

The Effect of the Occurrence of REM Sleep During Morning Naps on Mood After Sleep Deprivation in Patients with Major Depressive Disorder

D. Riemann, M. Wiegand, J. Zulley, Ch. Lauer, W. Schreiber, and M. Berger
(Mannheim and Munich)

Total sleep deprivation (TSD) has been established as a successful though short lasting treatment modality in depressed patients. Anecdotal reports (1) indicate that the beneficial effect of sleep deprivation may be reversed by naps even as short as 90 seconds during the day following the sleepless night. This paradoxical phenomenon, that sleeplessness ameliorates depressive symptomatology and that depression usually returns after the next night of sleep or even after short naps, raises the question, if in depressed patients, sleep may have a depressogenic influence? Nap studies conducted up to now draw an inconsistent picture. Gillin et al. (2) and Giedke (3) did not find detrimental effects of naps on mood, either during the morning or the afternoon, in successfully sleep deprived depressed patients. However, in one study (2), only very brief naps were allowed (≤ 10 minutes), whereas the patients in the other study (3) were not free of psychoactive medication. A study by our group (4) showed that naps at 1300 h reestablished depressive symptomatology in approximately half of the patients, particularly when the nap included REM sleep. This effect was, however, confounded with a longer sleep length in the patients with REM-naps. The present investigation aimed at evaluating the questions: (a) whether morning naps are capable of reinducing depression in sleep deprived depressed patients, (b) if a relapse is related to the occurrence of REM sleep during these naps, when controlling for sleep length?

Method

25 depressed inpatients (17 females, 8 males), all fulfilling Research Diagnostic Criteria (5) for major depressive disorder (subtype endogenous, with 3 patients being bipolar II), participated in the study. Mean age (\pm SD) was 49.3 ± 12.9 y. Only subjects with a baseline 21-Item Hamilton-Depression (HAMD) score ≥ 18 points were included. The mean 21-HAMD score (\pm SD) was 28.0 ± 4.9 (one rater). After a drug wash-out phase of at least seven days, subjects slept for two nights in the sleep laboratory, and sleep was recorded and scored according to standard procedures. Patients were sleep deprived the following night. The next morning at 0900 h a nap was scheduled. According to the design, naps were terminated: (i) if no sleep (i. e. stage 2) occurred after 60 minutes, (ii) after spontaneous awakenings (intermittent waking interval ≥ 5 minutes); or (iii) by experimental awakenings (either 2 minutes after the termination of a REM period or after a NonREM-sleep period equalling a previous REM-nap in length. Mood was rated prior to TSD (0830 h) and on the next day (0830 h, after the nap and at 1500 h) by means of the 6-HAMD by a rater unaware of the nap structure.

Results and Discussion

Of the 25 patients, 15 (60%) responded favourably to TSD when utilizing a response criterion of at least 30% improvement on the 6-HAMD. Responders did not differ from non-responders in terms of sex, age or severity of depression ratings (21-HAMD at beginning of the study). Regarding the baseline REM latency (second night of the study), responders tended to show shorter values (mean \pm SD: 40.3 \pm 30.2 min) than non-responders (55.4 \pm 30.4 min.).

For the analysis of nap effects, only data from patients responding to TSD will be presented. One of the 15 responders was not able to fall asleep within 60 minutes after 0900 h, and therefore, was discarded from the data analysis. Figure 1 depicts the course of mood for patients with REM-naps and patients with NonREM-naps.

As can be seen, both groups included seven patients. For the whole sample, and when comparing post-nap 6-HAMD values to 0830 h values, naps led to a significant impairment of mood ($p < 0.05$, Wilcoxon-test, two-tailed). By 1500 h, however, the 6-HAMD ratings did not differ significantly from pre-nap ratings. When analysing 6-HAMD values separately for the two groups (i. e. REM- and NonREM-naps) in neither group did mood worsen to a statistically significant extent after naps or at 1500 h in comparison with pre-nap values. When applying a Δ -HAMD difference of two points as an indicator of impairment of mood after naps, three patients from the REM group and four patients from the NonREM-group worsened after the nap. Impairment of mood was not coupled to the kind of awakening. Of the seven patients who worsened, six awoke spontaneously, and of the seven patients who showed no decrease in mood, two had been awoken experimentally. When comparing its nap-sleep structure of REM and NonREM-naps (Tab. 1), no statistical difference emerged between the two conditions.

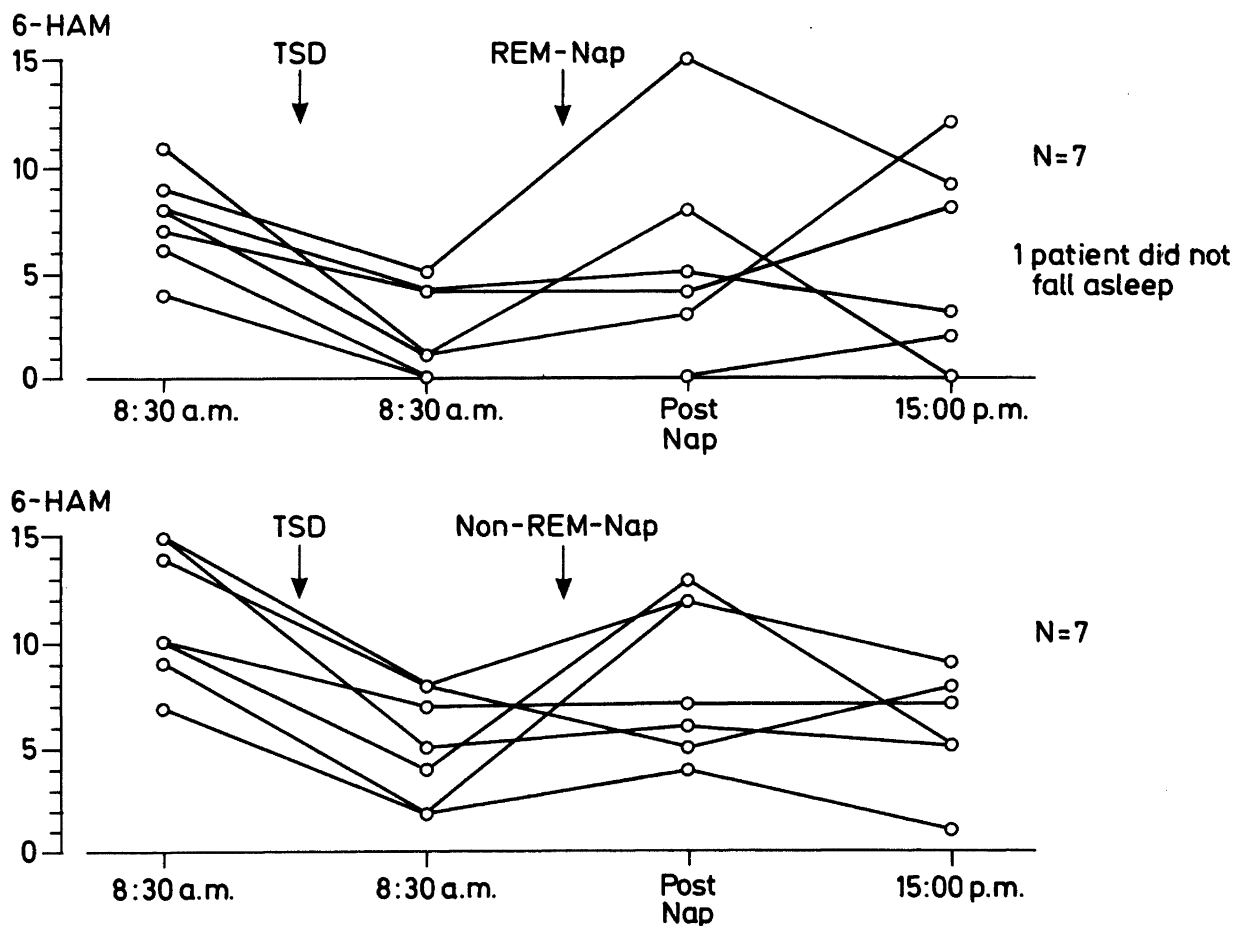


Fig. 1: Influence of REM- vs. NonREM-naps on mood in 14 TSD responders.

Tab. 1: Nap-sleep structure (Mean \pm SD).

	NonREM-naps n = 7	REM-naps n = 7	U-Test, two-tailed
Sleep latency min.	8.3 \pm 7.5	6.1 \pm 3.7	n.s.
SWS/min.	10.9 \pm 14.6	11.9 \pm 15.1	n.s.
Sleep period time min.	34.4 \pm 10.2	58.9 \pm 39.5	n.s.
REM latency min.	–	53.1 \pm 37.9	–
REM/min.	–	4.7 \pm 3.0	–

When correlating sleep length with its nap-induced mood change, a non-significant coefficient was found ($r = 0.12$; Spearman rank correlation).

In disagreement with two previous studies (2, 3), it was found that morning naps led to a significant impairment of mood in 14 successfully sleep deprived patients shortly after the naps. The worsening of mood was not related to the occurrence of REM sleep, to the length of sleep episodes, or to the kind of awakening. However, at 1500 h mood values were no longer impaired compared to pre-nap scores. The present study underlines the importance of keeping depressed patients fully awake after total sleep deprivation. Although the presumed depressogenic factor of naps could not be identified, the nap paradigm seems to offer a promising method of investigating mood changes in depressed patients during short time intervals.

References

1. Roy-Byrne, P. R., Uhde, T. W., Post, R. M. in: *Neurobiology of mood disorders*; Post, R. M., Ballenger, J. C. (eds.) 817–835, Williams & Wilkins, Baltimore, 1984.
2. Gillin, J. C., Risch, S. C., Janowsky, D., Kripke, D., *Sleep Res.* 16, 273, 1987.
3. Giedke, H., in: *Sleep '86*; Koella, W. P., Obál, F., Schulz, H., Visser, P. (eds.) 451–453, Gustav Fischer, Stuttgart, New York, 1988.
4. Wiegand, M., Berger, M., Zully, J., Lauer, C., von Zerssen, D., *Biol. Psychiat.*, 22, 389–392, 1987.
5. Spitzer, R. L., Endicott, J. E., Robins, E.: *Research diagnostic criteria for a selected group of functional disorders*, 3rd ed., National Psychiatric Institute, Biometric Research, New York, 1977.