THE CIRCADIAN PATTERN OF UNRESTRICTED SLEEP AND ITS RELATION TO BODY TEMPERATURE, HORMONES, AND ALERTNESS

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For many individuals shift work is detrimental to health and well-being (cf. Maurice, 1975; Agervold, 1976; Åkerstedt & Fröberg, 1976, 1979; Rutenfranz, Colquhoun, Knauth, & Ghata, 1977). The major problem is to keep awake during the night shift and to be able to sleep during the subsequent day. For a long time the cause of disturbed day sleep was attributed to factors in the environment and many survey studies tried to establish which factors were most disturbing. While extemely noisy environmental conditions, of course, will affect sleep, it now appears that the normal range of sleep environments (e.g. housing conditions) can hardly be of more than marginal importance as shown e.g., in survey studies by Aanonsen (1964), Åkerstedt and Zamore (1977), Åkerstedt and Torsvall (1977). This is supported by the fact that EEG-recorded sleep of shift workers has been found to be shorter and to have a different distribution during day sleep not only at home but also under noise-free environmental conditions in the laboratory (Bryden & Holdstock, 1973; Ehrenstein, Müller-Limmroth, Schaffler, & Thebaud, 1970; Foret & Benoit, 1972, 1974, 1978; Matsumoto, 1978). Also studies of night workers without a comparable day shift show unusually short sleep during the day (Lille, 1967; Kripke, Cook, & Lewis, 1971).

Typical sleep problems may be illustrated by results from one of our own studies. In Figure 1 are presented data from 3-shift work with an exceptionally early shift change schedule of 0445h-1245h-2045h. Self-ratings ranged from "never" to "practically always", scored from 1 to 4. The figure shows that the unrestricted sleep after the afternoon shift was longest and most satisfying. Night sleep before the morning shift was short and very early terminated (by the alarm clock). Day sleep after the night shift was as short as the night sleep and as little satisfying. In spite of day sleep being unrestricted, subjects reported that they were unable to retain sleep. In addition, sleep length after the night shift correlated $r = .65$ ($p < .001$) with satisfaction with sleep while no such relation was found for the other shifts Thus, the questionnaire data suggest that day sleep is inferior to night sleep because of "internal" factors terminating sleep too early.

The cause of the difficulties of day time sleep may be the conflict between the sleep/wake pattern demanded by work and the conditions offered by the circadian system (cf. Aschoff, 1978). Thus, sleep may presumably be differentially successful depending on circadian phase. The deactivation required by sleep may be interfered with by the high day time levels of activation while low levels of activation during the night may conflict with the demands required by work tasks. As far as we can see, the circadian aspects of unrestricted sleep have not been systematically studied. They have been touched upon mainly in comparisons of day and night sleep in shift work studies as

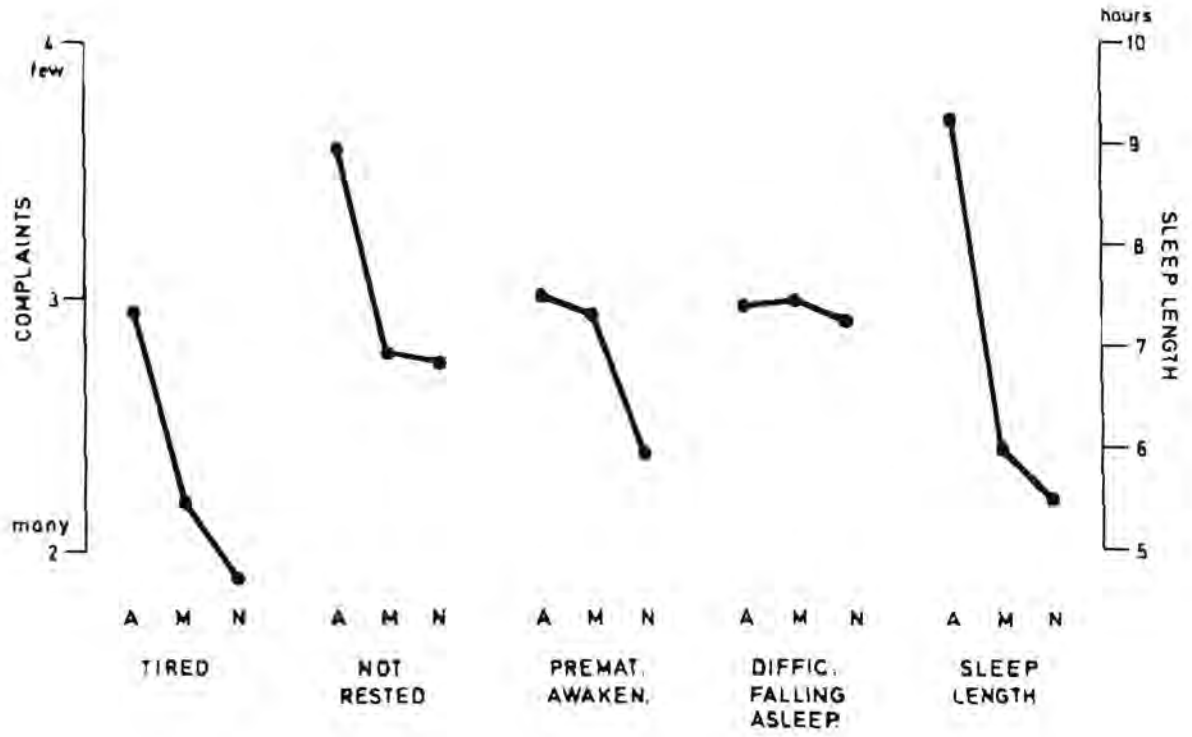

Figure 1. Sleep length and self-ratings of sleep quality in a group of 300 3-shift workers on morning (M), afternoon (A), and night shifts (N).

cited above, and in laboratory simulations of phase shifts (Weitzman, Kripke, Goldmacher, McGregor, & Nogeire, 1970; Berger, Walker, Scott, Magnuson, & Pollack,1971; Webb, Agnew, & Williams, 1971; Knauth & Rutenfranz, 1972; Evans, Christie, Lewis, Daly, & Moore-Robinson, 1972; Taub & Berger, 1973a, b, 1974, 1976; Webb & Agnew, 1978). The laboratory simulations, however, do not show the same impaired day sleep as the field studies.

Information on circadian properties of sleep has also been obtained from studies of scheduled napping. Studies of day time naps have shown that REM sleep is concentrated to the morning hours, while slow wave sleep (SWS) is a function of the amount of prior wakefulness (Maron, Rechtschaffen, & Wolpert, 1964; Webb, Agnew, & Sterntahl, 1966; Webb & Agnew, 1967; Karacan, Finley, Williams, & Hursch, 1970; Webb & Agnew, 1971a, b; Agnew & Webb, 1973). The day time nap studies have also been extended to cover the entire nychthemeron in regimes with several consecutive ultradian sleep/wake cycles (Weitzman, Nogeire, Perlow, Fukushima, Sassin, McGregor, Gallagher, & Hellman, 1974; Moses, Hord, Lubin, Johnson, & Naitoh, 1975; Webb & Agnew, 1975, 1977; Carskadon, & Dement, 1975, 1977; Lubin, Hord, Tracy, & Johnson, 1976; Hume & Mills, 1977; Moses, Lubin, Naitoh, & Johnson, 1978). Generally, these studies have confirmed the pronounced circadian patterning of sleep. However, interesting as these "nap" studies are, it is doubtful whether they can be applied to the shift work situation. Restricted ultradian sleep/wake regimes, as several authors point out, certainly are not miniatures of normal, unrestricted sleep.

If sleep does show a pronounced circadian rhythmicity there is an obvious interest in knowing why this occurs, or at least to know which other circadian parameters may be predictive of sleep characteristics. Nothing conclusive has been published on this subject although there exist some indirect data, particularly in relation to body temperature. Thus, the large number of isolation studies of the Aschoff group show that sleep during free-run tends to commence shortly before the temperature trough and end shortly before the peak (cf. Wever, 1979). Such results have been confirmed by Weitzman, Czeisler, Fusco, and Moore-Ede (1976). Zulley (1979) has shown a relation to exist between sleep during free-run and the temporal position of the circadian temperature trough. For night and morning sleep, results by Breithaupt, Hildebrandt, Dohre, Josch, Sieber, and Werner (1978) show shorter sleep being associated with increasing body temperature. For ultradian sleep/wake studies Moses et al. (1975) and Weitzman et al. (1974) have shown negative intraindividual correlations between body temperature and sleep length. Finally, case studies of patients with free-running rhythms have found clear relations between the circadian rhythm of body temperature and the positioning of sleep, the latter being difficult on the rising portion of the temperature rhythm (Kokkoris, Weitzman, Pollack, Spielman, Czeisler, & Bradlow, 1978; Miles, Raynal, & Wilson, 1977). Very little data exist on the relation between sleep and other circadian parameters.

As an independent variable, sleep strongly affects the physiology; it may e.g., synchronize the circadian system (Webb & Agnew, 1974; Aschoff, 1978; Wever,1979). Aside from such central effects on the pacemaker(s), the effects of sleep on the overt manifestations of rhythms will interfere with attempts to estimate the phasing of the central pacemaker. Such "masking" effects (Aschoff, 1978; Mills, Minors, & Waterhouse, 1978) may lead to entirely false

conclusions about the extent of circadian adjustment. However, little is yet known about such effects.

Some of our recent research has a bearing on the issues discussed above and may be of relevance to the theme of this symposium. The purpose of the present paper is to summarize the relevant results from this research. In particular, attention will be focussed on the circadian pattern of unrestricted sleep and its relation to, and effect on other circadian rhythms of possible importance to sleep/wake alternation, e.g., subjective alertness, body temperature, and the urinary excretion of catecholamines, cortisol, and melatonin. Data are still being analysed and the results to be presented are preliminary.

Methods

The investigation was carried out with six subjects in the age range 29-45 yr. The subjects were exposed to one sleep session/condition per week in such a way that the nychthemeron was covered with bedtimes in 4-hour intervals, beginning with a normal bedtime at 2300 h after 16 hours awake and ending with the seventh bedtime at 2300 hours after 40 hours awake (see Figure 2). The order of bedtime conditions was counterbalanced.

For each session the subject reported to the laboratory at 1800 h after a normal day of sedentary work. After electrode application, measurements started at 1900 h and continued to bedtime. During this time activity was controlled according to a 2-hour module system (see Figure 2). In each module urine was collected, self-ratings made, and 300 ml of water and a standard sandwich was consumed. All modules were standardized as far as possible also with respect to physical and mental activity. The subjects spent all time in the sleep laboratory and were isolated from external synchronizers, but had a rough idea of time of day because of the measurement intervals. At bedtime, particular care was taken to instruct the subjects to sleep until they felt that sleep no longer was needed. Immediately after rising the subjects completed self-ratings and voided urine before being allowed any contact with time cues.

The self-ratings of sleepiness consisted of a 13 point scale, varying between "extremely alert" and "extremely sleepy". Rectal temperature was recorded continuously. Biochemical analyses of catecholamines, cortisol, and melatonin were carried out according to Andersson, Hovmöller, Karlsson, and Swenson (1974); Gustavsson and Sigurdsson (1979); Wetterberg, Eriksson, Friberg, and Vangbo (1978),respectively. Scoring of sleep stages followed the recommendations of Rechtschaffen and Kales (1968). For statistical analysis data from the seven bedtime conditions were combined to a sequence to which a one factor analysis of variance for repeated measures was applied (Winer, 1971). In some instances also two-tailed t-tests for repeated observations were employed.

Results

Circadian Patterns of EEG-Sleep

Figure 3 illustrates the main results for EEG sleep parameters. Sleep

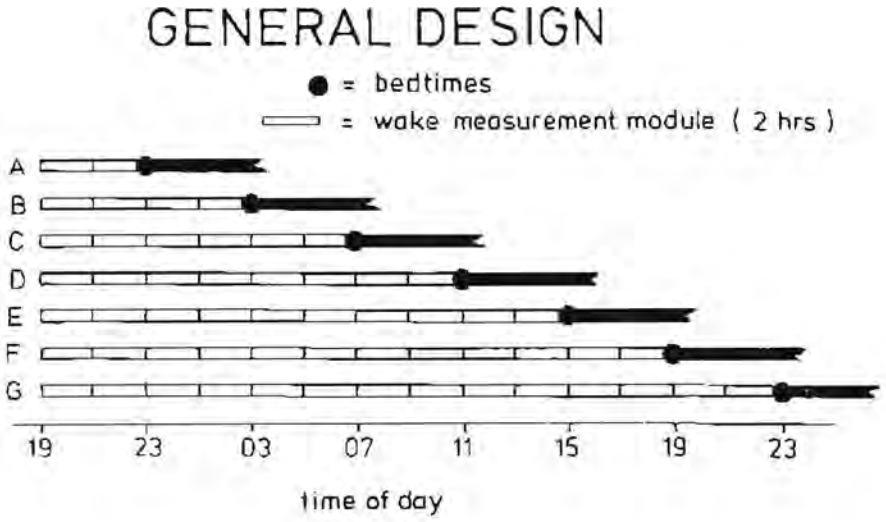

Figure 2. General design.

length showed a highly significant circadian variation with maximum sleep following after evening bed-times. Sleep after morning bedtimes was reduced to approximately half. The absolute amounts of Stage 2 and REM sleep showed the same pattern as total sleep while SWS did not vary significantly across bedtimes. Stage 1 sleep showed a significant gradual decrease. Sleep latencies were very short and did not vary over time. When relative amounts were computed Stage 2 retained the significant original pattern while the other stages were flattened.

Several points above need comments. Firstly, the circumstances around termination of sleep and the criteria for it is of central importance for many of the results in the present study--most studies of sleep/wake schedules have not allowed the subjects to spontaneously terminate sleep. In the present study, the last minute asleep before getting up was considered the end of sleep. In case of protracted oscillation between wake and Stage 1, sleep was considered to have ended immediately before a 10 minute sequence scored as waking. As the subjects had no way of knowing whether it would be day or night, they did not know whether it would be any "point in" leaving the bed. As a matter of fact, the subjects remained longer in bed after awakening from the short morning and day sleeps (about 30 minutes) than from the longer night sleeps (about 15 minutes). Presumably, this time was spent "deciding" whether to get up or not. Also, self-ratings of sleepiness were lower following the short day time sleeps. Thus, it appears that the day time awakenings and decisions to rise were due to a genuine feeling of having slept enough.

The short day time sleep is in line with the field studies of shift workers cited previously. As mentioned, these results contrast with those from artificial phase shift studies. Comparing the studies, it is noteworthy that the field studies and the present ("artificial" shift) study were carried out with subjects in age ranges rather representative of the working population while the other studies of artificial phase shifts have used young adults, mostly students. In our experience the ability to sleep during day time is sharply reduced with increasing age (Åkerstedt, 1976) and it is possible that age differences may be, at least, part of the cause of the discrepancy.

With respect to REM sleep the low absolute day time amounts agree with the previously mentioned phase shift studies. Most of the nap studies, however, show high amounts in the morning. To make our results comparable to these studies we isolated the first two hours of each bedtime condition and found a significant peak in REM sleep in the morning (see Figure 3). Thus, it appears that total REM sleep is cut short by the subjects' early awakening during morning sleep, as was also shown by Verdone (1967) in experiments on sleep satiation. SWS, in contrast, was not affected by the length of sleep but was practically always "finished" before it could be affected by the termination of sleep.

Sleep deprivation apparently did not affect the results to any great extent. Comparisons of conditions A and G suggested only a marginal increase in TST and SWS. The reason for this lack of effect could be that the present study falls in an intermediate range of wakefulness time. The latter varied between the normal prior wakefulness of 16 hours and the lowest amount of proper deprivation of sleep, i.e., 40 hours (if time of day is kept constant). From the results by Webb and Agnew (1971) it is clear that the linear relation

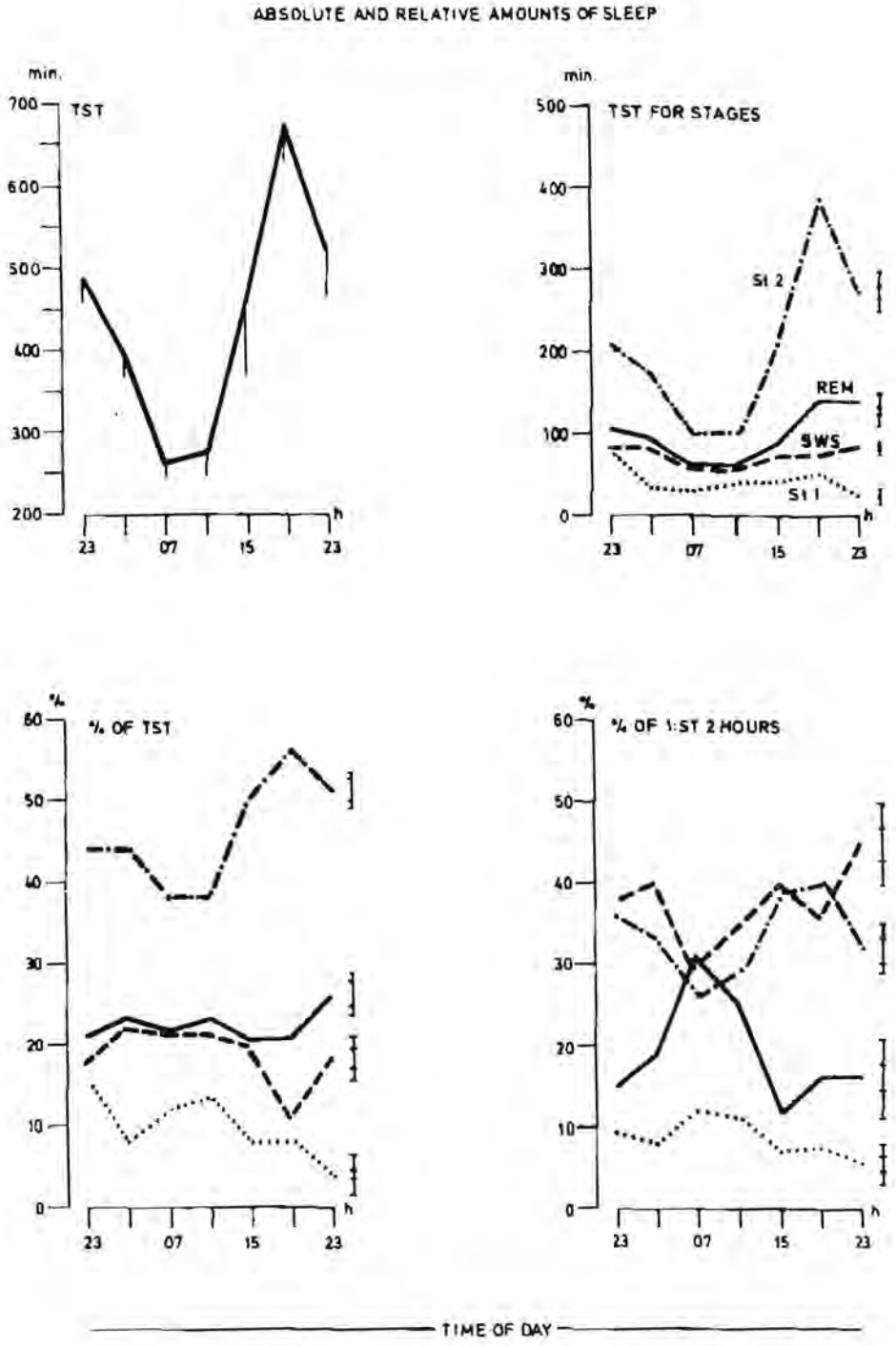

Figure 3. Means of different sleep parameters for a group of 6 subjects sleeping at 7 different times of day. Values plotted at bedtimes. 1 S.E. indicated either at where obtained or at the right hand side of each curve as largest and smallest value for the curve.

between prior wakefulness and SWS (Hume & Mills, 1977) is valid only within the normal wakefulness interval 0-16 hours. Thereafter the curve rapidly levels off. As to TST, the fact that sleep at 1900 h (condition F, see Figure 2) was longer than sleep at 2300 h (condition G) suggests that recovery sleep from sleep deprivation may be cut short by the rising arousal rhythm in the morning if that sleep is taken at the "normal" time (2300 h). It is interesting to speculate if recovery from long periods of sleep deprivation could be sped up by advancing bedtime, thus allowing a longer time in bed before the morning rise of arousal.

Effects of Sleep on Circadian Rhythms

To estimate the effects of sleep at different times of day on urinary excretion levels, two types of analyses were carried out. First, the excretion values during waking were averaged over those periods that correspond to sleep time and the two values were compared (Figure 4). Second, the excretion during sleep was compared with that of the immediately preceding two-hour-module during waking and a difference score obtained (Figure 5).

For adrenaline excretion there was a pronounced circadian rhythm during waking. This was not the case, however, for excretion during sleep. Rather, the impression was one of an almost complete shut-off, resulting in significantly reduced levels mainly during day time. Interestingly, the lowest levels while awake (around 0300 h) were near sleep levels. The shut-off during sleep provides an explanation for the results of a preceding series of experiments in which adrenaline excretion was shown to exhibit a pronounced circadian rhythm over several days of sleep deprivation, while sleep, when allowed at night, emphasized the rhythm by reducing the trough further, and day time sleep abolished it by reducing the peak (cf. Åkerstedt, 1979). The fact that, in the present study, the lowest excretion values during waking reached levels (at night) seen during sleep suggests that body position probably is not the major component of the low excretion during sleep (cf. also Reinberg, Ghata, Halberg, Gervais, Abulker, Dupont, & Gaudeau, 1970).

The excretion of noradrenaline did not exhibit any rhythmicity neither during waking, nor during sleep, the latter being significantly reduced at all times. Apparently, the pronounced circadian rhythm seen under conditions of normal or phase shifted sleep/wake alternation (Åkerstedt, 1979) is due to the sizeable reduction during sleep, either due to sleep or to lying down, or to both (Reinberg, 1970; Sundin, 1956, 1958).

As expected, the circadian pattern of cortisol excretion was pronounced both during waking and sleeping. This agrees with many studies of day and night sleep (cf. review by Weitzman et al., 1975). Melatonin excretion also showed a pronounced circadian pattern, with even less effect of sleep, which is similar to that seen for normal night or day sleep (Lynch, Wurtman, Moskowitz, Archer, & Ho, 1975; Jimerson, Lynch, Post, Wurtman, & Bunney, 1977; Lynch, Jimerson, Ozaki, Post, Bunney, & Wurtman, 1978; Åkerstedt, Fröberg, Friberg, & Wetterberg, 1979). Apparently the basic melatonin excretion rhythm is very persistent even when subjected to sleep/wake alterations.

Body temperature was measured continuously and averaged hourly. Figure 6 shows the mean temperatures surrounding each bedtime, plus/minus four hours.

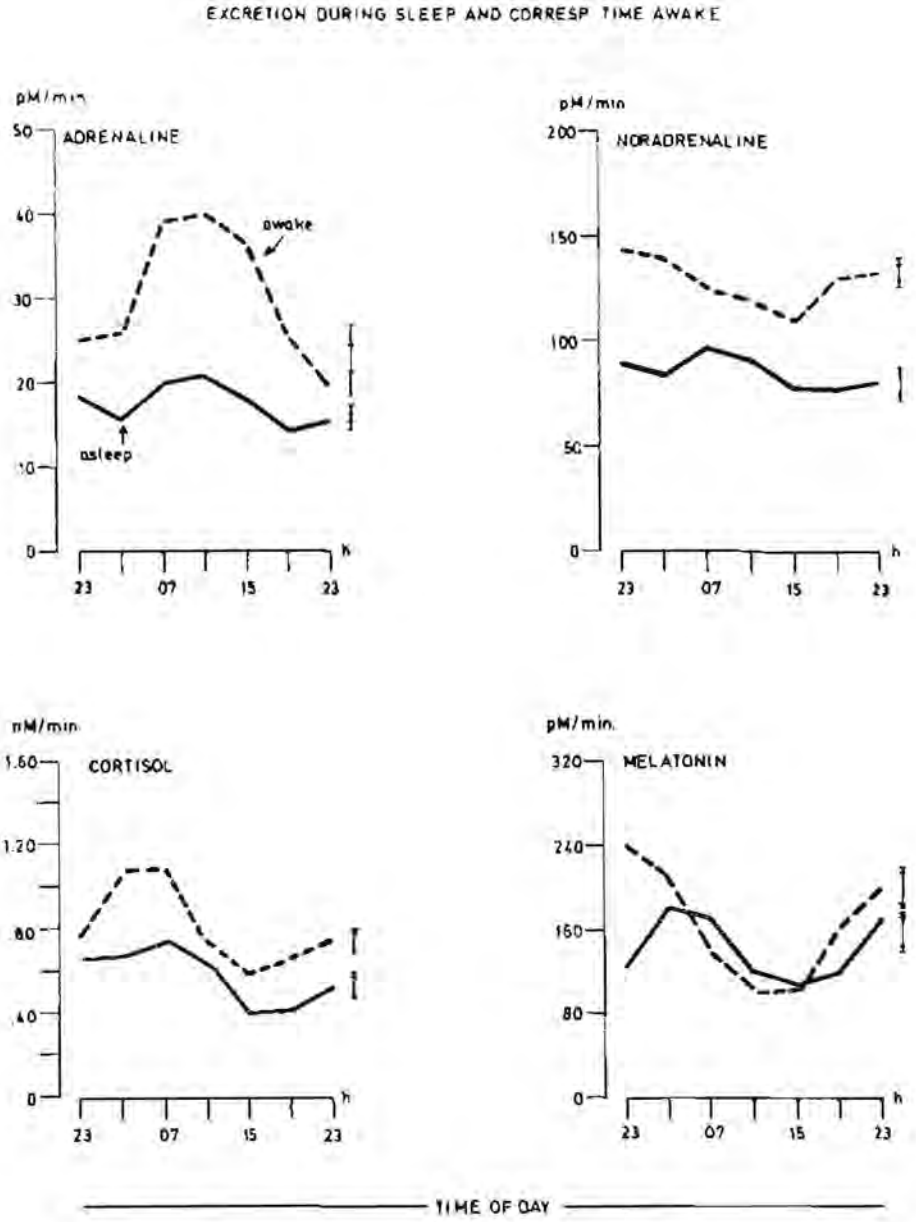

Figure 4. Mean excretion values during sleep for a group of 6 subjects sleeping at 7 different times of day. Also excretion values computed for the same periods during waking. Values plotted at bedtimes. S.E. as in preceding figure.

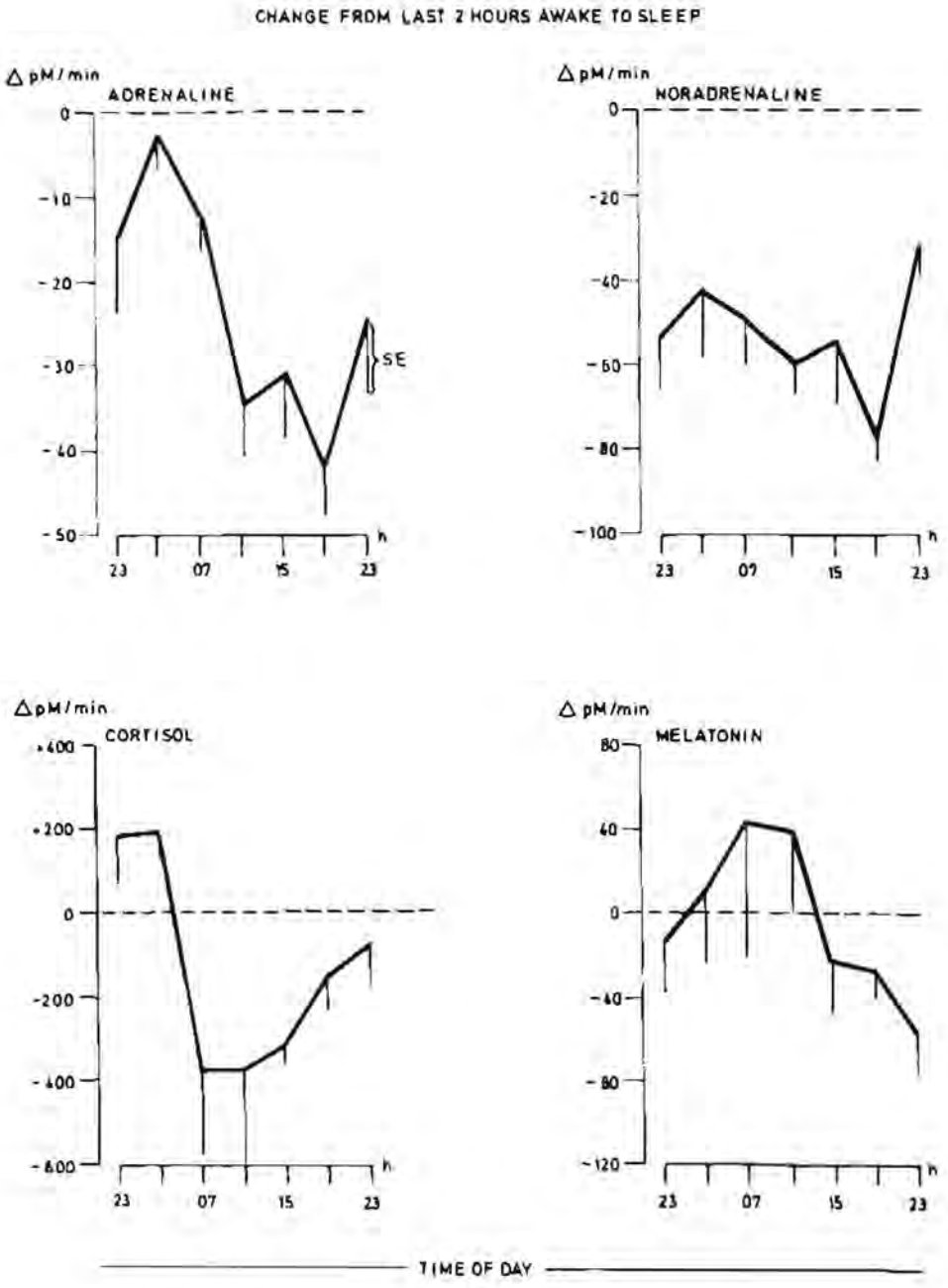

Figure 5. Mean change of excretion from last 2 hours awake to sleep for a group of 6 subjects sleeping at 7 different times of day. Values plotted at bedtimes. S.E. as in preceding figure.

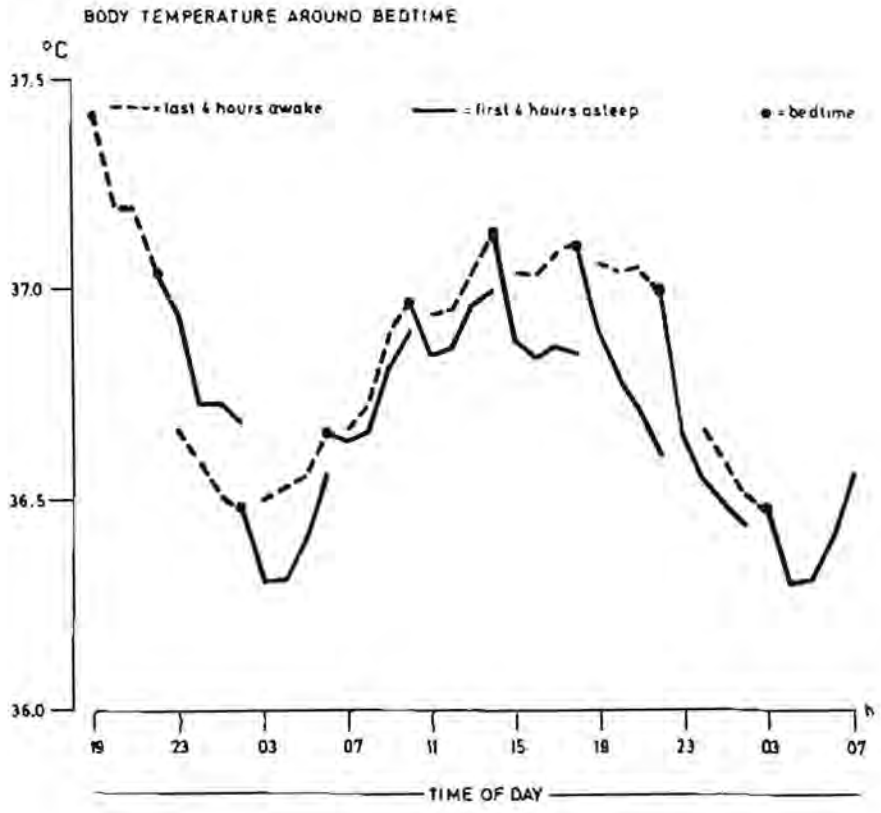

Figure 6. Rectal temperature during last 4 hours awake and first 4 hours asleep. Means for a group of 6 subjects sleeping at 7 different times of day. Second bedtime values (0300 h) repeated.

From the figure it is obvious that body temperature development during sleep is a direct function of circadian phase. Excluding the moderate initial drop, body temperature closely followed the usual pattern during wakefulness, i.e., morning and noon bedtimes were characterized by rising temperatures while evening temperatures fell. The results are similar to those of Mills, Minors, and Waterhouse (1978) in their study of split sleep periods, but cover the full nychthemeron.

Self-rated sleepiness showed a very pronounced circadian pattern peaking in the early morning (Figure 7). This pattern is in good agreement with several previous studies of sleep deprivation (Fröberg, Karlsson, Levi, & Lidberg, 1975a, b; Åkerstedt & Fröberg, 1977; Åkerstedt et al., 1979). Not surprisingly, ratings immediately after rising (before contact with any synchronizers) indicated greater alertness compared with corresponding times during the vigil. Of greater interest is the fact that sleepiness on rising followed approximately the same (significant) pattern as during the vigil. This suggests that an arousal rhythm continues to run also during sleep.

Phase Relations Between Sleep and Other Variables

With access to the present type of data it is tempting to compare sleep characteristics at different phases of the circadian cycle to the different arousal-related rhythms. Studying covariations certainly does not yield any evidence of causation, but it may generate new hypotheses or support old ones. Figure 8 summarizes the analyses. In the figure total sleep length has been plotted at the time of awakening, while for the other variables condition G (plotted twice) was used to represent the circadian cycle during waking.

From inspection of Figure 8 the general impression is that sleep is short when placed on the ascending portion of alertness, body temperature, and adrenaline rhythms, and the descending portion of the melatonin rhythm. The relationship with the cortisol rhythm is less apparent, while we refrain from interpreting the relation to noradrenaline as it failed to show a significant variation over time.

The pronounced circadian pattern of sleep length in the present study suggests regulation by an underlying oscillation of arousal (cf. Webb, 1971). This is supported by the close relation to sleepiness ratings during waking and upon awakening. Thus, awakening does not merely depend on having completed some sleep process of "restorative" value but also on the fact that a phase of the arousal cycle has been reached which does not seem to allow continued sleep. In any case, there is an interesting link between the psychological arousal rhythm and the outcome of sleep attempts at different times of the nychthemeron.

Also, the temperature rhythm is highly predictive of sleep characteristics (and of sleepiness). On the whole, sleep during the rising portion of the rhythm is greatly shortened, while sleep on the descending portion is lengthened. This is in close agreement with the other types of data on the relation between body temperature and sleep cited previously.

The excretion of adrenaline is a well established indicator of psychological arousal (cf. Frankenhaeuser, 1975). Essentially this fits in with the

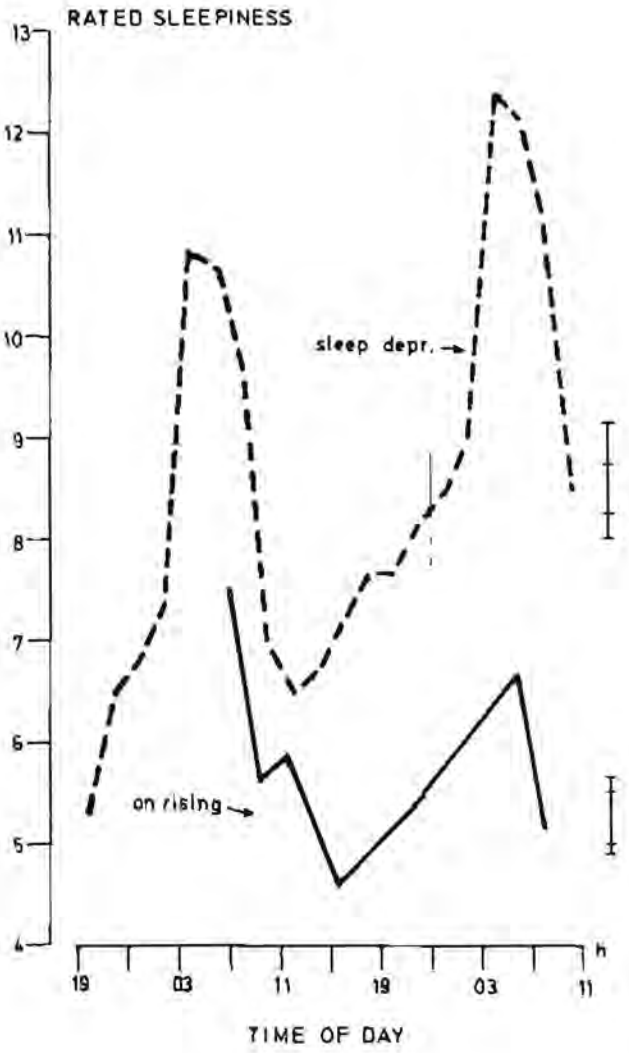

Figure 7. Self-rated sleepiness plotted 2-hourly during waking (broken line) and immediately after rising (continuous line) after each sleep condition. Ratings during waking were obtained from sleep condition G. To cover the whole period necessary the curve has been plotted twice. Ratings ranged between "extremely alert" = 1 and "extremely sleepy" =13. Means for a group of 6 subjects. S.E. as in preceding figures.

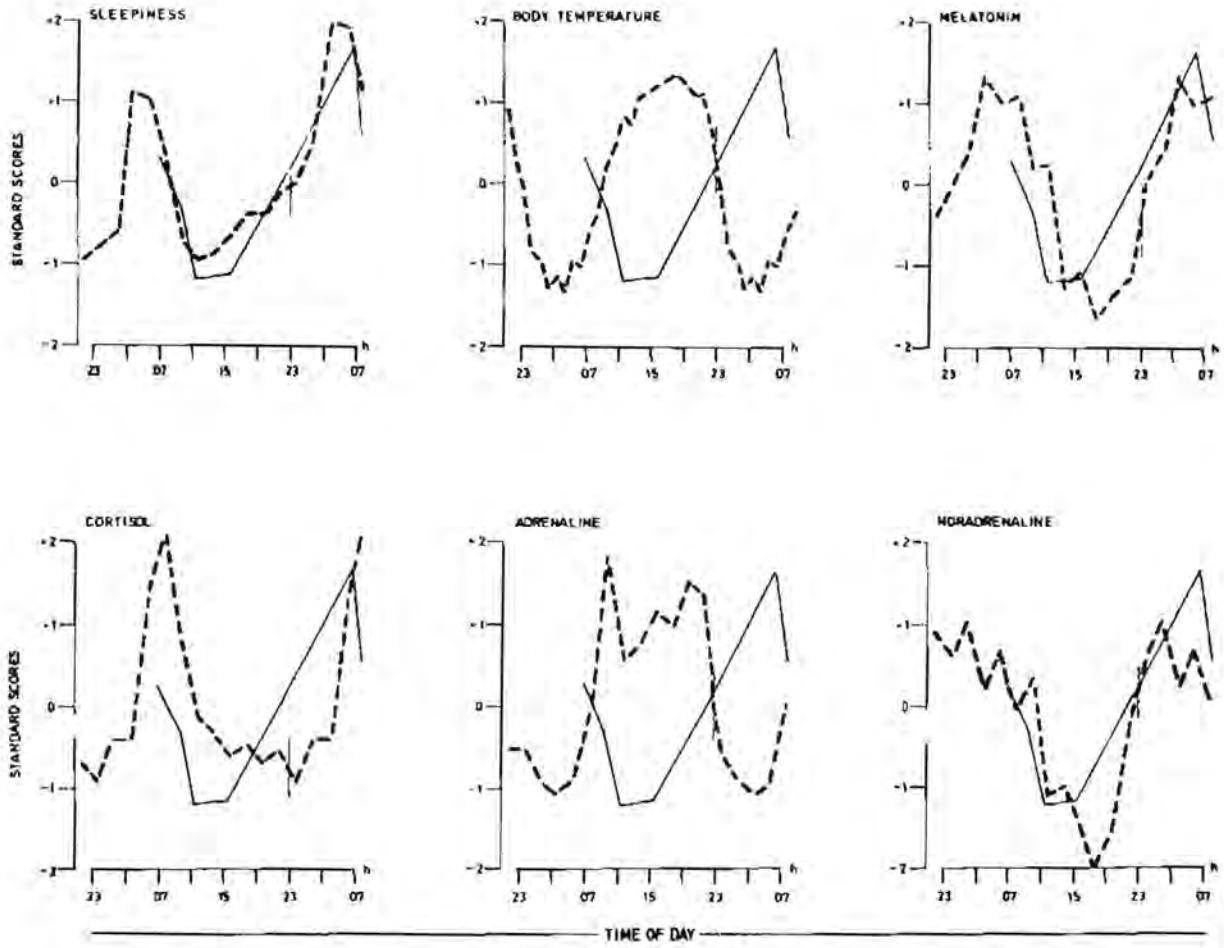

Figure 8. Sleep length plotted at the time of rising together with 2- or 1-hourly values from condition G for self-ratings, rectal temperature, and hormone excretion. All values from condition G have been plotted twice to cover the same time as the sleep length curve. Means for 6 subjects given as standard scores.

pronounced rhythmicity during waking and the lack of rhythmicity for sleep excretion. The results suggest that the connection with the central pacemaker is blocked during sleep and that the circadian rhythm of adrenaline excretion may be due to a passive dependence on some mediating rhythm.

The pineal has been suggested to be a major "tranquillizing" organ (Romijn, 1978), among other effects inducing sleep (Anton-Tay, Diaz, & Fernandez-Guardiola, 1971; Cramer, Rudolph, Consbruch, & Kendel, 1974). The high levels during long night sleep and low levels during short day sleep agree with such an interpretation. However, infusion experiments have been negative as well, failing to induce sleep (Wetterberg, 1978). For cortisol, the covariation is not as apparent as for the preceding variables. While awakening from night time sleep could be associated with the "preparatory" cortisol peak in the early morning, this peak, then obviously is not related to awakening from morning or noon sleep.

Conclusions

To conclude, it appears that sleep characteristics to a large extent are a a direct function of the circadian phase of an underlying arousal rhythm. Regardless of the particular functions responsible for the sleep rhythm, variables such as self-rated alertness, body temperature, melatonin or adrenaline excretion may predict sleep characteristics, at least on group level. Extended studies of the effects on sleep of manipulations of rhythmicity of various functions should eventually identify the major circadian determinants of sleep characteristics and possibly also part of the function of sleep. However, it is apparent that particular attention has to be paid to "masking" effects, perhaps by introducing the "constant conditions test" as suggested by Mills et al. (1978).

For work/rest scheduling the results clearly indicate that the sleep environment (housing, etc.) cannot be a major causative factor in the shift workers' sleep disturbances. Rather, sleep at day time is interfered with by internal factors. Clearly, there are certain portions of the nychthemeron which are suited for sleep while others are not. Possibly, simple self-ratings or body temperature registration could be used to predict outcomes of sleep attempts at different parts of the nychthemeron and be used as tools/criteria in designing work schedules and identifying reasons for maladjustment to shift work. However, there is also a need for extending the present type of study to the effects of sleep at different times of day on subsequent functioning and wellbeing; i.e.,is day sleep inferior to night sleep? If so, can poor day sleep be compensated through naps? What are the long term consequences of manipulations of sleep/wake schedules?

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