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The Effect of Afternoon Body Heating on Body Temperature and Slow Wave Sleep

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ABSTRACT

Recent evidence suggests that body temperature at sleep onset affects the subsequent level of slow wave sleep. According to one hypothesis, the actual temperature is the critical factor determining the relationship. An alternative proposal is that it is the rate of fall of body temperature following sleep onset. These hypotheses were tested by measuring rectal temperature and sleep, following late afternoon passive heating in a warm bath and during a control condition. Passive heating increased rectal temperature, which then returned rapidly toward the control level. However, immediately before lights out rectal temperature was still higher in the passive heating condition, a difference that continued throughout the night. Following passive heating the amount of slow wave sleep was higher in the early part of the night. These results support the hypothesis that body temperature at sleep onset and the amount of slow wave sleep are positively related.

DESCRIPTORS: Sleep, Slow wave sleep, Rectal temperature, Body temperature, Metabolic rate, Passive heating.

There has been considerable interest in recent years in the relationship between body temperature (T_b) and sleep. It has been shown that there is a reduction in T_b over the first 1–2 hours of sleep (Day, 1941; Geschichter, Andrews, & Bullard, 1966; Gillberg & Akerstedt, 1982), an effect that is largely independent of the circadian phase of sleep onset (Gillberg & Akerstedt, 1982). The fall in T_b is thought to be due to a regulated reduction in the hypothalamic set point for thermoregulatory control (Parmeggiani, 1980, 1987). Body temperature also affects sleep. In particular, sleep duration and the distribution of REM sleep are influenced by the circadian temperature rhythm (Czeisler, Weitzman, Moore-Ede, Zimmerman, & Knauer, 1980; Gillberg & Akerstedt, 1982; Zulley, Wever, & Aschoff, 1981). It has also been suggested that T_b affects the amount of slow wave sleep.

There have been two hypotheses regarding the relationship between T_b and the level of slow wave sleep. One proposes that the regulated reduction in T_b at sleep onset is a necessary prerequisite for the occurrence of slow wave sleep. An explicit corollary of this hypothesis is that the amount of slow wave sleep on any particular night will be a positive function of the rate of fall of T_b following sleep onset (Sewitch, 1987). The other hypothesis is that the amount of slow wave sleep is a positive function of T_b at and shortly after sleep onset (Berger, Palca, Walker, & Phillips, 1988; Berger & Phillips, 1988).

The observation that passive heating during wakefulness increases the amount of slow wave sleep in the subsequent sleep period (Bunnell, Agnew, Horvath, Jopson, & Wills, 1988; Bunnell & Horvath, 1985; Horne & Moore, 1985; Horne & Reid, 1985; Horne & Shackell, 1987; Horne & Staff, 1983) is quoted by both authors as evidence for their respective positions (Berger & Phillips, 1988; Sewitch, 1987). However, in view of their different hypotheses with respect to the relationship between T_b and slow wave sleep, the two theories necessarily predict a different relationship between passive heating and T_b. Thus, Sewitch (1987) argues that passive heating results in a faster decline in T_b following sleep onset and a lower minimum temperature, as compared to a control condition. In contrast, Berger and Phillips (1988) require that passive heating produce elevated T_bs during the early sleep period.

The issue is unresolved because, with one exception, studies investigating the passive heating

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effect have not measured T_b beyond the passive heating period. Horne and Staff (1983) measured the effect of passive heating on T_b during the subsequent sleep period in a single subject with one night's recording for each condition. They found a compensatory fall during sleep, as predicted by Sewitch (1987). However, Berger and Phillips (1988) have reported a positive relationship between both rectal (T_{re}) and tympanic (T_{ty}) temperature and the amount of slow wave sleep (Berger et al., 1988) and an increase in slow wave sleep as a result of increasing T_{ty} by facial heating (Berger & Phillips, 1988).

The aim of the present study was to examine these two hypotheses by measuring the effect of passive heating on T_{re} and slow wave sleep during the subsequent sleep period.

Method

Subjects and Design

Five male subjects with a mean age of 20.4 years (SD = 1.52) participated in the study. All were healthy at the time of testing, did not report a history of respiratory or sleep disorders, were non-smokers, and were not on medication or other drug use. The project was approved by the University of Tasmania, Human Ethics Committee, and all subjects completed a consent form.

The design of the study included two conditions. In the first, subjects were exposed to passive heating (PH) in the late afternoon, whereas the second was a non-bath control condition (CC). A temperate bath control procedure was not considered necessary because differences between this and a non-bath control have not been found (Bunnell & Horvath, 1985). Each subject was administered each of the two conditions on three occasions, the sequence being CC, PH, PH, CC, CC, PH for 3 subjects, and PH, CC, CC, PH, PH, CC for 2 subjects. The experimental strategy of running a relatively small number of subjects on a number of nights in each condition (rather than a larger number of subjects on a single night each) was selected in view of the importance of having subjects well adapted to the unusual sleeping circumstances. From a statistical perspective, the strategy was designed to reduce the within-subject error variance. Thus, in the statistical analysis, data were averaged over within-subject replications.

In order to facilitate subjects' familiarization with the equipment, the 6 experimental sessions were preceded by 2 adaptation nights and a number of afternoon adaptation sessions, during which the subjects accustomed themselves to wearing the various pieces of equipment, particularly a face mask. The experimental sessions were on non-consecutive days.

Procedures

Subjects reported to the laboratory at 1330 hrs. Rectal temperature recordings began at 1400 hrs and, with the exception of the time the subjects spent in the bath in the passive heating condition and two other brief periods in both conditions, continued until the end of the sleep period at approximately 0700 hrs the next morning. Passive heating was administered in two 40-min blocks with a 25-min inter-block interval beginning at 1715 hrs and ending at 1900 hrs. In addition to T_{re}, O_2 consumption was measured at various times during the afternoon and evening. In the passive heating condition measurements were taken for 15 min before the passive heating, 20 min between the two heating sessions, 20 min following the second heating session, and 30 min before lights out. Oxygen consumption measurements were then continued throughout the night. In the control condition measurements were taken during the 30 min before sleep onset and then throughout the sleep period. Some O_2 consumption measurement periods were less than the specified duration because of the need to maintain a strict time schedule throughout the sessions.

In addition to the periods in the bath, rectal temperature recordings were interrupted on two other occasions. The first was during the evening meal when some subjects left the laboratory. The second was immediately before the pre-sleep O_2 consumption measurements and was necessary to begin a new data acquisition file incorporating the O_2 consumption measurements. Also on some nights the probe was temporarily removed at this time. Thirty minutes of data were lost in association with the evening meal and 15 min before the pre-sleep O_2 consumption measurement. Sleep was assessed during the night in each condition. Subjects ate their evening meal between 1530 and 1630 hrs, and lights out was at 2300 hrs. The timetable of events for the laboratory sessions is shown in Table 1.

During the afternoon and evening, subjects were confined to the laboratory and were free to engage in a range of sedentary activities. Tea and coffee were not permitted and no food or drink other than water could be consumed following the end of the evening meal at 1630 hrs. Caloric intake at the evening meal was held constant at a level of each subject's choosing.

Table 1

Diary of events during each experimental session

Time		
Start	**Finish**	**Event**
1330		Arrive at laboratory
1400		Begin T_b recording
1530	1630	Evening meal
1645[a]	1700	O_2 consumption recording
1715[a]	1755	Passive heating 1
1800[a]	1820	O_2 consumption recording
1820[a]	1900	Passive heating 2
1910[a]	1930	O_2 consumption recording
2230	2300	O_2 consumption recording
2300	0700	Sleep period

Note.—The unaccounted for time was spent in sedentary activities within the laboratory.

[a]Passive heating sessions only.

The passive heating procedures were essentially the same as those developed by Horne and Staff (1983). Subjects sat on a chair in the bath with the water level at neck height. Water temperature was maintained between 42°C and 43°C by a thermostatically controlled valve in the attached hot water system. Subjects remained in the bath for two 40-min sessions. Although they were told that they could leave the bath at any sign of ill effects, all subjects adapted well to the procedures and this precaution was not utilized. The average elevation in T_{re} from the last 15 min before heating to the first minute following the second passive heating session was 1.64°C (SD=.36°C).

Rectal temperature was measured using a Yellow Springs, YSI series, 401 probe. The system was calibrated against known resistances and temperatures to an accuracy of .01°C at the beginning of each recording session. The probe was attached directly to the data recording system via a cable long enough to give the subject the freedom to move around the laboratory. The probe was inserted 10 cm into the rectum and maintained in place by surgical tape. The temperature signal was fed into a PDP 11/23 computer via an A/D board and sampled at 1-min intervals.

During the pre-sleep sessions O_2 consumption was measured with the subject lying on, or in, a bed in the supine position with eyes open and the room light on. Subjects were also instructed to remain in the supine position during the night. This was achievable because the mask made it difficult to sleep in any other position. The laboratory was maintained within a temperature range of 23–25°C and subjects could adjust their clothing or bedcovers to maintain thermal comfort. During the awake test an EEG recording was monitored to ensure that the subject did not enter stage 1 sleep, which is associated with a marked reduction in O_2 consumption (Fraser, Trinder, Colrain, & Montgomery, 1989). Expired air was collected by a face mask attached to a two-way breathing valve (Hans Rudolf #2600). The valve dead space was 53 cc with the total dead space of the mask and valve varying from 127–135 cc, depending on facial configurations. Ventilation was calculated by the computed integration of air flow as measured by a Fleisch Pneumotachograph and Gould PM 15E Pressure Transducer. The O_2 level in the expired air was measured by an Applied Electrochemistry S-3A Oxygen Analyzer. Signals were fed via a Beckman R411 Recorder to the computer for a breath-by-breath computation of O_2 consumption. Greater details of the laboratory procedures for measuring O_2 consumption are given in Colrain, Trinder, Fraser, and Wilson (1988). Due to technical problems, the O_2 consumption data for one subject were incomplete and the subject was dropped from the analysis of this variable.

In order to assess sleep, EEG (C_3/A_2), EMG, and EOG recordings were collected and scored according to standardized procedures (Rechtschaffen & Kales, 1968). Each night's record was scored blind by two scorers and disagreements were resolved by discussion (the initial level of agreement was 91% of all epochs). As noted above, lights out was at 2300 hrs and recording was ended at 0700 hrs.

Results

The data was first averaged over replications within conditions and subjects. The resulting values were used in all subsequent analyses, tables, and figures. Replications were not formally analyzed because their inclusion was designed to reduce error due to within-subject variability, rather than to be of specific interest. Further, preliminary data analyses had indicated that, although within a subject there was an expected level of variability in both initial temperature and sleep quality, from replication to replication, the experimental manipulation had highly consistent effects over replications on both variables.

The effect of passive heating on T_{re} is illustrated in Figure 1. As can be seen, passive heating caused a substantial elevation in T_{re}, an effect that persisted throughout the sleep period. In addition to the effect of passive heating, there was a reduction in T_{re} associated with sleep. The fall was first apparent following the non-recording period associated with the setting up of new data files and continued during the first 1–1½ hrs of sleep. The marked fall observed following the non-recording period was most likely associated with the attainment of a lateral position (Kleitman, 1963). There was no perceptible change in the rate of fall of T_{re} immediately following sleep onset. The rates for the intervals immediately before lights out, between lights out and sleep onset, and sleep onset to the onset of slow

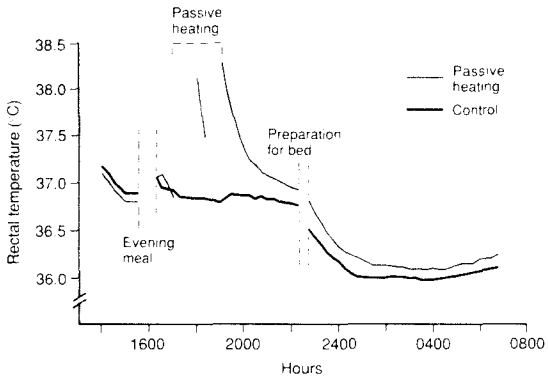

Figure 1. Rectal temperature as a function of experimental condition over the recording period. Data points are mean values averaged over 15 min, the 3 replications within each condition, and the 5 subjects. Temperatures were not available from all subjects during the evening meal and thus data from 1545–1615 hrs are not presented. Data collection was also discontinued during passive heating and for a 15-min interval before the evening O_2 consumption measurement. Lights out was at 2300 hrs.

wave sleep were .0090, .0069, .0060 and .0103, .0078, .0069°C/min for the control and passive heating conditions respectively. Tests of significance showed that the rate of fall over the interval, sleep onset to the onset of slow wave sleep, did not differ from either of the other two intervals (the t values for the before lights out comparison were $t(4)=1.58$, $p>.05$, and $t(4)=1.48$, $p>.05$, for the control and passive heating respectively, and for the lights out to sleep onset comparison were $t(4)=0.43$, $p>.05$, and $t(4)=0.82$, $p>.05$, for the control and passive heating respectively).

The mean temperature values for critical periods over the experimental sessions are shown in Table 2. These periods were the 15 min before passive heating, the 15 min following passive heating, the 5 min before lights out and following sleep onset, and minutes 61–65 following the onset of slow wave sleep. The analysis of these values was carried out using three separate ANOVAs. The first variable in each of these analyses was the experimental condition (passive heating or control). The other variable was time, the levels of which were: before versus after passive heating in the first analysis; after passive heating versus before lights out in the second; and before lights out, following sleep onset, before slow wave sleep (SWS), and 60 min after SWS onset in the third. Significance levels for the effect of time were assessed using $1/n-1$ degrees of freedom, the most conservative approach to dealing with the problem of sphericity.

The first analysis indicated a significant increase in T_{re} as a result of passive heating ($F(1/4)=96.96$, $p<.01$, for the Condition × Time interaction for the periods before and after passive heating); the second, a significantly greater reduction in T_{re} in the passive heating condition during the 4 hrs following passive heating ($F(1/4)=49.28$, $p<.01$, for the Condition × Time interaction for the periods after passive heating and before lights out); and the third, a significant fall in T_{re} in association with sleep ($F(1/4)=96.42$, $p<.01$, for the main effect of time from lights out to 60 min following SWS onset), and a significantly higher T_{re} in the passive heating condition ($F(1/4)=62.49$, $p<.01$, for the main effect of heating). The Condition × Time interaction for the period from before lights out to 60 min following the onset of slow wave sleep was not significant ($F(1/4)=1.50$, $p>.05$).

Table 2

Mean rectal temperatures for selected time intervals during the two experimental conditions

Time	Mean Temperatures (°C) (SDs in Parentheses)		
	Passive Heat	Control	
1645–1700 before heating	36.86 (.11)	36.91 (.09)	C, T, C×T
1845–1900 after heating	38.28 (.35)	36.81 (.09)	C, T, C×T
5 min before lights out	36.76 (.14)	36.57 (.14)	
5 min after sleep onset	36.63 (.12)	36.45 (.13)	
5 min before SWS	36.58 (.13)	36.39 (.15)	C, T, C×T
5 min after 60 min of SWS	36.28 (.10)	36.14 (.17)	

Note.—The statistical analyses are described in the text. Significant comparisons ($p<.01$) are shown on the right of the table. C=Conditions, T=Time, C×T=Interaction.

In addition to the changes in temperature that were of interest to the hypotheses under consideration, there was a small and brief increase in T_{re} following the evening meal. This increase was most likely due to a thermogenic effect of the meal, although because subjects had to leave the laboratory area to obtain their meal, there was also a small increase in activity at this time.

Passive heating was not associated with a significant increase in O_2 consumption. Of the 4 subjects for whom O_2 consumption data were available, 2 increased and 2 decreased O_2 consumption levels following passive heating. The mean change was an increase of 14ml/min, from 311ml/min to 325ml/min, which was not a significant difference ($t(3)=1.50$, $p>.05$). Further, there was no difference in O_2 consumption between the two conditions during measurements before lights out (255ml/min and 261ml/min for the control and passive heating conditions, respectively), or for the 15 min immediately following stage 1 sleep onset (227ml/min and 227ml/min). An analysis using a 2×2 ANOVA with repeated measures on both variables indicated that although neither the main effect of condition ($F(1/3)=0.09$, $p>.05$), nor the interaction ($F(1/3)=0.55$, $p>.05$) were significant, the effect of sleep onset was significant ($F(1/3)=138.34$, $p<.01$).

Inspection of Table 3 indicates that, despite considerable adaptation to the laboratory, the subjects' sleep was disturbed by the experimental conditions. The amount of disturbed sleep was higher and SWS and REM sleep lower than is usually found in this laboratory (e.g., Trinder, Paxton, Montgomery, & Fraser, 1985). However, the levels were consistent with studies, from this and other laboratories, in which respiratory variables were measured (e.g., White, Weil, & Zwillich, 1985).

Passive heating resulted in an increase in slow wave sleep during the early part of the night. Thus, slow wave sleep was significantly higher following passive heating in the first 150 min of sleep ($t(4)=3.42$, $p<.05$). (The first 150 min of sleep is the interval used in this laboratory to assess early night

Table 3

Mean time for selected sleep variables in the passive heating and control conditions

Sleep Variables	Mean Times (min) (SDs in Parentheses)	
	Passive Heat	Control
SWS (150 min)	43 (12.9)	35 (10.9)*
Stage 3	17 (2.7)	15 (5.5)
Stage 4	26 (12.1)	20 (13.4)
SWS (total night)	65 (25.5)	57 (21.5)
Stage 3	30 (9.9)	27 (12.1)
Stage 4	35 (19.1)	30 (22.7)
REM (150 min)	14 (8.4)	8 (8.1)*
REM (total night)	86 (15.4)	79 (19.6)
REM Latency[a]	101 (27.8)	154 (99.6)
Total sleep time	446 (24.3)	441 (29.6)
Stage 2	256 (12.0)	250 (19.8)
Sleep Latency[b]	10 (4.1)	10 (4.5)
Disturbed sleep (1+Wake)	73 (33.3)	95 (39.4)*
Stage 1	40 (13.9)	53 (23.4)
Time Awake	33 (21.5)	42 (25.1)
Sleep efficiency[c]	.93 (.04)	.91 (.06)

[a]Time from the first stage 2 to the first REM epoch.
[b]Time from lights out to the first stage 2 epoch.
[c]Ratio of total sleep time to time in bed.
*Conditions significantly different at $p<.05$.

effects.) The increase in slow wave sleep was largely the consequence of a change in stage 4 ($t(4)=2.74$, $.05<p<.1$) with a smaller increase in stage 3 ($t(4)=1.26$, $p>.05$). The magnitude of the effect on slow wave sleep was small, though consistent over subjects. Although the amounts of slow wave sleep and of stages 3 and 4 for the whole night were each higher following passive heating, the differences were not significant ($t(4)=1.54$, $p>.05$; $t(4)=0.71$, $p>.05$; $t(4)=1.26$, $p>.05$, for slow wave sleep and stages 3 and 4, respectively).

REM sleep was higher during the first 150 min of sleep following passive heating ($t(4)=3.36$, $p<.05$). Also REM latency was reduced and total REM increased, though neither significantly ($t(4)=1.53$, $p>.05$; $t(4)=1.29$, $p>.05$, for latency and total amount, respectively). The pattern of results for REM sleep indicates that the changes in slow wave sleep in the first 150 min were not due to a suppression of REM sleep. On the contrary, both REM and slow wave sleep increased in this period.

Sleep duration and sleep onset latency were unaffected by the experimental manipulation ($t(4)=0.80$, $p>.05$; $t(4)=1.05$, $p>.05$, respectively). However, there was some evidence that sleep was less disturbed following passive heating. The amount of disturbed sleep (stage 1, movement time, and time awake) was reduced ($t(4)=3.07$, $p<.05$) and sleep efficiency was increased, although the latter difference was not significant ($t(4)=1.16$, $p>.05$).

Discussion

The effect of passive heating on slow wave sleep was similar, though smaller in magnitude, to that which has previously been reported (e.g., Horne & Staff, 1983). The effect was observed despite the sleep disturbance usually associated with wearing a face mask and sleeping supine. However, it might be argued that the difference in slow wave sleep between the two conditions was an artifact of the recording environment, perhaps because of a suppression of the control values. We consider this unlikely, because there appears to be no reason why the recording situation would have differentially disadvantaged either of the two conditions and because the sleep values obtained are consistent with other studies in which the subjects were restricted by respiratory apparatus. If anything, the sleep disturbance associated with the recording conditions might be expected to minimize the effect of the independent variable. Thus, the present results testify to the robustness of the effect. Most critically, the observation of an increase in slow wave sleep allowed the temperature conditions that produced the effect to be assessed.

The temperature data clearly support the relationship between T_b and slow wave sleep proposed by Berger (Berger et al., 1988; Berger & Phillips, 1988). Rectal temperature was significantly elevated at sleep onset and at the beginning of slow wave sleep in the passive heating condition. Indeed, after an initial fall following the termination of passive heating, T_{re} remained approximately 0.19°C above the control condition. There was no evidence to support Sewitch's (1987) hypothesis that the elevation of slow wave sleep following passive heating is due to a faster rate of decline in T_b after sleep onset, because the rates of decline were essentially identical in the two conditions. Nor, as has been described above, was there any indication of a compensatory fall in T_b.

However, the present data is not entirely consistent with results from Berger's laboratory. Berger et al. (1988) reported that the rate of fall in both T_{re} and T_{ty} to the end of the last SWS period, was a positive function of the temperature at sleep onset, whereas in this study these two variables were unrelated. It is likely that this discrepancy is a consequence of procedural differences between the two studies. Subjects in the Berger et al. (1988) experiment were exposed to thermoneutral or cold ambient temperatures during sleep, whereas in this study subjects were exposed to thermoneutral or hot temperatures before sleep, and thermoneutral conditions during sleep. Despite this discrepancy, neither study reported a relationship between the rate of fall in temperature and the amount of slow wave sleep. Thus, the studies are consistent in

showing that T_b is the critical factor determining slow wave sleep.

It is of interest to speculate as to what factors may have sustained the elevation in T_b throughout the sleep period. One possibility is that it was due to a difference in heat production. However, this hypothesis is contradicted by the observation that O_2 consumption values were not different immediately before or after sleep onset. Nor is it likely that the effect was due to limitations in heat exchange, because heat loss appeared to proceed normally following the evening meal, immediately following the passive heating, and during sleep. We have previously speculated that passive heating may produce a delayed shift in the body temperature rhythm (Trinder, Montgomery, & Paxton, 1988). However, the data are not consistent with this hypothesis, because T_{re} remained elevated throughout the night. It remains possible that the passive heating produced a shift in body temperature set point, though the evidence is more that of exclusion, rather than being direct.

The pattern of fall in T_b during sleep was consistent with that described previously (Gillberg & Akerstedt, 1982). Rectal temperature fell approximately 0.75°C, and reached an asymptote 1–1½ hours after sleep onset in both conditions. Rectal temperature began to fall before lights out, during the measurement of resting O_2 consumption. A similar effect has been reported in rats by Obal (1984). Further, the rate of fall was not affected by sleep onset. This contrasts with other reports that have linked the rapid fall in T_b during the early sleep period with sleep onset (Gillberg & Akerstedt, 1982; Parmeggiani, 1980, 1987). The most likely explanation of the present data is that the effect of sleep onset has been masked by what appears to be a relatively large effect associated with the change in body position on going to bed (Kleitman, 1963). Parmeggiani (1980) has also noted that body position may contribute to the fall in cats. However, the contribution of body position to the fall in T_b at sleep onset in human subjects remains uncertain.

Passive heating did not have a consistent effect on O_2 consumption. In 2 subjects heating was associated with moderate increases, but in 2 others with slight decreases. In all subjects O_2 consumption was the same for the two conditions by lights out, despite differences in T_b. The failure to find an increase in O_2 in association with an increase in core temperature was unexpected. It is unlikely to be due to insensitivity in the recording equipment because other changes, such as the fall associated with sleep onset (Fraser et al., 1989), were identified. A more likely explanation is that the effect of the bath was masked by a thermogenic effect associated with the evening meal. In this context, the failure to measure O_2 consumption in the control condition at this time can be seen as an error.

REM sleep was significantly higher during the first 150 min of sleep in the passive heating condition. The effect was due largely to a tendency for subjects to miss the first REM period in the control condition. Bunnell et al. (1988) have reported that REM was suppressed by late evening, but not morning, afternoon, or early evening heating, whereas Horne and Reid (1984) found that afternoon heating reduced REM sleep. Other studies have reported REM sleep to be unaffected by passive heating (Bunnell & Horvath, 1985; Horne & Staff, 1983). In general, the data indicate that passive heating does not have a consistent effect on REM sleep. Further, the present results show that the facilitative effects of passive heating on slow wave sleep do not depend on a reduction in REM sleep.

The results of this study, in agreement with other recent studies, show that T_b influences the SWS component of sleep. The nature of the interaction is consistent with the hypothesis that the amount of slow wave sleep is a positive function of T_b at sleep onset (Berger et al., 1988; Berger & Phillips, 1988), but not with the hypothesis that it is a function of the rate of fall of T_b following sleep onset (Sewitch, 1987).

REFERENCES

Berger, R.J., Palca, J.W., Walker, J.M., & Phillips, N.H. (1988). Correlations between body temperatures, metabolic rate and slow wave sleep in humans. *Neuroscience Letters, 86*, 230–234.

Berger, R.J., & Phillips, N.H. (1988). Comparative aspects of energy metabolism, body temperature and sleep. *Acta Physiologica Scandinavica, 133*(Suppl. 574), 21–27.

Bunnell, D.E., Agnew, J.A., Horvath, S.M., Jopson, L., & Wills, M. (1988). Passive body heating and sleep: Influence of proximity to sleep. *Sleep, 11*, 210–219.

Bunnell, D.E., & Horvath, S.M. (1985). Effects of body heating during sleep interruption. *Sleep, 8*, 274–282.

Colrain, I.M., Trinder, J., Fraser, G., & Wilson, G.V. (1987). Ventilation during sleep onset. *Journal of Applied Physiology, 63*, 2067–2074.

Czeisler, C.A., Weitzman, E.D., Moore-Ede, M.C., Zimmerman, J.C., & Knauer, R.S. (1980). Human sleep: Its duration and organization depend on its circadian phase. *Science, 210*, 1264–1267.

Day, R. (1941). Regulation of body temperature during sleep. *American Journal of Diseases of Children, 61*, 734–746.

Fraser, G., Trinder, J., Colrain, I.M., & Montgomery, I.

(1989). The effect of sleep and circadian cycle on sleep period energy expenditure. *Journal of Applied Physiology, 66*, 830–836.

Geschichter, E.H., Andrews, P.A., & Bullard, R.W. (1966). Nocturnal body temperature regulation in man: A rationale for sweating in sleep. *Journal of Applied Physiology, 21*, 623–630.

Gillberg, M., & Akerstedt, T. (1982). Body temperature and sleep at different times of day. *Sleep, 5*, 378–388.

Horne, J.A., & Moore, V.J. (1985). Sleep EEG effects of exercise with and without additional body cooling. *Electroencephalography & Clinical Neurophysiology, 60*, 33–38.

Horne, J.A., & Reid, A.J. (1985). Night-time sleep EEG changes following body heating in a warm bath. *Electroencephalography & Clinical Neurophysiology, 60*, 154–157.

Horne, J.A., & Shackell, B.S. (1987). Slow wave sleep elevations after body heating: Proximity to sleep and the effects of aspirin. *Sleep, 10*, 383–392.

Horne, J.A., & Staff, L.H.E. (1983). Exercise and sleep: Body heating effects. *Sleep, 6*, 36–46.

Kleitman, N. (1963). *Sleep and wakefulness*. Chicago: University of Chicago Press.

Obal, F. (1984). Thermoregulation and sleep. In A. Borbely & J.L. Valatx (Eds.), *Sleep mechanisms* (pp. 167–172). Berlin: Springer-Verlag.

Parmeggiani, P.L. (1980). Temperature regulation during sleep: A study in homeostasis. In J. Orem & C.D. Barnes (Eds.), *Physiology in sleep: Research topics in physiology* (pp. 97–143). New York: Academic Press.

Parmeggiani, P.L. (1987). Interaction between sleep and thermoregulation: An aspect of the control of behavioral states. *Sleep, 10*, 426–435.

Rechtschaffen, A., & Kales, A. (1968). *A manual of standardized terminology, techniques and scoring system for sleep stages of human subjects*. Los Angeles: Brain Information Service/Brain Research Institute, University of California Press (NIH publ. 204).

Sewitch, D.E. (1987). Slow wave sleep deficiency insomnia: A problem in thermoregulation at sleep onset. *Psychophysiology, 24*, 200–215.

Trinder, J., Montgomery, I., & Paxton, S. J. (1988). The effect of exercise on sleep: The negative view. *Acta Physiologica Scandinavica, 133*(Suppl. 574), 14–20.

White, D.P., Weil, J.V., & Zwillich, C.W. (1985). Metabolic rate and breathing during sleep. *Journal of Applied Physiology, 59*, 384–391.

Zulley, J., Wever, R., & Aschoff, J. (1981). The dependence of onset and duration of sleep on the circadian rhythm of rectal temperature. *Pfleugers Archives, 391*, 314–318.

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