PAPER **220**

DISTINGUISHING BETWEEN TRANSCENDENTAL MEDITATION AND SLEEP ACCORDING TO ELECTROPHYSIOLOGICAL CRITERIA

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More powerful statistical re-analyses of the data of Pagano et al. (1976) revealed significant differences in the EEG distributions of the Transcendental Meditation technique and 'napping'. Other studies indicating similar differences are discussed.—EDITORS

Wallace et al. (1972) stated that Transcendental Meditation (TM) produced a "wakeful hypometabolic state." Yet Pagano et al. (1976) claimed no significant difference in time spent asleep during TM and napping. More powerful statistical reanalyses of Pagano's data reveal significant differences in EEG distributions of TM and napping–with substantially less sleep stage 2 and more wakefulness during TM. An independent study we conducted yielded similar EEG differences between TM and napping–except that no sleep stages 3–4 were observed during TM in our sample. Recent investigations based on larger samples also indicate absence of sleep stages 3–4 during TM.

INTRODUCTION

The initial research of Wallace et al. (1) proposed that the Transcendental Meditation (TM) technique produced a unique, "wakeful, hypometabolic state" distinct from ordinary wakefulness and sleep. Since that time, more than 150 research investigations have been published on the TM program (2), and currently more than one million Americans have learned the TM technique (3). Numerous studies have supported Wallace's (1) claims that TM induces a psychophysiological condition of "restful alertness" (4) and that long-term benefits may accrue from its regular practice (5).

The conclusions stated in some studies, however, have not been consistent with the claim that the states or effects induced by TM are "unique" to the practice (6). Most notably, a controversy has arisen with respect to the central claim that TM states are, in fact, distinct from sleep phenomena according to standard EEG criteria (7). Younger et al. (8) observed stage 1 and 2 sleep during TM in their eight subjects. Fenwick et al. (9) suggested that TM states may be best understood as a subset of "stage onset sleep," though statistical analysis according to standard criteria (7) was not reported. The most significant challenge to the Wallace hypothesis came from Pagano et al.'s (10) carefully designed experiment indicating that five experienced meditators spent 19% of their TM sessions in stage 1 sleep and 40% in sleep stages 2–4. The striking feature of their study was its multiple trial, controlling statistical comparison of TM to napping in the same subjects under identical conditions. They hypothesized (10), "If TM produces the wakeful state described by Wallace, one would expect to find less sleep during meditation than during a nap session." Yet they concluded (10), "analysis of variance of time spent in sleep stages 2, 3, or 4 revealed no significant differences between meditation and nap sessions $p > .1$)." Due to variability in states observed, they cautioned against presuming that TM and napping

produce identical distributions of EEG stages. Nevertheless, they ended by questioning whether (10), "beneficial effects reported for meditation are due to the sleep that occurs during meditation or to some other feature of that process."

The Pagano et al. study fueled the TM as sleep controversy and has been one of the more frequently cited of recent physiological investigations on TM (2). Though their experimental design permitted a critical test of the TM as sleep hypothesis, Pagano et al.'s data analyses lacked sufficient statistical power to reveal major differences between TM and napping that are apparent upon more detailed analyses of their data.

ANALYSIS OF SLEEP

Pagano et al. presented several quantitative analyses of the percent time spent asleep. Sleep stages 2–4 were pooled in their analyses because it is customary to identify such stages as unambiguous sleep (11). Apparently, they performed a two-way analysis of variance (ANOVA) with subject and treatment (TM vs. napping) factors on table 1 data (10), averaged across the four separate sessions per subject/treatment combination. If main effects for both factors are determined in such an ANOVA,a test on the treatment main effects ($F_{1,4}=3.2, p<.14$) is consistent with that reported by Pagano et al., (12).

The correct interpretation of this result of no significant difference between TM and napping requires application of statistical power analysis. Consideration of power analysis has been generally lacking in the behavioral (13, p. 16) and biomedical (14) sciences. Power is the probability of detecting an effect in one's sample if it is present in the population from which the sample is drawn. It is equal to the complement of the probability of a Type II error (B): power = 1 − B. A Type II error reflects failure to reject the null hypothesis (i.e., a zero relationship in the population), when the null hypothesis is in fact false. Said another way, power is the probability of *not* making a Type II error.

Power is always dependent upon sample size. Other things being equal, the larger the sample the greater the power to detect such effects. Pagano et al.'s statistical analyses were based on pooling of their data accross EEG stages which reduced their effective sample size to too few observations to detect even very large effects that may exist in their population: i.e., their analyses were predisposed to committing a Type II error.

A measure of effect size for an ANOVA that is independent of sample size is presented by Cohen (13, p. 8). Effect size is a general measure of the degree to which the null hypothesis is false, or how large the effect is in the hypothetical population from which is drawn a particular sample of a given size. The effect size in question concerns the difference in sleep between TM and napping. In the simple case of two groups, one of several equivalent measures of the effect size is r_{pb}, the point biserial correlation (13, p. 23). In the case of more complex ANOVA, regression and covariance analysis, R_p, the multiple partial correlation coefficient, represents a generalization of r_{pb} (15). According to Cohen (13, p. 284), "values of f [or equivalently R_p (15)]

TABLE 1

PERCENTAGE OF TIME SPENT IN EACH EEG STAGE DURING THE TM TECHNIQUE AVERAGED ACROSS SUBJECTS IN DIFFERENT STUDIES

STUDY	N	AGE	YEARS OF TM	TRIALS	TM SESSION (min.)	W	1	2	3, 4
Younger et al. (1975)	8	. . .	3	4	37	59	23	17	0*
Pagano et al. (1976)	5	20–30	≥2.8	4	40	39	19	23	17
Dash and Alexander (1976)	5	19–28	3.8	1	27	80	14	4	0
Hebert and Lehmann (1977)	78	18–62 (mostly 20–30)	4.7	1–5	25	85	10	5	0
Jevning et al. (1978) Sample 1	15	22–29	3–5	1	40	70	22	8	0
Jevning et al. (1978) Sample 2	15	20–27	0.3	1	20	72	19	9	0
Orme-Johnson et al. (1979)	35	18–28	3.7	3	30	78	20	1	0
Warrenburg et al. (1980)	9	$\bar{X}$=30	3.4	2	13–15	77	21	2	0

* Percentages may not sum to 100 in a given study because of rounding error or nonscorable epochs due to movement time or atypical patterns observed.

so large as 0.50 (R_p=.45) are not common in behavioral science, so f=0.40 (R_p=.37) is adopted as a 'large' effect size" (parenthetical statements ours). When we estimate the effect size of Pagano et al.'s ANOVA, it is rather large by such standards (R_p=.49) (16). Supposing this reflected the true effect size in the population, the probability B of accepting the null hypothesis when false would be 0.72 (17). There would be a more than likely probability (72%) of a Type II error even in the presence of such a large effect.

Thus Pagano et al.'s negative result is to be expected and does not provide a statistical basis for concluding that there is a lack of significant difference in amount of sleep during TM and napping. Rather, the null finding reflects experimental design constraints (e.g., having too few subjects) or application of analytical procedures that did not afford sufficient statistical power to detect even large effects.

A more powerful analysis of the effects present within their five subjects is given in the following reanalysis of table 1 (10) data. Concerning parameters of the hypothetical *population* from which the five subjects were drawn, no stronger inferences are possible than those provided by ANOVA (10) because of the large between-subject variability and small size of their sample. However, much stronger inferences can be made concerning the fixed effects present within the five subjects (18).

We perform a two-way ANOVA with repeated trials in order to take advantage of the available replication of four trials for each subject and treatment rather than averaging across trials as did Pagano et al. We analyze a mixed model (18) with both fixed and random effects which includes fixed treatment (TM or nap) main effects, random subject main effects and subject/treatment interactions, and a random error for each trial. The replication within subjects not only permits estimation of the interaction term which allows separation of within subject and between subject variation (19), but also amplifies the power to detect which subjects respond differently to TM and nap sessions. In the original ANOVA (10) these two sources of variation were confounded by the pooling of trials. Pagano et al. did note a substantial amount of between and within subject variation but no quantitative analysis was presented.

For inferences about the five subjects, the two-way ANOVA with repeated observations in each cell reveals a highly significant treatment difference between TM and napping ($F_{1,30}$=11.67, R_p=.48, p<.0018, B=.09), a significant subject/treatment interaction ($F_{4,30}$=3.36, R_p=.52, p<.016, B=.18) and no significant subject main effect ($F_{4,30}$=0.83, R_p=.28, p<.52, B=.78). Thus, significantly less sleep stages 2–4 did take place during TM than napping for these five subjects. Furthermore, there were also significant differences between individual subjects in their differential response to TM or nap sessions.

Since this interaction is quite significant, it is justified to investigate which subjects respond differently to TM and nap sessions. *T*-tests on contrasts in the ANOVA (16, p. 30) corresponding to differences between treatment responses for the same subject indicate significant results for subject 2 (t_{30}=3.66, R_p=.50, p<.001) subjects 3 (t_{30}=2.11, R_p=.32, p<.043) and subject 5 (t_{30}=2.69, R_p=.39, p<.001) (20). Performing *t*-tests on their table 1 (10) data, Pagano et al. had stated that only subject 2 slept significantly less during TM than napping. Contrary to that report, three of the five subjects clearly slept less during TM than napping (20). Both Pagano et al.'s and our *t*-tests involve inferences on fixed effects in the five subjects. Assuming equality of variance across subjects, more sensitive detection of the fixed effects within the individual subjects is permitted by performing contrasts on the ANOVA than by use of separate *t*-tests [Green and Tukey (18)].

Thus within the five subjects studied, large treatment effects (R_p) are present at high levels of statistical confidence (P). However because of the small number of subjects, it is not possible to infer from table 1 (10) data that significant effects are present in the population of long-term meditators. In contrast to the above analysis of unambiguous sleep (stages 2–4), the following analysis of table 2 (10) data reveals highly significant population effects in the difference in EEG profiles between TM and napping.

ANALYSIS OF EEG PROFILES

An in-depth investigation of the differences between TM and napping involves more than the sleep/no-sleep dichotomy, and concerns differences in *distributions* of EEG stages. The pooled stage 2–4 sleep data does not permit study of specific differences in EEG stage distributions. Table 2 (10) did

present percent time spent in each EEG stage (wakefulness, stage 1, 2, 3–4) averaged over four trials for each subject and treatment, but no quantitative analysis of that data (10) was reported.

We perform an ANOVA on table 2 with random effects for subject, fixed effects for EEG stage and treatment main effects, a fixed treatment/stage interaction, and a random error term (21). The difference in the EEG stage distribution between TM and napping is characterized jointly by the treatment main effect and treatment/stage interaction, both of which are zero under the null hypothesis. The pooled sum of squares from treatment main effects and treatment/stage interactions indicates a significant difference in EEG stage distributions between the two treatments ($F_{3,20}=5.27$, $R_p=.59$, $p<.01$, B=.13). T-tests on contrasts of the ANOVA reveal that the difference is due primarily to significantly more wakefulness ($t_{20}=2.58$, $R_p=.43$, $p<.02$) and less sleep stage 2 ($t_{20}=2.89$, $R_p=.46$, $p<.009$) during TM than napping (see fig. 1).

These highly significant differences yielded by statistical analysis of table 2 (10) were, essentially, replicated in an independent study that we conducted. Concurrently with Pagano et al., we had independently implemented a pilot investigation (22) based on a similar TM vs. napping design with subjects serving as their own controls. In most respects, our subjects [table 1 (22)] and experimental conditions were comparable to Pagano et al., though our subjects underwent only one TM and nap session each.

Reanalysis of our table 1 (22) data, following the same ANOVA design employed in the analysis of table 2 (10), also indicates a significant difference in the distributions of EEG stages during TM and napping ($F_{3,20}=5.93$, $R_p=.61$, $p<.005$, B=.09). T-tests for differences in response between treatments for each EEG stage also reveal similar findings to those of Pagano et al., with significantly more wakefulness ($t_{20}=3.50$, $R_p=.54$, $p<.002$) and a trend toward less stage 2 sleep ($t_{20}=1.94$, $R_p=.33$, $p<.07$) occurring during TM than napping (see fig. 2). Most notably, there was a complete absence of sleep stages 3–4 in our TM records [as opposed to 17% (10)]. Four subjects did not enter sleep (stage 2) at all, one exhibited stage 2 in one-fifth of his meditation.

COMPARISON WITH OTHER STUDIES

Considerably less time (by a factor of 10) was spent asleep (stages 2–4) during TM in our study (22) than in Pagano et al.'s. However, both of these studies were based on small sample sizes. As a consequence of Pagano et al., several subsequent meditation studies based on larger sample sizes, directed primarly toward measurement of other variables and not involving a direct comparison to napping, recorded percentage sleep during TM [see table 1 (23)]. Each scored EEG patterns according to standard criteria (7). The studies sampled subjects similar to those of the prior investigations in age, commitment to TM and years of practice [with the exception of Jevning et al., (23)]; the length of experimental TM sessions varied (see table 1). Little unambiguous sleep was observed in these studies, and the overall EEG distributions more closely approximated our (22) EEG profile of TM.

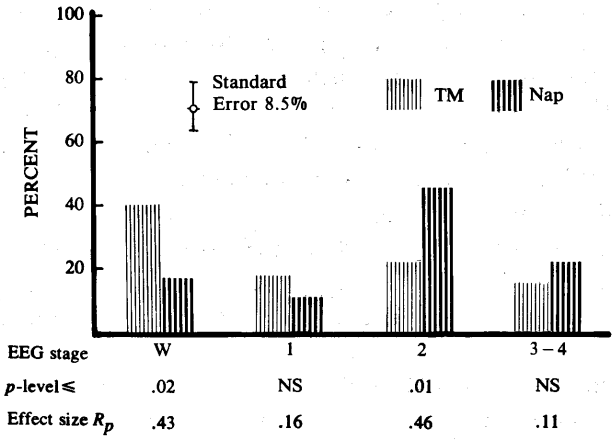

FIG. 1. PERCENT TIME SPENT IN EACH EEG STAGE FOR TM AND NAP BASED ON DATA FROM PAGANO ET AL., (10) AVERAGED ACROSS SUBJECTS.

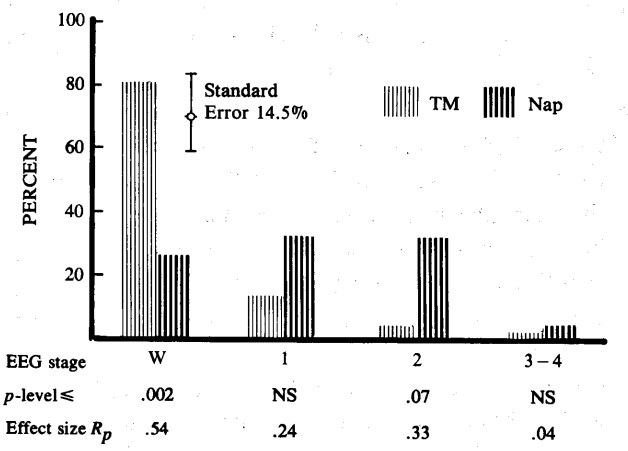

FIG. 2. PERCENT TIME SPENT IN EACH EEG STAGE FOR TM AND NAP BASED ON DATA FROM DASH AND ALEXANDER (22) AVERAGED ACROSS SUBJECTS.

During TM in these studies, clearly most of the time was spent in wakefulness (range: 70–85%); some in stage 1 (10–23%); little in stage 2 (1–9%); and virtually no sleep stages 3–4 was reported. Even if the Younger et al. data is included in this composite profile, the mean percent time spent by Pagano et al.'s subjects in each EEG stage, with the exception of stage 1, falls outside of the corresponding range for that stage in other studies. Our percent times are within the range for each stage. Interestingly, in their own subsequent study, Warrenburg, Pagano et al., (6) (see table 1) did not observe any sleep stages 3–4 during TM and very little stage 2 (2%). However, as they point out, their TM sessions were only 13–15 minutes in length (24).

Though some sleep stage 1 during TM was consistently recorded across all samples, it was certainly too small in amount to warrant support of Fenwick et al.'s hypothesis that TM may be virtually identical to sleep onset phenomena, at least as typically defined (7). However, consistent with Fenwick's (9) observation, in our sample (22) the stage 1 that was typically recorded was "nondescending" in character, as opposed to the descending stage pattern associated with sleep onset during napping or night sleep (25). During our napping control, stage 1 descended to deeper stages of sleep in four of five subjects; whereas during TM subjects tended to maintain stage 1 or momentarily shift into it and then return to a dominant alpha rhythm. In addition to a difference in temporal sequence, it has been suggested that stages may differ in morphology, topographic distribution, reactivity and function during meditation and sleep (26). No REM sleep was observed during TM in any of the above studies. Clearly, as normally practiced, the EEG correlates of TM are distinct from those associated with classical, vivid dream phenomena (27).

The relatively higher percentage of sleep stages 2–4 during TM in Pagano et al. might be explained by several factors. Pagano et al. stated that the absence of sleep in Wallace's data may have been due to obtrusive laboratory measurements (e.g., breathing through a mouthpiece) or first session effects, both of which may have unduly aroused subjects. Laboratory measurements in our study (22), Orme-Johnson et al. and Hebert and Lehmann (23), were rather unobtrusive like those of Pagano et al. Our subjects did undergo only one TM and napping session each. However, if deeper sleep had been inhibited during a first trial, it would presumably occur during the later trials, yet no sleep stages 3–4 was indicated in the four other studies (see table 1) for which multiple trials were available. Even if a first session effect were present, its impact would have been diluted and partially counterbalanced because in our study (22) only three first trials were TM, while two were napping (28).

Due to unspecified differences in the subpopulation studied or the method of subject selection from that subpopulation, in combination with subject variability due to small sample size, Pagano et al. may have initially selected subjects more prone to fall asleep during TM. Nevertheless, even within their sample (10), subjects still rated 6 of their 13 (46%) TM sessions in which stage 2 sleep or deeper took place as atypical. In light of the absence of stage 3–4 sleep in any of the other studies, it would follow that the sessions that did contain the most sleep stages 3–4 would be more likely judged as atypical (10). If such sessions are excluded from analysis, the remaining typical sessions (including 7 with sleep) more closely approximate the EEG profile of the other TM studies.

Particular experimental procedures employed by Pagano et al. may have contributed to the presumed atypically large amount of sleep experienced by some of their subjects. Warrenburg et al., citing Bohlin (29), suggest that the continuous presentation of a white noise masking stimulus during their prior study (the only study in table 1 to follow such a procedure) may have had a "sleep-promoting effect" (6).

Also, experimental delay of TM starting time may have induced more sleep in some of Pagano et al.'s subjects. Subjects who report being tired immediately before starting the TM period are more prone to fall asleep during the practice (30). Individuals are standardly informed of this possibility during TM instructions (30). If in the laboratory, subjects begin their TM sessions at a later time than they would at home, they may experience increased drowsiness prior to and be more likely to fall asleep during TM. Experimental delay of starting time would then result in an overestimation of the amount of sleep during TM under normal circumstances. We controlled for TM starting time by determining when subjects typically began their afternoon meditation at home (range: 4–6:30 P.M.) and arranged for subjects to begin in the laboratory at or before their regular time (3:30–5 P.M.) (22). Pagano et al. did

state that all their sessions were conducted within two hours of the same time each afternoon (presumably ± two hours) but the normal starting time of their subjects at home and degree of fit with starting times in the lab were not reported nor apparently controlled. If some individual sessions deviated in the critical delay direction towards later in the evening than was usual for the subject at home, atypical amounts of sleep may have resulted.

CONCLUSION

Contrary to Pagano et al.'s conclusions, reanalyses of their data and the results of our independent study and those of other investigators suggest that the EEG profile of TM is strikingly different from that of napping—with significantly more wakefulness and appreciably less sleep (stages 2–4) occurring during TM (31). Considering its relatively infrequent appearance during TM, unambiguous sleep is unlikely to account for the supposedly dominant hypometabolic state(s) (32) observed during the practice. Alternative mechanisms to explain the technique's effects should be considered (33). Presumably, some features of the predominantly waking components of TM are primarily responsible for the technique's influence. Clearly, standard EEG sleep criteria were not constructed to discriminate between different nonsleep conditions (34). What would constitute appropriate control groups and sensitive measures for assessing the distinctiveness of TM induced waking states is a complex and important question. It remains for future research to determine the similarities and differences between TM induced substates and those associated with other conditions of wakefulness and relaxation, stylized or unstylized (35).

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12. Because neither degrees of freedom (*df*) nor the exact *p* value were supplied, we cannot be absolutely certain if this was the analysis leading to the result reported.

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15. $f^2 = \sigma^2_H/\sigma^2_e = R^2_p/(1-R^2_p)$ has the interpretation of the proportion of the variance σ^2_H in the dependent or observed variables that is predictable from the tested effects relative to the error variance σ^2_e, given that all other modeled effects are "held constant" or "statistically controlled" (13, pp. 280–284, 365–366, 407–412).

16. Effect sizes were estimated (13, pp. 376–378). Power calculations were done employing the completely general procedure of Scheffe [H. Scheffe, *The analysis of variance*, (New York: Wiley and Sons, 1959), pp. 37–42.] with $\delta^2 = f^2$ N where N is total sample size. Pearson and Hartley charts in Scheffe were then used

which give a more accurate result for complex factorial designs than Cohen (11). We have derived a corrected $f' = f\sqrt{N/(df_e + df_H + 1)}$ where the degrees of freedom are the error (df_e) and numerator (df_H) of the F test. Application of our corrected f' along with Cohen's corrected n' (13, p. 365) to power tables 8.3 (13) will yield the same result as Scheffe. Cohen's procedure overestimates the effect size of the ANOVA (10) as R = .67 whereas Scheffe's procedure gives R = .49.

17. Determination of B depends upon the significance criterion α, which is equal to the probability of Type I error: i.e., the rejection of a null hypothesis when it is true. In our analyses, the significance criterion $\alpha = .05$.

18. Caution is required in making inferences using randomly selected subjects about a model with subject/treatment interactions. A test of the null hypothesis that the treatment main effect is zero in the hypothetical population from which the subjects are drawn requires use of the interaction mean square ($F_{1,4} = 3.2$, $p < .14$) and gives exactly the same result as the ANOVA of Pagano et al. (10). However for inferences about the *particular subjects* comprising the sample, the random session error mean square (within subjects) is used which is 3.6 times smaller with 30 degrees of freedom ($F_{1,30} = 11.67$, $p < .0018$). Our analysis of table 1 (10) will be conditional, applying only to fixed effects within the five subjects: i.e., the subject main effects and subject/treatment interactions are considered to pertain only to the five subjects. For a discussion of inference on random effects models see B. F. Green and J. W. Tukey, *Psychometrika* 25 (1960): 127.

19. The standard deviation between subjects within treatments was 25.9% of the session and nearly as large as that within subjects which was 27.2%. This confirms the reports of substantial variation between subjects and within a subject from session to session [10; J. Taty, Ph. Brenot, and J. M. A. Faure, (1977). *Psychologia Medicale* 9:1]. Nevertheless, averaged over many sessions a particular subject may display a consistent and often distinctive pattern, as suggested by the significant subject/treatment interaction in our ANOVA of table 1 (10) and longitudinal observation of novice and advanced TM practitioners for over a year [J. A. Wadda, and A. E. Hamm. Paper presented at the 27th annual meeting, American EEG Society, Boston, Massachusetts. June 15–16, 1973; J. A. Wadda: Personal communication, December 1980].

20. We initially performed several t-test analyses to attempt to duplicate Pagano et al.'s result (i.e., subject two: $t = 7.3$, $p < .01$). The closest was a test for a difference between means of the two treatment populations assuming the same variance for each. This approach yielded a significant result not only for subject two ($t_6 = 7.34$, $p < .0003$), but also for subject five ($t_6 = 2.83$, $p < .03$), which Pagano et al. had not reported (10).

21. Prudence is recommended in applying ANOVA methods to frequency data involving exhaustive classification. In this case, percentages always sum to 100 across EEG stages for each subject. To allow for this deterministic constraint, only stages W, 1, and 2 were included in the ANOVA with stage wakefulness expressed deterministically as essentially 100% minus the sum of the other stages. Such an approach decreases the significance level estimates and produces a more conservative result. An ANOVA was first performed with the additional interactions subject/treatment and subject/EEG stage included. These interactions were small compared with the random error term. As discussed in Green and Tukey (18), these small interactions are treated as if they are zero and the corresponding sum of squares pooled with the random error giving 20 degrees of freedom.

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23. HEBERT, R., and LEHMANN, D. 1977. *Electroencephalography and Clinical Neurophysiology* 42: 397; values for sleep stages 2–4 in table 1 (R. Hebert: Personal communication, December 1980). JEVNING, R.; WILSON, A. F.; and DAVIDSON, J. M. 1978. *Hormones and Behavior* 10: 54; less than 1% stage 3 was indicated (R. Jevning: Personal Communication, December 1980). ORME-JOHNSON, D. W.; WALLACE, R. K.; DILLBECK, M. C.; LUKENBACH, E. E.; and ROSENBERG, W. A. 1979. Paper presented at the 132nd annual meeting, American Psychiatric Association, Chicago, Illinois, 17 May, 1979; average percent time spent in wakefulness /sleep in table 1 based also on a 3rd trial conducted within a few days of trial 2, 35 subjects completed all 3 trials (D. W. Orme-Johnson: Personal Communication, December 1980).

24. Such sessions may have been too short to permit the onset of sleep stages 3–4 as typically observed during an afternoon nap [I. Karacon, W. W. Finley, R. L. Williams, and C. J. Hursch. *Biological Psychiatry* 2 (1970): 261] or night sleep onset [H. W. Agnew, W. B. Webb, and R. L. Williams. *Psychophysiology* 2 (1966): 263]. However, according to Pagano et al. (10), shorter sessions should not have precluded the appearance of stage 2 sleep. They earlier established that within the first 20 minutes of the TM sessions, their subjects averaged slightly more time (42.5%) in sleep stages 2–4 (presumably, predominantly stage 2) than during the remaining 20 minutes of their TM sessions (10). Also, though a longer session may be a necessary condition for deeper stages of sleep (3–4),

it certainly is not a sufficient one (for example, see Jevning et al., table 1).

25. VOGEL, G.; FOULKES, D.; and TROSMAN, H. 1966. *Archives of General Psychiatry* 12: 238.

26. ELSON, B. D.; HURI, P.; and CUNIS, D. 1977. *Psychophysiology* 14: 52; ORME-JOHNSON, D. W., et al. (23).

27. DEMENT, W. C., and KLEITMAN, N. 1957. *Electroencephalography and Clinical Neurophysiology* 673; DEMENT, W. C. 1972. *Some must watch while some must sleep*, p. 116. San Francisco: Freeman.

28. WARRENBURG et al. (6) did report significant first-day effect on EEG stage 2 pooling across their groups [Long-term TM (LTM) vs. Long-term Progressive Relaxation (LPR)] and conditions (eyes closed control vs. treatment), but examination of group means indicated that it was LPR who entered sleep stage 2 substantially more during the second treatment session ($\bar{D} = +24.5\%$) and not LTM ($\bar{D} = -1.4\%$). Also, the marginally significant decrease in wakefulness indicated during the second day's sessions (6), appears to be due more to a decrease in stage W during LPR ($\bar{D} = -28.2\%$) than LTM ($\bar{D} = -12.9\%$).

29. BOHLIN, G. 1971. *Electroencephalography and Clinical Neurophysiology* 31: 593.

30. WALLACE, R. K. 1976. *Science* 193: 719. WACHSMUTH, D., and DOLCE, G. 1980. Rechnerunterstützte Analyse des EEG während Transzendentaler Meditation und Schlaf. *EEG and EMG* 11: 183–188.

31. Conceivably, the EEG distribution of napping in an upright position is closer to that of TM. However, there is apparently a tendency toward spontaneous occurrence of TM-like states when long-term meditators sit upright with eyes closed outside their practice [Banquet (4); Jevning et al. (23)]. Also, repeated elicitation of napping over longer periods might possibly alter the EEG to take on TM-like characteristics. But a study of "regular" non-TM nappers under comparable conditions yielded an EEG profile similar to nap, not TM [J. Taub; P. E. Tanguay; and D. Clarkson. *Journal of Abnormal Psychology* 85 (1976): 210].

32. A controversy still remains over the degree of metabolic related changes that occur during TM relative to basal values on those produced by other procedures [for example, Fenwick et al. (9); Warrenburg et al. (6)].

33. For example, BANQUET, J.-P. (4); GLUECK, B. C., and STROEBEL, C. F. (5); JEVNING, R. and O'HALLORAN, J. 1979. *Journal of Chronic Diseases and Therapeutic Research* 13: 206.

34. Small amounts of sleep have also been observed during unstylized relaxation [R. Jevning et al. (23)], another form of meditation [Elson et al. (26); J. C. Corby, W. T. Roth, V. P. Zarcone, and B. S. Kopell. *Archives of General Psychiatrics* 35 (1978): 571]; and in novice practitioners of stylized relaxation [S. Warrenburg et al. (6)].

35. Efforts in that direction have begun, especially with respect to distinguishing TM from unstylized relaxation. For example, sensitive measures of regional distribution of blood flow [R. Jevning et al. (4); H. C. Pirkle, and A. F. Wilson, *The Physiologist* 21 (1978): 60]; high amplitude EEG "theta bursts" [R. Hebert and D. Lehmann (23)]; and stability of phase relationship ("coherence") between topographically distinct EEG signals [P. H. Levine. Electrophysiological and EEG Changes: EEG Coherence. *Proceedings of the San Diego Biomedical Symposium* 15 (1976): 237; D. W. Orme-Johnson. *Electroencephalography and Clinical Neurophysiology* 43 (1977): 581] have discriminated between TM and relaxation with eyes closed in sitting non-meditator controls. Phase stability of EEG among frontal and central derivations was also higher during waking periods of TM than waking with eyes closed in the same subjects [Levine (1976); D. W. Orme-Johnson and C. T. Haynes, *International Journal of Neuroscience* 12 (1981)], and significant short-term longitudinal enhancement of frontal alpha coherence was demonstrated during TM but not during relaxation control sessions in the same subjects [M. C. Dillbeck and E. C. Bronson, *International Journal of Neuroscience* 13 (1981)]. Preliminary biochemical investigations have yielded seemingly contradictory results but assaying methods, etc., differed between studies [Michaels et al. (6); Jevning et al. (23); Jevning and Wilson (4)].

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