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Effect of Continuous Heat Exposure on Sleep During Partial Sleep Deprivation

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Summary: This study examined the effects of continuous heat exposure on sleep structure during a partial sleep-deprivation regime. The experimental protocol was divided into three periods. After a baseline period (5 days and nights at 20°C), the sleep of the subjects was restricted to the second half of the night (3 a.m.–7 a.m.) for four consecutive nights. The restricted-sleep period was followed by two recovery days and nights. During the deprivation and recovery periods, the ambient temperature was 20°C for six of the 12 subjects and 35°C for the others. Sleep, esophageal and mean skin temperatures were continuously recorded. At 20°C, the expected effect of sleep debt was apparent. There were significant reductions in time spent awake and in latencies for sleep and stage 4 sleep. The duration of stage 4 sleep significantly increased during the four successive restricted-sleep nights, whereas esophageal temperature significantly decreased over the successive days. When heat was added, esophageal temperature decrease was weakened, and the significant increase in stage 4 duration seen at 20°C was not found. The findings suggest that the heat load imposed in our experimental condition has a suppressive effect on sleep stage 4 increase, which is induced by sleep restriction. The hypothesis that an increase in this sleep stage serves as a mechanism for energy conservation should be also considered. **Key Words:** Body temperature—Heat—Human—Sleep restriction.

The findings of studies involving partial sleep deprivation are in general agreement (1–6). They all report a reduction of sleep stage 2 and rapid eye movement (REM) sleep, whereas stage 4 sleep increased or remained constant. Brunner et al. (6) showed that stages 3 and 4 remain unchanged when sleep is restricted for 2 nights to the first 4 hours of the habitual bedtime period. Webb and Agnew (3) found that sleep stage 4 increases with the length of prior wakefulness and that this increase could reflect a behavioral drive. According to Dement and Greenberg (1), there is a need for sleep stage 4 that has priority over the other stages. Because sleep stage 4 is preserved with sleep deprivation, it has been hypothesized that this stage has some essential function. According to Berger et al. (7), slow-wave sleep (SWS) evolved as a mechanism for energy conservation through downward regulation of metabolism. SWS, therefore, has a restorative function and limits the rate of energy expenditure. Increase in SWS in humans after 2 or 3 days of fasting has been interpreted as evidence confirming this hypothesis (8).

The duration of SWS is also influenced by heat. SWS can be induced by moderate peripheral and brain heating. In kangaroo rats, warming of the hypothalamus facilitates SWS (9). In humans, body heating during the day leads to an increase in SWS, particularly in the duration of stage 4 sleep, during the subsequent night (10,11). This effect disappears when the heat load stops 5–6 hours before bedtime (11). The amounts of SWS correlate positively with internal temperatures recorded at bedtime (10,11). This positive relationship can be interpreted as a thermoregulatory response preventing hyperthermia (12,13), because increased sweat gland activity observed during SWS (14) enhances body cooling from skin evaporation.

From exposure to thermal loads during the night only, it is found that SWS is at a maximum level within the thermoneutral zone when compared with heat and cold exposure. This observation reflects a suppressive effect of high ambient temperature on SWS.

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In contrast to acute thermal conditions, little attention has been paid to continuous thermal stresses. Palca et al. (15) reported that cold increased wakefulness and decreased sleep stage 2 during prolonged cold exposure, without any modification of the other sleep stages. A similar observation was made by Buguet et al. (16) on subjects exposed to 17 consecutive Arctic winter nights. A previous study (17) performed on subjects exposed to five consecutive day-nights at 35°C showed that total sleep time was significantly reduced, whereas the duration of sleep stages 3 and 4 was not affected. These results suggest that the enhanced SWS induced by daytime thermal exposure is overcome by nighttime heat exposure. From these data in hot or in cold environments, it appears that the effect of ambient temperature (Ta) on human sleep architecture is more important in acute than in prolonged exposures.

The present study was designed to assess the effects of an external continuous heat load on the temporal distribution of sleep stages—particularly on the need of SWS—during nights when sleep was restricted to the last 4 hours of the night for four consecutive 24-hour periods.

METHODS

Subjects

Twelve male students (age: 19–24 years; height: 1.71–1.81 m; weight: 59–81 kg) participated in the experiment. All subjects were informed of the general nature of the experiment and gave their informed consent. Subjects were given the morningness-eveningness questionnaire (18) and the Eysenck personality inventory (19). There were no deviant scores on these tests. All subjects were established residents of France and they had not lived in a hot climate during the previous year. The subjects habitually slept for 8 hours per night, had no sleep or medical problems and were not drug users.

Experimental design

The experimental design is shown in Fig. 1. The experiment was performed in a climate-controlled apartment (surface area 70 m^2) where the subjects lived continuously throughout the 2-week study period. Subjects were run in threes and recordings were made simultaneously on the three subjects.

A screening night was performed to ensure that normal sleep was present. This night was followed by 5 days and 5 nights (B_1–B_5) in a steady thermal environment (air temperature: T_a = 20°C; dew-point temperature = 7°C; air velocity = 0.2 m·second^{-1}). During the day, the subjects wore cotton track suits and tennis shoes (clothing insulation: 1 CLO). During the night, they slept in pajamas (0.4 CLO) in a bed with two cotton sheets and a wool blanket for covering. They slept from 11 p.m. to 7 a.m. On three of the five consecutive nights (B_1, B_3 and B_5), sleep was monitored. These nights were used as baseline nights. Following the baseline period, sleep time was restricted to 4 hours per night by scheduling bedtime from 3 a.m. to 7 a.m. for four consecutive 24-hour periods (P_1–P_4). Two recovery days and nights (R_1–R_2) followed partial sleep deprivation. Sleep time for recovery nights was 8 hours (11 p.m.–7 a.m.).

For six of the 12 subjects, the sleep restriction regime and the recovery nights were performed at 20°C T_a (group 20), whereas for the others T_a was 35°C (group 35); the air humidity and velocity remained unchanged. T_a was increased the day before night P_1. In the warm condition, daytime clothing consisted of a T-shirt, shorts and tennis shoes (0.3 CLO). At night, the subjects slept wearing only underpants (0.1 CLO) and covered by a cotton sheet.

All subjects followed the same regime; they ate standard meals at 8 a.m., 1:30 p.m. and 7 p.m. They drank bottled spring water ad libitum, but the amounts were measured. They did not take drugs, coffee or alcohol. Throughout the experiment, the clothing and daily activities of the subjects were strictly controlled and monitored via closed-circuit television. Subjects were not allowed to nap. They spent their spare time reading, listening to music, watching TV or doing parlor games.

Physiological recordings

Esophageal temperature (T_{es}) was measured continuously by a thermistor introduced into the esophagus through the nose, at 36–40 cm beyond the external nasal opening. The thermistor was located close to the segment between the left atrium and the aorta. Four local skin temperatures were measured by thermistors sandwiched between two thin copper adhesive ribbons. The thermistors were located on the skin surface according to the sites defined by Ramanathan (20): right calf, right thigh, right arm and right pectoral region. The mean skin temperature ($\bar{T}_{sk}$) was calculated by using the weighting area factors. During the day, T_{es} and $\bar{T}_{sk}$ were recorded in the morning (9 a.m.–10 a.m.) and in the afternoon (4 p.m.–5 p.m.). During the night, all temperatures were recorded at 1-minute intervals. The accuracy of all temperature readings was 0.05°C, and sensitivity to variations was 0.01°C.

Sleep recordings included C3 and F3 electroencephalograms (EEGs) referenced to the opposite mastoid (A2), right and left electrooculograms from the outer canthus referenced to the left mastoid (A1), an elec-

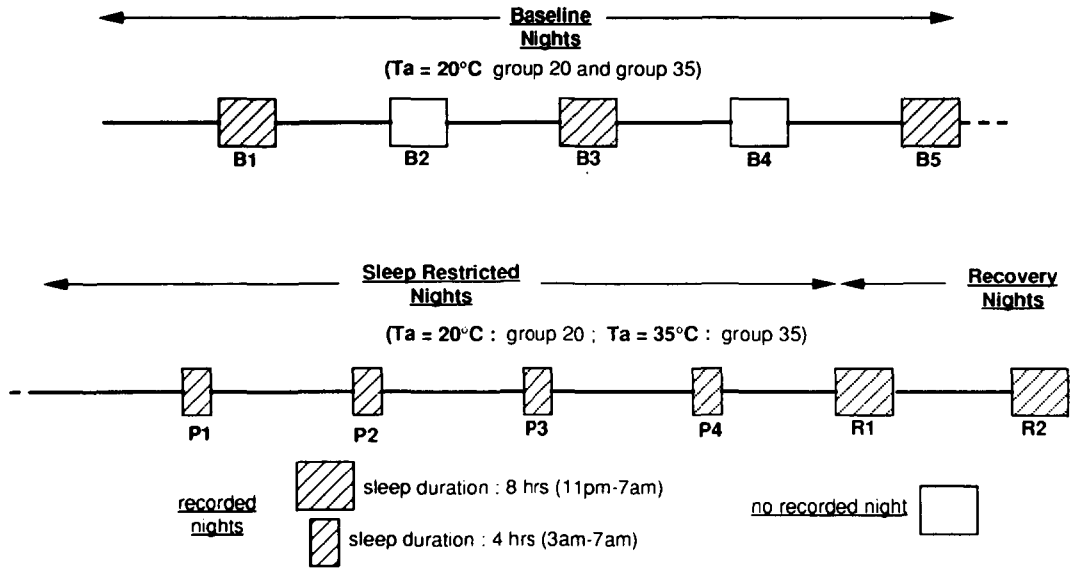

FIG. 1. Experimental design. Shaded areas indicate recorded nights.

tromyogram (EMG) of the chin and an electrocardiogram.

Sleep stages were scored for every 30-second period of the night, following the recommendations of Rechtschaffen and Kales (21). The rate of transient activation phases (TAPs) was measured. The TAPs were characterized by concomitant and reversible modifications on the electrophysiological records (22): on the EEGs there was a short replacement of usual activities by fast frequencies; the EMG showed an increase of muscular tone; and heart rate increased. Most, but not all, of the TAPs were accompanied by body movements.

Statistical analysis

Data were subjected to a multiple analysis of variance for repeated measures. The factors were air temperature (group), night and temperature × night as interaction.

The sleep-deprivation period included the values of nights (P_1–P_4) as dependent variables; mean baseline values were introduced as constant covariates. This enabled us to assess the effect of the different factors (group, night or sleep period) and the interaction (group × night or group × period) after adjusting for differences in covariates. The same procedure was used for recovery nights (R_1 and R_2).

Within-group analyses were also made. They offered indirect evidence of T_a effect and thus supported between-group findings.

The degrees of freedom of the F-value were corrected by the ϵ value of Geisser and Greenhouse (23). When overall F-values were significant ($p < 0.05$), t tests were computed.

All sleep measures, expressed as percent of total sleep time, were tested after the arcsine transformation to stabilize the variance (24).

Statistical analyses on sleep stage durations were computed on data expressed in percent of total sleep time, in percent of global duration of sleep recordings and in minutes. Because results were consistent, only the first ones are reported in the text and tables.

RESULTS

Body temperatures

Nocturnal body temperatures. Figures 2 and 3 show the $\bar{T}_{sk}$ and T_{es} for each group of subjects during baseline, sleep-restricted and recovery nights. In both groups, transient increase of $\bar{T}_{sk}$ was observed at the beginning of the baseline nights. Afterwards, $\bar{T}_{sk}$ decrease, reaching its minimal value at 2–4 a.m., and then it increased until 7 a.m.

During partial sleep deprivation, there was a difference in the shape of $\bar{T}_{sk}$ nocturnal profiles between the two groups of subjects. At 20°C (Fig. 2), $\bar{T}_{sk}$ patterns were similar to the first half of the baseline nights. For group 35, $\bar{T}_{sk}$ did not increase at sleep onset (Fig. 3) on sleep-deprivation nights; there was an initial decrease followed by a slow rise. $\bar{T}_{sk}$ profiles during recovery nights were similar to those recorded during baseline nights, except for the initial $\bar{T}_{sk}$ increase, which was absent in group 35.

At 20°C partial sleep deprivation did not strongly influence T_{es}. Similar to the same time period baseline

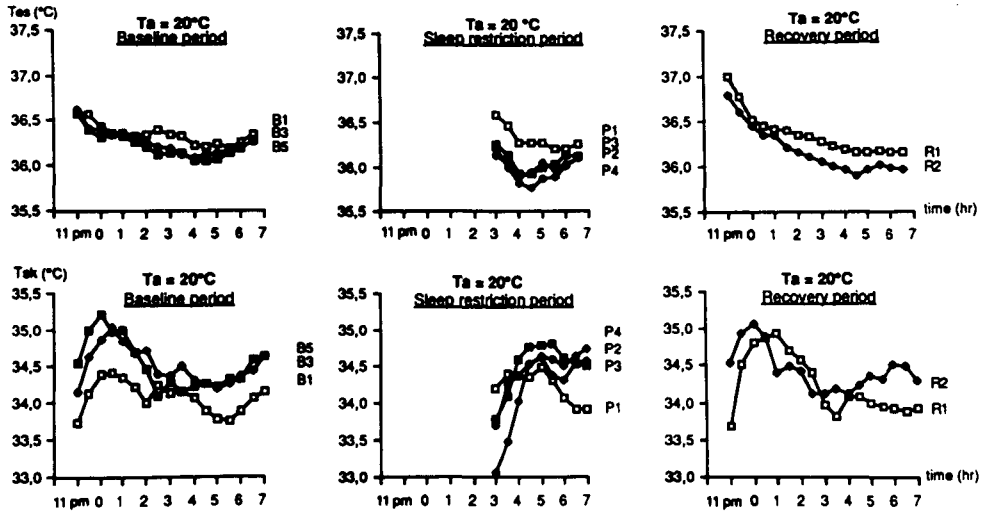

FIG. 2. Nocturnal patterns of mean skin ($\bar{T}_{sk}$) and esophageal (T_{es}) temperatures during the different nights in group 20. Mean values are calculated for the six subjects.

nights, during sleep restriction there was an initial decrease in T_{es} from bedtime to 4:30 a.m., and then T_{es} increased until 7 a.m. (Fig. 2). T_{es} levels observed at the beginning of sleep-deprivation nights at 20°C were significantly lower than those at 35°C ($F_{1,10} = 8.29$; $p = 0.016$). Addition of heat only affected T_{es} during the first 30 minutes (at 3:30 a.m.: $F_{1,10} = 8.15$; $p = 0.017$), but thereafter no significant difference appeared. Inspection of initial T_{es} values showed that there was a significant interaction in $T_a \times$ night ($F_{2,18} = 12.15$; $p < 0.001$). For group 20, the initial value of T_{es} decreased from the first to the last sleep restriction night (36.53 ± 0.35°C; 36.21 ± 0.32°C; 36.25 ± 0.31°C; 36.14 ± 0.29°C; $F_{3,30} = 10.07$, $p < 0.001$), whereas in group 35 T_{es} increased (36.36 ± 0.11°C; 36.42 ± 0.20°C; 36.48 ± 0.19°C; 36.61 ± 0.16°C; $F_{3,30} = 4.03$, $p = 0.016$).

During recovery nights, T_{es} levels were significantly increased by heat throughout the nights when the two groups were compared ($p \leq 0.05$).

Diurnal body temperatures. Mean values for T_{es} and $\bar{T}_{sk}$ recorded during the morning and the afternoon are shown in Fig. 4. At 20°C, daytime T_{es} observed during the sleep-deprivation period decreased over the successive days (morning: $F_{1,30} = 6.52$, $p = 0.060$; afternoon: $F_{1,30} = 5.61$, $p = 0.024$), whereas $\bar{T}_{sk}$ remained at a constant level.

At 35°C, daytime T_{es} increased on the first day of exposure, then decreased until the end of the deprivation period (morning $F_{1,30} = 16.18$, $p < 0.001$; afternoon: $F_{1,30} = 6.48$, $p = 0.016$). $\bar{T}_{sk}$ increased on the first 2 days (morning: $F_{1,30} = 26.80$, $p < 0.001$; afternoon: $F_{1,30} = 85.72$, $p < 0.001$), then reached a near steady-state level.

Sleep measures

Sleep measures with their significant effects analyzed on percent of total sleep time are reported in Tables 1, 2 and 3 for the different nights and for each group of subjects.

For baseline nights, no significant difference appeared in sleep variables between the two groups of subjects, except for small increases of sleep stage 1 durations ($F_{1,10} = 7.45$; $p = 0.021$) and number of arousals ($F_{1,10} = 6.29$; $p = 0.031$). Night effects were significant for durations of wakefulness ($F_{2,9} = 5.84$; $p = 0.027$), sleep stage 1 ($F_{2,9} = 4.25$; $p = 0.044$), sleep stage 2 ($F_{2,9} = 6.40$; $p = 0.021$) and REM sleep ($F_{2,9} = 7.29$; $p = 0.013$). This indicates that the sleep structure changed over the baseline nights. However these changes were similar for the two groups of subjects (nonsignificant interactions).

Compared to baseline nights, restricting sleep to 4 hours during the last part of the night led to a significant decrease in the duration of wakefulness ($F_{3,28} = 26.23$; $p < 0.001$) and sleep stage 1 ($F_{4,34} = 7.50$; $p < 0.001$). Latencies for sleep ($F_{2,18} = 12.65$; $p < 0.001$), for sleep stage 4 ($F_{2,13} = 8.20$; $p = 0.005$) and for REM sleep ($F_{3,26} = 6.47$; $p = 0.003$) were also reduced. There were differences between both groups of subjects for stage 2, stage 4 and REM sleep durations. At 20°C, stage 4 increased between the baseline period and the sleep restriction nights (+16% of total sleep time; $F_{4,17} = 5.56$, $p < 0.001$) mainly at the expense of sleep stage 2 (−13%; $F_{3,15} = 12.62$, $p < 0.001$) and REM sleep (−5%; $F_{3,14} = 4.24$, $p = 0.025$). These effects were also significant when both groups were considered together, but not for group 35. Similar results were observed for

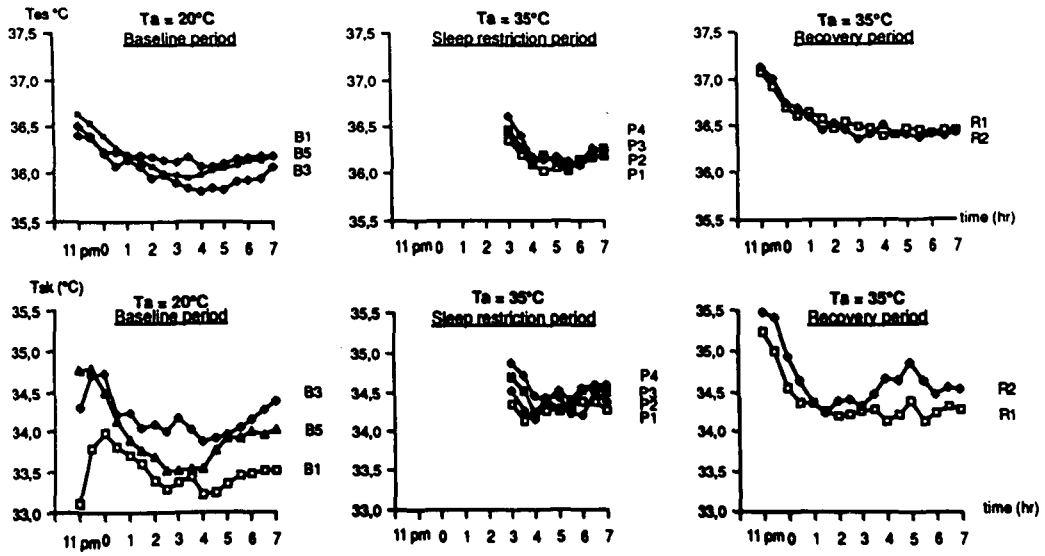

FIG. 3. Nocturnal patterns of mean skin ($\bar{T}_{sk}$) and esophageal (T_{es}) temperatures during the different nights in group 35. Mean values are calculated for the six subjects.

both groups when the duration of stage 4 sleep recorded during the restriction period was compared to that recorded in the first 4 hours of baseline nights.

A night effect was observed for most of the variables, indicating that changes in sleep structure occurred over the four sleep-deprivation nights. For the two groups, the durations of wakefulness after sleep onset ($F_{3,8}$ = 4.51; p = 0.040) and of sleep stage 1 ($F_{3,8}$ = 9.50; p =

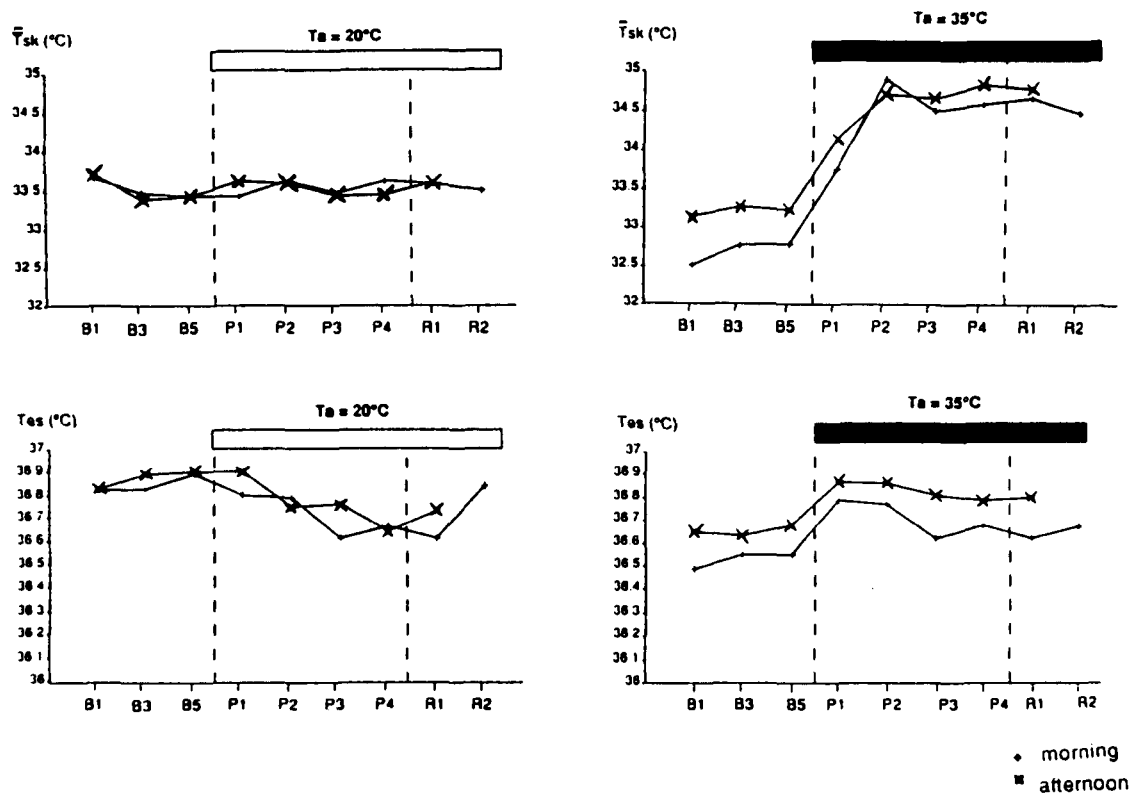

FIG. 4. Diurnal patterns of mean skin ($\bar{T}_{sk}$) and esophageal (T_{es}) temperatures in group 20 (left) and group 35 (right) across the experimental design. B_1–B_5: days after baseline nights; P_1–P_4: days after sleep-restricted nights; R_1 and R_2: days after recovery nights. +: morning; ×: afternoon.

TABLE 1. *Mean durations ± 1 SD (TST: minutes; W: % of duration of global sleep recording; other sleep stages: % TST) of total sleep time and of the different sleep stages during the different nights for each group of subjects*

Sleep variables	Baseline nights			Statistical results[a]	Sleep restriction nights	
	B1	B3	B5		P1	P2
TST						
Group 20	413 ± 47	448 ± 17	453 ± 11		219 ± 13	236 ± 3
Group 35	389 ± 63	404 ± 93	438 ± 23		207 ± 37	236 ± 4
Wakefulness						
Group 20	11 ± 8	7 ± 4	5 ± 2	a	8 ± 5	1 ± 1
Group 35	17 ± 12	9 ± 4	8 ± 4		14 ± 16	1 ± 1
Stage 1						
Group 20	4 ± 2	4 ± 1	2 ± 1	ab	4 ± 1	2 ± 1
Group 35	6 ± 3	5 ± 3	4 ± 1		5 ± 2	2 ± 1
Stage 2						
Group 20	54 ± 10	49 ± 2	50 ± 4	a	44 ± 4	38 ± 9
Group 35	51 ± 9	45 ± 9	46 ± 6		48 ± 7	43 ± 11
Stage 3						
Group 20	12 ± 4	10 ± 5	9 ± 3		12 ± 6	12 ± 5
Group 35	10 ± 4	10 ± 4	11 ± 1		12 ± 3	13 ± 7
Stage 4						
Group 20	11 ± 5	10 ± 5	12 ± 4		18 ± 5	27 ± 9
Group 35	10 ± 4	12 ± 7	12 ± 7		18 ± 10	19 ± 8
REM sleep						
Group 20	19 ± 5	27 ± 2	27 ± 3	a	22 ± 5	21 ± 9
Group 35	23 ± 6	28 ± 5	27 ± 5		17 ± 8	23 ± 9

[a] Significant ($p < 0.050$) statistical results are indicated for the following effects: a: night effect within a given sleep period (baseline, sleep restriction or recovery); b: group effect within a given sleep period; c: interaction night × group within a given sleep period; d: effect of the sleep period (baseline vs. sleep restriction, baseline vs. recovery).

0.005) significantly decreased between the first sleep-deprivation night and the others. Latencies for sleep ($F_{3,8} = 18.02$; $p = 0.001$), for sleep stage 4 ($F_{3,8} = 4.63$; $p = 0.037$) and for REM sleep ($F_{3,8} = 8.60$; $p = 0.007$) also decreased. The duration of sleep stage 4 significantly increased over the four successive nights ($F_{3,8} = 6.54$; $p = 0.015$) but only at 20°C (interaction group × night: $F_{3,8} = 4.28$, $p = 0.044$; group effect: $F_{1,9} = 18.80$, $p = 0.002$) and mainly at the expense of sleep stage 2 (night effect: $F_{3,8} = 4.40$, $p = 0.045$; group effect: $F_{1,9} = 6.49$, $p = 0.031$).

During recovery nights, there was a significant increase of REM sleep duration in both groups (+3%: $F_{1,10} = 6.14$; $p = 0.030$) from the first to the second night at the expense of sleep stage 2 (−5%: $F_{1,10} = 7.47$; $p = 0.021$). The sleep latency increased (+16 minutes: $F_{1,10} = 6.30$; $p = 0.027$) at 35°C (interaction group × night: $F_{1,10} = 12.24$; $p = 0.006$). On R2, warm exposure significantly increased sleep stage 1 duration, but only in group 35 (interaction: $F_{1,10} = 8.38$; $p = 0.016$). Warm exposure also increased the duration of wakefulness (+8% of global duration of sleep recording), the number of sleep stage changes (+12), the number of sleep stage changes towards arousal (+5) and total sleep TAPs (+3%) between R1 and R2. Although these measures did not reach statistical significance, sleep appeared to be more restless, suggesting that after the first recovery night the disruptive effect of heat overcame the changes in sleep structure that occurred with sleep restriction. These disturbances were not observed for the first recovery night, during which sleep debt pressure was more important and sleep quality was less impaired by heat.

Except for sleep, which occurred 8 minutes earlier ($F_{1,10} = 9.37$; $p = 0.012$), and sleep stage 4, which occurred 22 minutes earlier ($F_{1,10} = 12.77$; $p = 0.005$) on recovery nights than on baseline nights, independent of air temperature, no significant rebound in sleep structure could be observed.

DISCUSSION

The most striking finding of the present study was that the principal change in sleep architecture induced by air temperature involved sleep stage 4. The length of stage 4 increased at 20°C during the sleep-restriction period. This did not occur when heat was added. The thermal load decreased the importance of prior sleep loss on sleep architecture, especially the need for sleep stage 4.

During sleep restriction at 20°C, EEG data were similar to the findings of other studies showing reductions

TABLE 1. *Continued.*

Sleep variables	Sleep restriction nights			Recovery nights		
	P3	P4	Statistical results	R1	R2	Statistical results
TST						
Group 20	218 ± 17	237 ± 3	a	438 ± 27	458 ± 11	
Group 35	237 ± 2	236 ± 3		465 ± 5	426 ± 32	
Wakefulness						
Group 20	2 ± 1	1 ± 1	ad	8 ± 5	4 ± 2	
Group 35	1 ± 1	1 ± 1		2 ± 1	10 ± 6	
Stage 1						
Group 20	2 ± 1	2 ± 2	ad	3 ± 1	2 ± 1	c
Group 35	2 ± 1	3 ± 1		2 ± 1	4 ± 2	
Stage 2						
Group 20	39 ± 5	35 ± 4	abcd	54 ± 5	48 ± 5	a
Group 35	43 ± 10	39 ± 10		50 ± 3	44 ± 7	
Stage 3						
Group 20	14 ± 6	10 ± 3		8 ± 3	9 ± 3	
Group 35	13 ± 5	16 ± 10		12 ± 3	11 ± 3	
Stage 4						
Group 20	27 ± 7	36 ± 5	abd	13 ± 5	16 ± 4	c
Group 35	18 ± 8	19 ± 11		13 ± 3	13 ± 4	
REM sleep						
Group 20	18 ± 7	17 ± 3	d	22 ± 5	25 ± 3	a
Group 35	24 ± 9	23 ± 5		23 ± 6	28 ± 6	

of sleep stage 2, wakefulness and an increase in sleep stage 4 (1–4,25). From the first to the last sleep-restriction night, significant reductions in sleep stage 1, sleep latency and time spent awake indicate that sleep became more efficient as total sleep time decreased. Stage 4 sleep mainly increased at the expense of sleep stage 2 and wakefulness, confirming the findings of Johnson and Mac Leod (2). Sleep stage 4 also occurred earlier during sleep-restricted nights. The priority to preserve sleep stage 4 by limiting the amounts of the other sleep stages suggests that this stage is an important component in sleep regulation and is under homeostatic control. Brunner et al. (6) did not find any significant enhancement of stage 4 in their experiment. These discrepant results might be explained by differences in thermal condition or in the duration of the sleep-restriction period. It might be that the increase in sleep stage 4 observed during the four successive nights of sleep restriction is only apparent when the amount of sleep deprivation reaches a critical level. As stated by these authors, a minor and nonsignificant enhancement of SWS activity appears on the second night of sleep restriction.

According to some authors, the increases in stage 4 and SWS seem to be related to the wakefulness time before sleep (3) and/or the influence of circadian factors (26). In our experiment, however, length of prior wakefulness and amount of sleep loss were the same for both groups. The explanation regarding the influence of circadian factors can also be discarded; sleep deprivation occurred at the same time of the night. Moreover, the circadian influence is modest and is not significant relative to that of prior wakefulness (3,26,27). In the same way, the positive relationship between the level of the internal temperature measured at sleep onset and SWS (7,28) is not consistent in our study; the lowest T_{es} level occurred at 11 p.m., the longest SWS duration (group 20). It is likely that the discrepancy between these observations is a result of differences in protocol routines (acute vs. chronic exposure to thermal load, sleep-restricted subjects vs. nondeprived subjects). A first explanation for an increase in sleep stage 4 refers to sleep quality. Muzet et al. (29) found that to enter stage 4 sleep, the sleeper had to have a cessation of body movements. Pregnant women have less stage 4 sleep during the last 3 months of pregnancy, as they have increased restless sleep. TAPs, which are often accompanied by body movements, were more frequent at 35°C than at 20°C, especially in sleep stage 2 (sleep of group 35 was more restless). However, this does not explain the progressive increase in sleep stage 4 that was observed during the sleep

TABLE 2. *Mean durations ± 1 SD of sleep latencies and transient activation phases (minute^{-1}) during the different nights, for each group of subjects*

Sleep variables	Baseline nights			Statistical results[a]	Sleep restriction nights	
	B1	B3	B5		P1	P2
Sleep latency						
Group 20	30 ± 17	21 ± 37	24 ± 11		15 ± 9	3 ± 3
Group 35	26 ± 20	18 ± 7	23 ± 23		15 ± 17	3 ± 3
Stage 4 latency						
Group 20	55 ± 27	57 ± 18	41 ± 13		31 ± 12	25 ± 12
Group 35	99 ± 72	60 ± 56	53 ± 26		36 ± 22	23 ± 7
REM sleep latency						
Group 20	114 ± 41	94 ± 16	90 ± 22		74 ± 10	67 ± 7
Group 35	126 ± 54	100 ± 31	103 ± 24		73 ± 25	64 ± 14
TAPs TST						
Group 20	0.15 ± 0.04	0.17 ± 0.04	0.15 ± 0.02		0.13 ± 0.01	0.11 ± 0.02
Group 35	0.22 ± 0.14	0.22 ± 0.11	0.22 ± 0.07		0.18 ± 0.07	0.15 ± 0.03
TAPs stage 2						
Group 20	0.15 ± 0.08	0.15 ± 0.04	0.14 ± 0.04		0.13 ± 0.03	0.10 ± 0.03
Group 35	0.25 ± 0.22	0.22 ± 0.14	0.25 ± 0.13		0.21 ± 0.08	0.20 ± 0.12
TAPs stage 4						
Group 20	0.10 ± 0.06	0.09 ± 0.06	0.11 ± 0.08		0.07 ± 0.03	0.06 ± 0.02
Group 35	0.08 ± 0.04	0.07 ± 0.04	0.08 ± 0.03		0.09 ± 0.04	0.07 ± 0.03
TAPs REM sleep						
Group 20	0.20 ± 0.10	0.20 ± 0.10	0.20 ± 0.10		0.19 ± 0.06	0.20 ± 0.07
Group 35	0.17 ± 0.05	0.30 ± 0.12	0.28 ± 0.15		0.31 ± 0.34	0.18 ± 0.08

[a] Significant ($p < 0.050$) statistical results are indicated for the following effects: a: night effect within a given sleep period (baseline, sleep restriction or recovery); b: group effect within a given sleep period; c: interaction night × group within a given sleep period; d: effect of the sleep period (baseline vs. sleep restriction, baseline vs. recovery).

deprivation period at 20°C, because there was no reduction in the frequency of TAPs from the first to the last sleep-restriction night in any sleep stage. Another explanation is related to the fact that the Ta of 35°C was above thermoneutrality and could have counteracted an increase in sleep stage 4 induced by sleep deprivation. The suppressive effect of nocturnal heat on SWS and sleep stage 4 has been observed in acute thermal exposures (30–32) during which there was a significant reduction of sleep stage 4. However, this has not been confirmed by any other study analyzing the effect of continuous heat exposure on non-sleep-deprived subjects, stressing that there was no change in sleep stage 4 from night to night (17). Another explanation for differences in stage 4 duration during the sleep-restriction period in both groups refers to the primary function of SWS to conserve energy (7,33). Hypothalamic thermosensitivity is reduced at the onset of SWS, causing a decrease in heat production to conserve energy (34). Compared to baseline values, subjects of group 20 showed a fall in daytime T_{es} of about 0.3°C throughout the sleep-restriction period. Most sleep-deprivation investigations have also found a fall in body temperature, indicating that the range of air temperatures defining the thermoneutrality was increased. Subjects felt colder probably because their body temperatures fell below their thermoregulatory set point. In the present study, air temperature of 20°C could be considered a mild cool load for sleep-deprived subjects as they were dressed in our study. The subjects may have fought against the cool during the day by increasing heat production and/or body movements. The increased amount of stage 4 sleep observed at 20°C might be attributed to a cumulative effect of sleep restriction and thermal condition. This finding agrees with the study of Sewitch et al. (35), who reported that an increase in sleep stage 4 in response to mild nocturnal cold stress in women might be viewed as an adaptation for energy conservation that offsets the costs of energy expenditure caused by increased muscular activity or thermogenesis during prior wakefulness. When the cool effect is abolished ($T_a = 35$°C), the cost of daytime energy expenditure decreases and SWS or stage 4 sleep is unchanged when compared to baseline data. This could suggest that during sleep, the function of energy conservation related to sleep stage 4 overcomes the protective nocturnal thermoregulatory processes of body temperature. This hypothesis is based on speculations that, at 20°C, the subjects increased their diurnal muscular activity and their energy expenditure. This does not agree with Fiorica et al.'s study (36), which suggested that during sleep depri-

TABLE 2. *Continued.*

Sleep variables	Sleep restriction nights			Recovery nights		
	P3	P4	Statistical results	R1	R2	Statistical results
Sleep latency						
Group 20	6 ± 2	2 ± 2	ad	19 ± 4	16 ± 5	acd
Group 35	3 ± 2	5 ± 3		6 ± 3	23 ± 13	
Stage 4 latency						
Group 20	23 ± 9	12 ± 3	ad	44 ± 21	37 ± 14	d
Group 35	22 ± 6	19 ± 7		22 ± 6	51 ± 32	
REM sleep latency						
Group 20	67 ± 15	72 ± 8	ad	96 ± 25	86 ± 15	
Group 35	88 ± 35	73 ± 10		94 ± 15	89 ± 25	
TAPs TST						
Group 20	0.09 ± 0.02	0.11 ± 0.06		0.13 ± 0.02	0.13 ± 0.02	
Group 35	0.15 ± 0.03	0.14 ± 0.04		0.15 ± 0.04	0.18 ± 0.04	
TAPs stage 2						
Group 20	0.11 ± 0.04	0.11 ± 0.05		0.13 ± 0.09	0.13 ± 0.06	
Group 35	0.21 ± 0.07	0.19 ± 0.07		0.17 ± 0.07	0.19 ± 0.04	
TAPs stage 4						
Group 20	0.05 ± 0.02	0.06 ± 0.03		0.07 ± 0.04	0.06 ± 0.03	
Group 35	0.07 ± 0.02	0.06 ± 0.05		0.07 ± 0.04	0.09 ± 0.04	
TAPs REM sleep						
Group 20	0.17 ± 0.06	0.17 ± 0.10		0.19 ± 0.12	0.19 ± 0.12	
Group 35	0.17 ± 0.17	0.16 ± 0.08		0.15 ± 0.08	0.22 ± 0.08	

TABLE 3. *Mean values ± 1 SD of the number of sleep stage episodes and sleep stage changes during the different nights, for each group of subjects*

Sleep variables	Baseline nights			Statistical results	Sleep-restriction nights				Recovery nights	
	B1	B3	B5		P1	P2	P3	P4	R1	R2
Wakefulness										
Group 20	10 ± 5	5 ± 3	5 ± 3	b[a]	3 ± 3	1 ± 1	1 ± 1	1 ± 2	5 ± 4	1 ± 2
Group 35	18 ± 15	13 ± 9	10 ± 4		5 ± 3	1 ± 2	1 ± 1	1 ± 1	5 ± 3	8 ± 5
Stage 1										
Group 20	19 ± 7	16 ± 4	13 ± 3		9 ± 3	6 ± 3	5 ± 2	5 ± 4	13 ± 5	10 ± 4
Group 35	28 ± 17	24 ± 13	20 ± 6		11 ± 5	7 ± 4	6 ± 2	8 ± 2	13 ± 4	20 ± 10
Stage 2										
Group 20	26 ± 7	26 ± 2	24 ± 6		15 ± 5	14 ± 4	13 ± 4	15 ± 5	23 ± 6	22 ± 5
Group 35	30 ± 15	30 ± 12	29 ± 5		16 ± 3	15 ± 4	13 ± 3	14 ± 4	25 ± 2	27 ± 9
Stage 3										
Group 20	7 ± 2	8 ± 2	10 ± 5		6 ± 3	7 ± 2	6 ± 3	7 ± 3	8 ± 2	9 ± 2
Group 35	7 ± 1	6 ± 2	9 ± 1		5 ± 1	6 ± 2	6 ± 2	5 ± 3	8 ± 4	9 ± 2
Stage 4										
Group 20	3 ± 1	3 ± 2	5 ± 3		3 ± 1	4 ± 1	3 ± 2	4 ± 2	4 ± 2	5 ± 2
Group 35	3 ± 1	2 ± 1	4 ± 2		3 ± 1	4 ± 2	3 ± 2	3 ± 2	4 ± 3	4 ± 2
REM sleep										
Group 20	7 ± 2	7 ± 2	8 ± 3		5 ± 2	6 ± 2	5 ± 2	8 ± 4	8 ± 4	8 ± 3
Group 35	5 ± 1	7 ± 2	7 ± 2		5 ± 4	6 ± 2	4 ± 2	5 ± 1	8 ± 3	7 ± 2
Sleep change										
Group 20	74 ± 20	69 ± 6	66 ± 18		43 ± 14	40 ± 11	34 ± 11	41 ± 12	62 ± 13	56 ± 14
Group 35	93 ± 48	84 ± 33	83 ± 13		47 ± 8	41 ± 12	33 ± 8	37 ± 9	66 ± 12	78 ± 22
Sleep change towards arousal										
Group 20	22 ± 8	20 ± 4	18 ± 6		12 ± 4	9 ± 4	8 ± 3	9 ± 4	17 ± 5	15 ± 3
Group 35	29 ± 19	26 ± 14	23 ± 6		12 ± 5	9 ± 3	9 ± 2	9 ± 4	17 ± 3	22 ± 9

[a] Statistically significant ($p < 0.050$) for group effect within a given sleep period.

vation the hypothalamic set point is lowered, based on the finding that decreased body temperature was not accompanied by a greater energy expenditure in resting sitting subjects. They reported that metabolism measured at rest in a thermoneutral (26.7°C) or cold (10°C) environment was not different, although rectal temperature was 0.5°C lower in sleep-deprived subjects. Thus, they concluded that sleep-deprived subjects can maintain a thermal balance at a lower body temperature without any increase in thermogenesis. Further experiments analyzing energy expenditure and body motility during daytime exposure to thermal stress would be necessary to demonstrate this hypothesis in active subjects.

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