# POLYPHASIC SLEEP/WAKE PATTERNS AND THEIR SIGNIFICANCE TO VIGILANCE (\*)

Jürgen Zulley and Josef Bailer Max Planck Institute for Psychiatry, Munich, F.R.G.

# SUMMARY

Under conditions in which no restraints on sleep and minimized alternatives to sleep are given, the sleep/wake pattern exhibits a polyphasic distribution. This result was found in a study in which 12 subjects were polygraphically recorded in two sessions, each lasting 32 hours. During one session, the subjects had constant bedrest for the entire period, while the other session started with 8 hours of sleep deprivation, followed by constant bedrest. During the day, subjects slept at average intervals of 4 hours. After sleep deprivation, the recovery sleep covered the time interval of the first two nap episodes of the condition without sleep deprivation. Compared to baseline alertness, mood and performance are decreased during these two conditions. This result suggests the presence of an underlying polyphasic placement of sleep and wakefulness. Under the given condition, the realization of such a sleep/wake pattern leads to oversleeping, which results in a deterioration in the psychological variables.

# Introduction

In numerous studies (e.g. Wever, 1979) it has been concluded that the human sleep/wake system is governed by a circadian clock mechanism. Such an underlying regulation has also been assumed in studies which focused on the influence of the sleep/wake pattern on performance (Taub & Berger, 1973). Other studies in this context have analyzed the psychological consequences of restricted sleep (Mullaney et al., 1983), the recuperative function of a nap after sleep deprivation (Naitoh, 1981) and the influence of additional sleep given as scheduled naps (Dinges et al., 1981; Godbout & Montplaisir, 1986).

<sup>(\*)</sup> Dedicated to Prof.Dr. D. v. Zerssen on the occasion of his 60th birthday

The initial experiments only considered circadian aspects of sleep and wakefulness. Therefore, interfering influences of the circadian pattern were excluded. Additional sleep episodes (naps) are such influences. By giving the experimental instruction "Please avoid napping", such sleep episodes were suppressed (Wever, 1979; p. 52). If subjects slept in spite of the instructions, these sleep episodes were not considered as sleep in subsequent data analysis.

Recently it has been shown that the assumption of a monophasic circadian sleep/wake modulation has to be questioned (Campbell & Zulley, 1985). Simply by removing the instruction "Please avoid napping", the frequency of napping increases remarkably (Webb & Agnew, 1974). Nevertheless, experimental instructions are not the only factor which can influence the sequence of sleeping and waking. Self-imposed behavior of the subjects also seems to be effective. Subjects who get tired but do not want to go to sleep can choose an alternative behavior to sleep (i.e. drinking a cup of coffee or doing exercise).

These aspects led to experiments where the occurrence of spontaneous sleep was favored. In these experiments no instructions were given as to when or when not to sleep. Furthermore, the subjects had minimized behavioral alternatives to sleep. In such a "disentrained" environment a clear bimodal distribution of sleep and wakefulness could be shown within the circadian day (Campbell & Zulley, 1985). This supports the assumption that the human sleep/wake system is characterized not only by a strong propensity to obtain a sleep episode once per circadian day, but also by a prominent tendency for a shorter sleep episode to occur halfway between successive "night sleep" episodes. This phase position would correspond to the early afternoon, a time when the vast majority of napping occurs in daily life (Dinges, 1983; Ogunremi, 1978; Okudeira, 1983; Webb, 1978).

This finding has been interpreted by assuming that a decrease in a putative sleep threshold reveals a second, less robust phase position for sleep. In real life such a decrease in the sleep threshold may be due to life-style (students, geriatric population). In research such a decrease may be caused by experimental conditions (no instructions about when or when not to sleep; minimized behavioral alternatives to sleep).

A reduction of motor activity caused by constant bedrest would additionally decrease such a putative sleep threshold. In such a study it was shown that napping occurs more frequently, leading to the assumption of a

polyphasic distribution of sleep and wakefulness in the circadian day (Campbell, 1984). Nevertheless, the link between the sleep/wake pattern and mood, alertness and performance has not yet been examined under such conditions. Therefore, the question arises to what extent a spontaneous polyphasic expression of sleep and wakefulness would influence the course of mood, alertness and performance.

## Methods

Twelve healthy volunteers (7 females, 5 males; ages 17-46) participated in this study. Each subject underwent two sessions in the sleep laboratory, each lasting 32 hours, starting at 23:00. One session began with constant bedrest (condition S). The other session comprised of an 8-hour period of total sleep deprivation followed by constant bedrest for the remaining 24 hours (condition TSD). In constant bedrest the subjects had to constantly lie in bed, where they could take their snacks as they wished.

The control of the dim illumination in the room was at the discretion of the subject, who had no knowlege of the time of day. Subjects had no possibility to read, write, listen to music, or exercise. It was possible to communicate with subjects (e.g. regarding experimental instructions) by an intercom system.

The following measurements were recorded continuously: EEG, EOG, EMG, rectal temperature and wrist activity.

Subjects were asked to estimate their mood and alertness hourly on separate 100 mm visual analogue scales (alertness: 0=drowsy, 100=alert; mood: 0=negative, 100=positive). At the same time a performance test (Test d2: concentration speed test, Brickenkamp 1975) was carried out.

The sequence of the two conditions (S; TSD) was randomized. Before each session, each subject underwent baseline measurements in which he estimated his mood and alertness and carried out the performance tests hourly during his normal daily life.

The criterion used here to define a sleep episode was: a sequence of successive uninterrupted 6-minute intervals of polygraphically recorded sleep if these were not interrupted by sequences of wakefulness lasting longer than 30 min.

A major sleep episode (MSE) was defined as any sleep episode initiated in the night phase (20:00 - 7:00). A nap was defined as any sleep episode initiated between 7:00 and 20:00.

#### Results

# Overall sleep/wake pattern

# Condition S

In this session, the subjects slept an average of  $18.8 \pm 1.8$  hours (59%). The distribution of sleep over the day and the nights can be seen in Table 1. It is shown that the relative amount of sleep taken during the day was about half of the amount taken during the nights. Furthermore, compared to the first night, the amount of sleep was slightly reduced in the second night.

On average, the first major sleep episode (MSE) started at  $23:54 \pm 24$  min while the second MSE was initiated at  $23:16 \pm 88$  min. Sleep onset latency in the first MSE had a mean of  $28.0 \pm 16$  min. The duration of the sleep episodes throughout the entire experiment showed a clear circadian variation with long sleep episodes initiated during the first night, short sleep episodes initiated during the day and again long sleep episodes initiated in the second night (Table 2). The waking episodes (only those longer then 30 min) were longer when initiated during the day than during the night (Table 2).

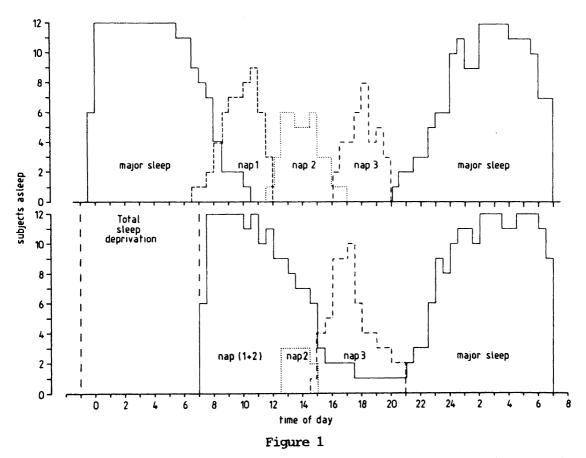
The frequency of napping (sleep episodes initiated between 7:00 and 20:00) per subject showed a clear peak with 3 naps (6 subjects), while 3 subjects took 2 naps, 2 subjects took 5 naps, and one subject took 6 naps. With respect to the placement of the most frequent 3-nap structure, it was possible to clearly differentiate their time of occurrence. All first naps (nap 1) were taken between 7:00 and 12:00 with a mean at 9:30. All second naps (nap 2) were taken between 11:30 and 16:30 with a mean at 13:45. All third naps (nap 3) were taken between 16:00 and 20:00 with a mean at 18:15. The placement of these maps is used in the following for the differentiation of all naps. The upper part of Figure 1 shows a summation histogram of the number of subjects as leep with a differentiation into different groups of naps. The three groups of naps can be seen to be between two MSEs. The phase positions of the nap groups and the amount of sleep within these groups can be seen in Table 3. The placement of the overall groups of naps is nearly identical to the placement of naps by those subjects showing the 3-nap structure.

#### Condition TSD

One subject was excluded from the following data analysis because he slept continuously for 24 hours with the exception of one interruption

(intervening wakefulness longer than 30 minutes). In this session, the other subjects slept an average of  $17.5 \pm 2.3$  hours (73%). The sleep was distributed over the day and the night with more absolute amount of sleep during the day than during the night (Table 4).

The onset of the first sleep episode after TSD (recovery sleep) was at 7:17  $\pm$  14 min, while the MSE began at 23:27  $\pm$  75 min in the following night. Sleep onset latency in the recovery sleep was 6.0 min  $\pm$  5.4 min. The duration of the sleep episodes showed long sleep episodes after TSD (recovery sleep), shorter episodes during the rest of the day and long sleep episodes again in the night phase (Table 5).



Summation histogram of the number of subjects asleep over the entire experimental episode in local time. Upper part: condition S; lower part: condition TSD. Sleep episodes are differentiated with respect to a 3-nap structure (see text).

<u>Table 1.</u> Amount of sleep (mean $\pm$ SD) in each phase in absolute (hours)	and
relative values (percentage of each phase) under condition S.	

Phase	Time Span	Mean amount of sleep		
		hours	8	
1st night	23:15 - 07:00	6.6 ± 0.5	85	
Day	07:01 - 23:00	6.2 ± 1.5	39	
2nd night	23:01 - 07:00	6.0 ± 0.8	75	

<u>Table 2.</u> Duration and frequency of sleep and waking episodes (mean **±** SD) initiated in the respective phase under condition S.

Phase	Time span	Duration of			
		Sleep episodes		Wake episo	des
		hours	n	hours	n
Night	23:00 - 07:00	8.1 ± 1.5	12		0
Day	07:00 - 20:00	1.5 ± 1.0	41	2.3 ± 1.6	53
Night	20:00 - 07:00	7.3 ± 1.7	17	$1.1 \pm 0.4$	5

	Mean phase position	Hours	*
Nap 1	10:00	1.9 ± 0.6	41
Nap 2	14:00	1.9 ± 0.7	30
Nap 3	18:00	$1.3 \pm 1.0$	38

<u>Table 3.</u> Mean position of each nap phase and mean amount of sleep in each nap phase (hours: mean  $\pm$  SD; %: mean) under condition S.

<u>Table 4.</u> Amount of sleep in each phase in absolute (mean  $\pm$  SD) and relative values (mean percent of each phase) under condition TSD.

Phase	Time span	Amount of sleep	
		hours	*
Day	07:00 - 23:00	10.6 ± 1.7	66
Night	23:00 - 07:00	6.9 ± 0.8	86

<u>Table 5.</u> Duration and frequency of sleep and waking episodes (mean  $\pm$  SD) initiated in the respective phase under condition TSD.

Phase	Time span	Duration of			
		Sleep episode	Wake episode	es n	
Morning	07:00 - 08:00	6.7 ± 2.0	11	andan - Calaberry a try may be the second provided and	0
Day	09:00 - 20:00	$2.0 \pm 1.4$	19	1.8 ± 1.7	27
Night	20:00 - 07:00	6.0 ± 3.3	13	2.6 ± 2.7	8

With respect to the placement of sleep, Figure 1 (lower part) shows that the first sleep episode after TSD (recovery sleep) covers the time of nap 1 and nap 2, while nap 3 can again be clearly distinguished, occurring at about the same time as under condition S (mean 17:45; range 14:30 - 21:00). The relative amount of sleep in this time span is similar to the amount in the respective time span under condition S (i.e. 40%). Under both conditions, the MSE in the second night does not differ with respect to placement and duration (Table 5).

#### Alertness and mood

For these scales nonparametric statistics were necessary because the distribution showed significant deviations from the normal distribution. Under the baseline condition the subjects hourly estimated their subjective alertness with a median of 67 and mood with a median of 74 on a visual analogue scale. Comparing the conditions, the values decreased under condition S for both variables with a median of 55 for alertness and 62 for mood.

On average, the subjects felt more drowsy and less comfortable under the bedrest condition.

With regard to the diurnal variation after the respective sleep episodes under condition S, alertness shows a steady increase after each succeeding nap. Mood, after an initial increase, decreases after the first nap.

After sleep deprivation, alertness and mood show the same mean values during bedrest as under condition S with a median of 54 for alertness and 63 for mood.

The diurnal variation after TSD (following the recovery sleep) is equivalent to that of condition S, with a steady increase in alertness and a decrease in mood after the initial increase. It can be summarized that TSD initially caused a decrease in alertness and mood, which was then compensated after the recovery sleep episode.

# Performance

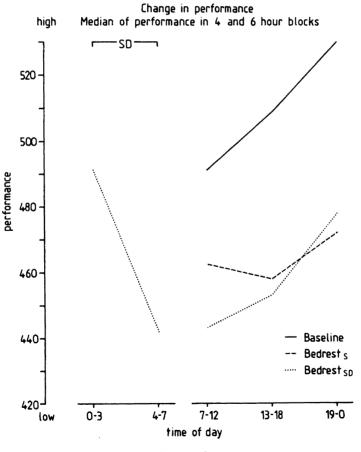
Under both experimental conditions subjects showed a decrease in their performance test compared to the baseline condition. As can be seen in Figure 2, the highest values were observed under the baseline condition, showing a steady increase throughout the day. Condition S showed significantly lower values with a trough in the afternoon. Sleep deprivation caused a dramatic decrease in performance, which recovered throughout the bedrest episode (condition TSD), reaching the same value as under condition S.

A more detailed analysis shows that under condition S, the first nap had

a beneficial effect, showing the highest value followed by a steady decrease after each succeding nap. Under condition TSD, a further decrease in performance became obvious. This sleep deprivation effect is compensated during the course of the bedrest condition.

# Discussion

Under conditions allowing spontaneous expression of the sequence of sleeping and waking, a polyphasic distribution of the occurrence of sleep becomes obvious. Under these conditions, the placement of sleep shows preferred phase positions throughout the day besides major sleep episodes in the night phase. The periodic occurrence of day sleep at intervals of about 4 hours has also been found in earlier studies. Nakagawa (1980) reported a sleep/waking cycle length of approximately 4 hours for subjects confined to





Performance measurements in the three conditions and during sleep deprivation. Values (median) are averaged over 4 and 6-hour blocks.

# ZULLEY & BAILER

bed for 10-12 hours during the day. In experiments without time cues, when subjects showed internal desynchronization of rhythms, several peaks in the distribution of subjective night sleep could be observed during the circadian day (Zulley and Wever, 1982). These peaks had a mean interval of 5.74 hours. In a study where subjects were recorded during 60 hours of enforced bedrest, the median day phase sleep/waking cycle length was 4.65 hours (Campbell, 1984).

These results support the assumption that the human sleep/wake system is more accurately described by a polyphasic distribution with a 4-hour period. The actual frequency of napping seems to depend on the degree to which the conditions allow such napping and on the presence of behavioral options to sleep. This aspect can be described by a putative sleep threshold which is decreased under the above mentioned experiments. This sleep threshold has been raised experimentally by allowing more behavioral options to sleep (Campbell & Zulley, 1985). In this study, subjects showed a clearly bimodal distribution of sleep and wakefulness. The nap peak occurred at about the same time as map 2 in the present experiment. In other words, map 1 and map 3, the two adjacent phase positions for sleep, were masked by the decrease of sleep propensity while map 2 still remained. A possible interpretation of this is that, by raising the sleep threshold, the two less robust phase positions for sleep were diminished, while the more robust phase position (nap 2) can still be seen, placed halfway between the two major sleep episodes. Such a bimodal distribution can also be seen in experiments in which no instructions regarding sleep were given or when subjects napped in spite of the instructions (Zulley & Campbell, 1985). If, in addition, the propensity for sleep is still decreased by experimental instructions, the sleep/wake pattern becomes monophasic. This result can be seen in experiments where napping is effectively suppressed by instructions (Wever, 1979). The result that some subjects show a monophasic sleep placement, even when napping is allowed, could be explained by the assumption that the sleep threshold shows strong individual differences. This results in the phenomenon that, at a given sleep threshold (when napping is allowed), some subjects feel unable to take a nap (Zulley & Campbell; in preparation).

This variation in the degree of sleep propensity can also be seen in daily life. In newborns, Meier-Koll (1979) showed "that the ultradian 4-hour rhythm established already at birth does not disappear during the first month of life".

It can be assumed that, in newborns, the sleep threshold is decreased and that, with aging, the threshold increases. This assumption is supported by the finding that, in children, the polyphasic pattern becomes biphasic, which in our western culture is replaced by a monophasic pattern in adults. This hypothesis is also supported by studies which show that, in other cultures with less structured sociocultural conditions, a biphasic pattern still remains (Soldatos et al., 1983). In the geriatric population the biphasic pattern reappears, assuming a decrease of the sleep threshold with age. This pattern can become polyphasic as has been shown in demented patients (Spiegel et al., 1985). In all these cases, the different preferred phase positions for sleep correspond to those positions found in the present experiment.

Thus, it can be concluded that the human sleep/wake system is basically expressed by polyphasic distribution of sleep and wakefulness with a period of about 4 hours. Yet, it depends on the environmental conditions and on self-imposed behavioral controls to which extent the separate peaks of sleep propensity become obvious. By decreasing the sleep threshold, a monophasic circadian pattern changes into a biphasic (about 12 hours) pattern, and a further decrease reveals a polyphasic (about 4 hours) pattern.

In our daily life, we are able to function well without napping. This does not diminish the relevance of naps in understanding the nature and functions of sleep. It might be assumed that variations in the sleep threshold partially have the function of adapting to environmental and individual needs. This can clearly be seen in childhood and old age. In this sense, the present study forced the subjects to sleep more than they actually needed. The consequence was a decrease in alertness and mood as well as in performance. Such effects can also be seen in cases where subjects oversleep (Taub et al., 1971). In condition S it is obvious that enforced bedrest after a normal night sleep causes oversleeping. In condition TSD it can be assumed that, after the first long sleep episode, the subjects had recovered from sleep deprivation and that the subsequent sleep again caused oversleeping. Vigilance is negatively influenced by oversleeping (Taub et al., 1971). This was also seen in the present study. In daily life such oversleeping is limited by social and occupational pressure, keeping not only an effective time schedule but also more efficient vigilance.

If, however, the normal amount or sequence of sleeping and waking cannot be continued, a significantly beneficial effect of napping has been found

(Dinges et al., 1986). By assuming a polyphasic organization of sleep and wakefulness, it can be hypothesized that a scheduling of sleep and wakefulness with regard to the underlying sleep propensity may be beneficial under circumstances which do not allow a normal amount of sleep.

# References

- Brickenkamp, (1975). <u>Test d2</u>, <u>Aufmerksamkeits-Belastungs-Test</u>, <u>Handan-</u> weisung. Göttingen: Hogrefe.
- Campbell, S. S. (1984). Duration and placement of sleep in a "disentrained" environment. <u>Psychophysiology</u>, <u>21</u>, 106-113.
- Campbell, S. S., and Zulley, J. (1985). Ultradian components of human sleep/ wake patterns during disentrainment. In: H. Schulz and P. Lavie (Eds.). <u>Ultradian Rhythms in Physiology and Behavior. Experimental Brain</u> <u>Research, Suppl. 12</u>, (pp. 234-255). Berlin: Springer.
- Dinges, D. F., Orne, E. C., Evans, F. J., and Orne, M. T. (1981). Performance after naps in sleep-conductive and alerting environments. In: L. C. Johnson, D. I. Tepar, W. P. Colquhoun and M. P. Colligan (Eds.). <u>Biological Rhythms, Sleep and Shift Work</u>, (pp. 539-552). New York: Spectrum Press.
- Dinges, D. F., Orne, E. C., and Orne, M. T. (1983). <u>Napping in North Ameri-</u> <u>ca: a siesta rhythm?</u>. Symposium Abstracts, 4th International Congress of Sleep Research, 86.
- Dinges, D. F., Orne, M. T., Whitehouse, W. G., and Orne, E. C. (1986). Napping to sustain performance and mood: Effects of circadian phase and sleep loss. Sleep Research, 15, 214.
- Gillberg, M., and Akerstedt, T. (1983). Effect of a 1-hour nap on performance following restricted sleep. In: W. P. Koella (Ed.). <u>Sleep 1982.</u> <u>6th European Congress of Sleep Research, Zürich 1982</u>, (pp. 392-394). Basel: Karger.
- Godbout, R., and Montplaisir, J. (1986). The performance of normal subjects on days with and days without naps. <u>Sleep Research, 15</u>, 71.
- Lubin, A., Hord, D. J., Tracy, M. L., and Johnson, L. C. (1976). Effects of exercise, bedrest and napping on performance decrement during 40 hours. Psychophysiology, 13, 334-339.
- Meier-Koll, A. (1979). Interaction of endogenous rhythms during postnatal development. International Journal of Chronobiology, 6, 179-189.
- Mullaney, D. J., Kripke, D. F., and Fleck, P. A. (1983). Sleep loss and nap

effects on sustained continuous performance. <u>Psychophysiology</u>, 20, 643-651.

- Naitoh, P. (1981). Circadian cycles and restorative power on naps. In: L. C. Johnson, D. I. Tepar, W. P. Colquhoun and M. P. Colligan (Eds.). <u>Biological Rhythms, Sleep and Shift Work</u>, (pp. 553-580). New York: Spectrum Press.
- Nakagawa, Y. (1980). Continuous observation of EEG patterns at night and in daytime of normal subjects under restrained conditions. I. Quiescent state when lying down. <u>Electroencephalography and Clinical Neurophysiology</u>, 49, 524-537.
- Ogunremi, O. (1978). The subjective sleep pattern in Nigeria. <u>Sleep</u> <u>Research, 7</u>, 163.
- Okudaira, N., Fukuda, H., Ohtani, K., Nishihara, K., Endo, S., and Torii, S. (1983). <u>Napping patterns in the elderly in Vilcambamba</u>, <u>Ecuador</u>. Symposium Abstracts, 4th International Congress of Sleep Research, 8.
- Soldatos, C. R., Medianos, M. G., and Vlachonikolis, I. G. (1983). Early afternoon napping: A fading Greek habit. In: W. P. Koella (Ed.). <u>Sleep</u> <u>1982. 6th European Congress of Sleep Research, Zürich 1982</u>, (pp. 202-205). Basel: Karger.
- Spiegel, R., Stähelin, H. B., Seiler, W. O., and Allen, S. R. (1985). Seventy-two hour polygraphic recordings in dementia: Circadian aspects of sleep and behavior. In: W. P. Koella, E. Rüther and H. Schulz (Eds.). Sleep '84, (pp. 439-442). Stuttgart: Gustav Fischer.
- Taub, J. M., Globus, D. D., Phoebus, E., and Drury, R. (1971). Extended sleep and performance. <u>Nature, 223</u>, 142-143.
- Taub, J. M. and Berger, R. J. (1973). Performance and mood following variations in length and timing of sleep. Psychophysiology, 10, 559-570.
- Webb, W. B. (1978). Sleep and naps. <u>Speculations in Scientific Technology</u>, 1, 313-318.
- Webb, W. B., and Agnew, H. W. (1974). Sleep and waking in a time-free environment. <u>Aerospace Medicine, 45</u>, 617-622.
- Wever, R. (1979). The Circadian System of Man. New York: Springer.
- Zulley, J., and Wever, R. (1982). Interaction between the sleep-wake cycle and the rhythm of rectal temperature. In: J. Aschoff, S. Dann, and G. Groos (Eds.). <u>Vertebrate circadian systems</u>, (pp. 253-261). Berlin: Springer.
- Zulley, J., and Campbell, S. S. (1985). Napping behavior during "spontaneous

internal desynchronization": Sleep remains in synchrony with body temperature. <u>Human Neurobiology</u>, 4, 123-126.