The Influence of Daytime Naps on the Therapeutic Effect of Sleep Deprivation

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Introduction

The antidepressant effect of total sleep deprivation has been noted by several observers (Gerner et al. 1979; Gillin 1983). It has also been reported that in some cases, even a short nap on the day following sleep deprivation can provoke a mood setback, or even a marked exacerbation of depression in patients who had improved (Pflug and Tolle 1971; Pflug 1972; Knowles et al. 1979; Roy-Byrne et al. 1984). These anecdotal accounts, however, have thus far not been systematically repeated in an experimental manner.

Beyond its clinical importance, the possible reversal of the benefits of sleep deprivation by a short nap carries some interesting theoretical implications. Stated simply, the depressogenic sleep theory declares that sleep may induce depression and that sleep deprivation relieves it. The additional hypothesis has been advanced
that the depressiogenic effect of sleep might be specifically due to rapid eye movement (REM) sleep. This hypothesis can be inferred from the antidepressant effect of selective REM sleep deprivation (Vogel et al. 1975, 1980) and from the fact that almost all antidepressant drugs suppress REM sleep (Chen 1979).

In the present preliminary study, we examined the effect of a daytime nap subsequent to total sleep deprivation on the psychopathology of depressive patients. We were especially interested in the relationship between the occurrence of REM sleep during naps and mood deterioration.

Methods

Twelve patients (eight women, four men) with a major depressive disorder were included in the study, eight of whom were classified as “endogenous subtype” according to Research Diagnostic Criteria (RDC). The mean age of the patients was 40.7 ± 14.2 years, and the mean baseline score in the 21-item Hamilton Depression Scale was 26.52 ± 6.84. All patients were free from antidepressant medication for at least 8 days prior to the first night of sleep deprivation and for the duration of the whole study period. A drug-free interval of 7 days has been found to be sufficient to exclude significant medication effects on nocturnal sleep (Berger et al. 1983).

After the washout period, two adaptation nights in the sleep laboratory were arranged. These were followed by one night of total sleep deprivation. Next day, the patients were asked to take a nap in the sleep laboratory at 1:00 PM, during which a sleep electroencephalogram (EEG) was recorded. This procedure was repeated 1 week later. The two procedures differed only with respect to the time of termination of the nap: an “REM nap” was terminated by an awakening after the first REM episode and a “non-REM nap” by waking the patient up immediately when an REM episode occurred. Those patients who woke up spontaneously without having had an REM episode were included in the latter group. The sequence of both procedures was randomized and was blind to patient and rater. In two patients, there was an additional repetition, with naps terminated by spontaneous awakenings. Mood changes during sleep deprivation and naps were scored by means of the 6-item version of the Hamilton Depression Scale (Bech et al. 1975). Efficacy of the sleep deprivation was arbitrarily defined as a decrease of at least 30% in the score of the Hamilton Scale. “Mood change” during nap was defined as an increase of at least 2 points. Self-ratings were performed using the Adjective Mood Scale (AMS) (von Zerssen 1986).

Results

Twelve patients underwent a total of 23 sleep deprivations with succeeding naps. Only 3 of them responded repeatedly to deprivations. As this number was too small for intraindividual comparisons of REM naps versus non-REM naps, we confine ourselves to a cumulative description. As the data are partially interdependent (due to the repetition of the sleep deprivation procedure), statistical analysis has to be restricted to selected basic descriptive parameters.

Twelve responses and 11 nonresponses to sleep deprivation occurred. In the cases of nonresponse, a subsequent nap, in general, had no apparent effect on mood. In the cases of response, six exhibited a relapse of depressive symptomatology after a nap (Figure 1). There was a high correlation (0.88) between observer-rated mood changes and patients' self-ratings of changes according to the AMS. In five of six naps that led to a worsening of mood, REM sleep appeared. In the six naps that had no effect on mood, REM sleep was either not present or was seen in a very rudimentary form (1.5 min or less). There was, however, a difference in mean nap sleep time between those cases who felt worse (119.67 ± 39.2 min) as opposed to those who did not show mood change (61.8 ± 33.2 min). There was no obvious difference in other nap sleep parameters or in the baseline psychopathology. We found no indication of age and gender effects, but a definite statement is not possible because of the limitations of statistical analysis mentioned above.
Discussion

These results support the hypothesis that sleep may have a potentially depressiogenic effect in depressed patients. A short sleep episode after one night of sleep deprivation can reverse a mood improvement and can cause a marked relapse into depression, going well beyond the slight temporary discomfort that might result from being awakened. In contrast, a mood decline is not observed in healthy subjects, who exhibit a restoration of mood after a nap following sleep deprivation (Taub et al. 1976; Naitoh 1981). This favors the hypothesis that the depressiogenic effect of sleep is specific to depressive patients. However, nothing has yet been reported about the effect of naps in depressive patients without previous sleep deprivation.

Our data point to the role of REM sleep as a crucial factor involved in mood deterioration. Nevertheless, a definite conclusion about this
role cannot yet be drawn, as the effect of nap sleep time could not be differentiated from that of the occurrence of REM sleep. Further studies are needed to clarify this problem.

References


