nap + PNN = DIA in BN.

Although the delta conservation in both nap studies was quite precise, it is important to note that this is an average effect that does not hold within each individual. This variability is characteristic of all sleep cycle sums and is one of the factors that led us to propose that these cycles are more likely the result of pulsatile endocrine processes than of the metabolic consumption of an accumulated substrate.

The compensatory DIA reduction in NREMP1 on both PNNs resulted mainly from the substantial truncation of NREMP1 duration (i.e. earlier REM onset) noted above. In addition, Table 1 shows that the rate of delta production (average DIA/20 seconds) was significantly reduced after the afternoon nap as a result of a significant decrease in average delta wave amplitude and a near-significant decrease in delta wave incidence (delta time/20 seconds). Similar effects were observed after the morning nap, but these changes were smaller and not statistically significant.

Figure 2 plots total DIA/NREMP across NREMPs 1–4 for baseline and postnap nights. The DIA effects of the nap were clearly limited to NREMP1 of postnap sleep. As a consequence, the DIA patterns in Fig. 2 do not describe an exponential decline on either PNN.

Our main focus in this study was on the effects of nap DIA on the quantity of DIA/NREMP in subse-

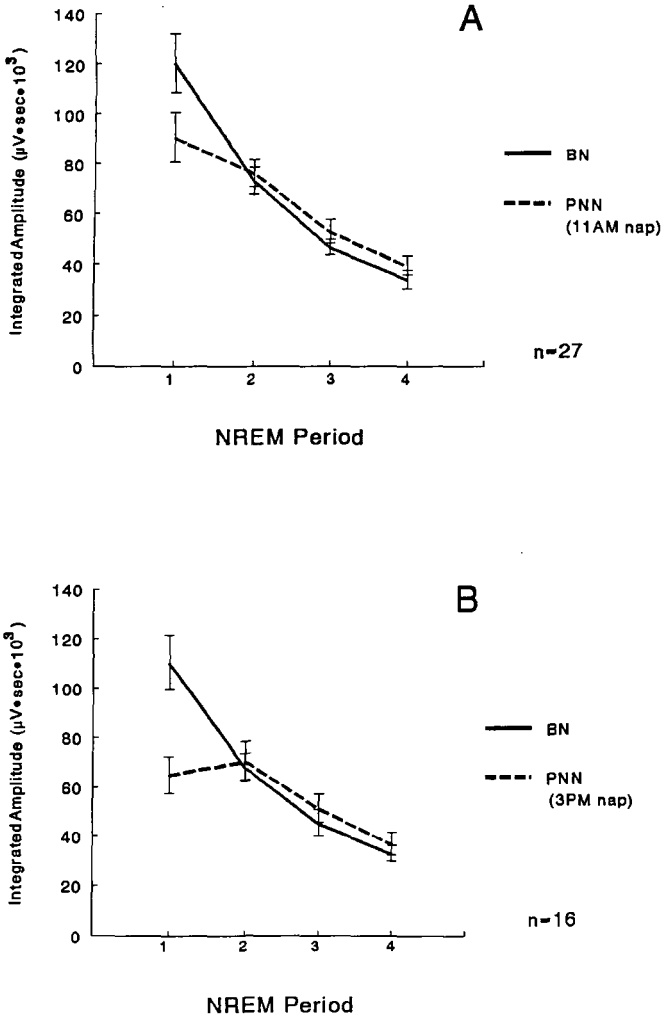

FIG. 2. Total delta integrated amplitude in NREMP1–4 for BN and PNN in 11:00 a.m. nap condition (A) and 3:00 p.m. nap condition (B). DIA was significantly reduced on both PNNs only in NREMP1. DIA in NREMP2–4 was slightly (but not significantly) higher than the corresponding BN levels in NREMP2–4. The declining trend of DIA across NREMPs on the baseline night replicates our original description (3). The flattening of the DIA curve on PNNs produced by the depressed value in NREMP1 is inconsistent with the hypothesis (7) of an exponential decline of DIA across NREMPs and with quantifications (8) based on that hypothesis.

quent sleep. However, we also examined the perturbations produced by naps on the instantaneous rate of DIA production (DIA/20 seconds epoch), because this rate is critical to the hypotheses underlying the recovery models (7,8) of delta sleep.

Table 2 shows that the morning nap did not affect delta rate on the PNN. However, the afternoon nap produced a significant reduction in DIA/epoch on the PNN that was limited to NREMP1. Subsequent NREMPs were clearly unchanged from baseline, a result inconsistent with the hypothesis that delta rate declines exponentially across sleep. Also inconsistent with this hypothesis (and with the pattern of baseline sleep) was the observation that the decrement from

TABLE 2. *Rate of expression of delta integrated amplitude (DIA/20 seconds) by nonREM period on baseline night (BN) and postnap night (PNN) for naps initiated at 11:00 a.m. and 3:00 p.m. Mean (and SEM) in μV·seconds*

	NonREM period			
	1	2	3	4
11:00 a.m. nap				
BN	433 (22)	340 (24)	241 (14)	208 (14)
PNN	406 (25)	334 (23)	247 (16)	220 (14)
3:00 p.m. nap				
BN	422 (28)	302 (21)	209 (17)	201 (16)
PNN	368[a] (35)	315 (31)	234 (21)	213 (14)

[a] Paired two-tailed *t* test BN vs. PNN, p = 0.016.

NREMP1 to NREMP2 on PNN_{PM} (53 μV × seconds) was actually smaller than the decrement from NREMP2 to NREMP3 (93 μV × seconds).

DISCUSSION

The results of these experiments are consistent with the major premise of the homeostatic model (7) of delta sleep: the quantity of delta is conserved. These data show that this conservation is remarkably precise for a biological measure such as sleep. However, the homeostatic model would not have predicted the pattern of conservation, i.e. that delta effects on the postnap nights would be limited to NREMP1.

The original homeostatic model of delta sleep (7) conceptualized the restorative process (process SW) as metabolic, in which a substrate produced by waking brain activity was consumed during sleep. This model proposed that the density/amplitude of delta EEG was proportional to the rate of consumption. Since the rate of a metabolic process declines exponentially as its substrate is consumed, delta density/amplitude (measured either as integrated amplitude or as power density per epoch) should decline exponentially across sleep. This basic notion was incorporated in Daan et al.'s quantification (8) of the two-process model. However, the data here are squarely inconsistent with an exponential decline of delta per NREMP or per epoch. This model would predict that DIA/NREMP and DIA/epoch on the postnap nights would be at lower levels than baseline on NREMP1 and continue at reduced levels on subsequent NREMPs, following the logarithmic slope of baseline.

Just as existing sleep models do not predict that delta conservation following daytime naps would occur entirely within the first NREMP, they would not predict the manner of conservation within NREMP1. A priori, the DIA of the naps could have been subtracted from postnap NREMP1 either by shortening its duration or by lowering its average rate of delta production. That DIA was reduced primarily by shortening NREMP1

(i.e. earlier REM onset) must hold important implications for sleep regulatory mechanisms.

That REMP1 onset on the PNNs occurred when the homeostatic DIA level in NREMP1 was reached supports (but does not prove) the hypothesis we proposed for NREM/REM alternation (9,10): REM sleep occurs when an NREM stimulus has been partially depleted. According to this hypothesis, some unknown stimulus (perhaps neuroendocrine) periodically triggers pulses of NREM across the night. These pulses depress arousal, produce neural inhibition and functional deafferentation and prevent or impair memory consolidation. In the EEG, these pulses stimulate delta and suppress beta (5). When the effectiveness of the hypothetical NREM stimulus wanes below some critical level, REM sleep occurs either as a passive escape from inhibition or because a REM control center is triggered. The decline in delta across sleep could reflect diminishing strength of successive NREM pulses, increasing resistance to their effects or both. Isolated bursts of REM sleep (as in narcolepsy) could result from very weak pulses. We think that this pulsatile model may explain existing data better and more parsimoniously than the metabolic hypothesis.

The results here also have clinical implications because an abnormally short NREMP1 (REM latency) is sometimes seen in psychiatric illness. Our data provide experimental support for an hypothesis we advanced (11) in 1969 [and frequently reiterated (cf. 12,13)]: short REM latencies could reflect abnormal NREM sleep rather than heightened REM pressure. Further insight into the mechanisms that produced the delta homeostasis found here could shed light on altered brain physiology in mental illness.

Acknowledgements: This research was supported in part by the Department of Veterans Affairs, by AG 5 RO1 07224 and by PHS SBIR 5R43 43066. We thank F. Travis, Ph.D., for assistance in all phases of the study and Edward Wakabayashi for meticulous data collection and scoring. Jean K. Moore, Ph.D., and Martin Wilson, Ph.D., made helpful suggestions on earlier versions of this manuscript.

REFERENCES

1. Feinberg I, March JD, Floyd TC, Jimison R, Bossom-Demitrack L, Katz PH. Homeostatic changes during post-nap sleep maintain baseline levels of delta EEG. *Electroenceph Clin Neurophysiol* 1985;61:134–7.
2. Karacan L, Williams RL, Finley WW, Hursch CJ. The effects of naps on nocturnal sleep: influence of the need for stage 1 REM and stage 4 sleep. *Biol Psychiatr* 1970;2:391–9.
3. Feinberg I, March JD, Fein G, Floyd TC, Walker JM, Price L. Period and amplitude analysis of 0.5–3 c/second activity in NREM sleep of young adults. *Electroenceph Clin Neurophysiol* 1978;44:202–13.
4. Travis F, Maloney T, Means M, March JD, Feinberg I. Acute deprivation of the terminal four hours of sleep does not increase Delta (0–3 Hz) electroencephalograms: a replication. *Sleep* 1991; 14:320–4.
5. Ktonas PY, Gosalia AP. Spectral analysis vs. period-amplitude analysis of narrow band EEG activity: a comparison based on the sleep delta-frequency band. *Sleep* 1981;4:193–206.
6. Uchida S, Maloney T, Feinberg I. Beta (20–28 Hz) and delta (0.3–3 Hz) EEG oscillate reciprocally across NREM and REM sleep. *Sleep*, 1992 (in press).
7. Feinberg I. Changes in sleep cycle patterns with age. *J Psychiatr Res* 1974;10:283–306.
8. Daan S, Beersma DGM, Borbely A. Timing of human sleep: recovery process gated by a circadian pacemaker. *Am J Physiol* 1984;246:R161–R178.
9. Feinberg I, March JD. Cyclic delta peaks during sleep: result of a pulsatile endocrine process? [letter]. *Arch Gen Psychiatr* 1988; 45:1141–2.
10. Feinberg I, March JD, Flach K, Maloney T, Chern W-J, Travis F. Maturational changes in amplitude, incidence and cyclic pattern of the 0 to 3 Hz (Delta) electroencephalogram of human sleep. *Brain Dysfunction* 1990;3:183–92.
11. Feinberg I, Braun M, Koresko RL, Gottlieb F. Stage 4 sleep in schizophrenia. *Arch Gen Psychiatr* 1969;21:262–6.
12. Feinberg I, Hiatt JF. Sleep patterns in schizophrenia: a selective review. In: Williams RL, Karacan I, eds. *Sleep disorders: diagnosis and treatment.* New York: J. Wiley & Sons, Inc., 1978; 205–31.
13. Hiatt JF, Floyd TC, Katz PH, Feinberg I. Further evidence of abnormal non-rapid-eye-movement sleep in schizophrenia. *Arch Gen Psychiatr* 1985;42:797–802.