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# The Dynamics of the First Sleep Cycle

*Mats Gillberg and †Torbjörn Åkerstedt

**National Defence Research Establishment, Department 5, and †IPM and Stress Research, Karolinska Institute, Stockholm, Sweden*

**Summary:** Eight subjects participated in an experiment in which sleep stages and electroencephalographic (EEG) power density during the first sleep cycles (and where such appeared, also second cycles) were studied in a design involving 8, 4, 2 or 0 hr of progressively postponed night-time sleep. Each of these four manipulations was followed by a day-time sleep beginning at 1100 hr. No significant changes in the duration of the first sleep cycle appeared. As the prior sleep loss increased both SWE (slow-wave energy; accumulated EEG delta power density) and SWA (slow-wave activity; EEG delta power per minute) increased during the 1100-hr sleeps. This was observed for the entire cycles, the nonrapid eye movement (NREM) periods, and the SWS periods, respectively. SWS latency decreased and SWS duration increased, respectively, markedly with prior waking. Also, for the progressively postponed sleeps (started at 2300 hr, 0300 hr, 0500 hr and 1100 hr) there were changes, but not as clear. After 28 hr of continuous waking there was a marked increase of SWA during SWS. Also, at this level there was a spill over of SWA to the second cycle. It is suggested that there might be a limit to the amount and intensity of SWS that can be accommodated in the first sleep cycle and that this limit is reached before the appearance of REM sleep.
**Key Words:** Sleep cycle—Sleep loss—Spectral analysis—Slow-wave energy—Slow-wave activity—Slow-wave sleep.

Slow-wave sleep (SWS) usually decreases progressively across a sleep episode (1–3), and sleep loss causes an increase in the overall level of this process (e.g. 4, 5). Similar observations have been made for the intensity and the energy content of the delta and theta bands (3,6–8). Furthermore, these homeostatic responses seem mainly to be confined to the first part of sleep (3,5,8–10).

Rapid eye movement (REM) sleep, in contrast, does not exhibit the same homeostatic behavior as a response to manipulations of prior waking but is influenced by the circadian rhythm and exhibits a very stable cyclic pattern of occurrence during sleep (11,12). This stable pattern is remarkably intact even after extended periods of prior waking.

As a consequence, when the period of prior waking increases, the resulting increased need for SWS (or delta) activity must in some way adapt to the pattern of sleep cycles. Of special interest is what happens during the first sleep cycle, because it coincides with the most intense SWS/delta activity. Given the high priority of SWS/delta activity after sleep deprivation, it might be expected that the first REM period would be postponed and hence that the first sleep cycle would be longer than subsequent cycles. Under normal sleep–wake conditions the first sleep cycle is actually shorter than the second (13–16). Also, if the period preceding waking is increased by, for example, 24 hr by skipping one night's sleep, the duration of the first sleep cycle or the first nonREM (NREM) period seems to be unaffected (17,18). One study (8) has, however, reported that the first cycle becomes longer after sleep loss. When both circadian phase and preceding waking time varied as naps were systematically shifted across time of day, the duration of the first cycle was found to vary considerably (7).

Three questions then arise. First, how does increased prior wakefulness affect the amount, intensity and dynamics of SWS/delta activity within the first sleep cycle? Second, does the first sleep cycle increase its duration to accommodate the high initial need for SWS/delta activity as prior wakefulness increases? Third, what happens if the cycle does not increase its duration—does SWS/delta activity "spill over" into subsequent cycles? To answer these three questions we reanalyzed the results from a study (5,6) in which recovery sleep was scheduled to 1100 hr after 8, 4, 2 or 0 hr of preceding night sleep. Hence, the material contains data on sleep at the same circadian phase, but with different prior sleep/wake histories, as well as on sleep appearing at successively later circadian phases

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Address correspondence and reprint requests to Mats Gillberg, Department of Stress Research, Karolinska Institute, P.O. Box 60 205, S-104 01 Stockholm, Sweden.



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after different amounts of prior waking. Another important feature of the design is that it includes moderate levels of prior sleep loss at which the relation between loss and recovery is more likely to be linear (19).

## METHODS

*Subjects and procedure.* Eight healthy male subjects (age range 20–47 yr, mean age 33.6 yr) participated on four occasions. They were allowed to sleep 8, 4, 2 or 0 hr during the night. The sleep periods were scheduled at 2300 hr, 0300 hr or 0500 hr, respectively. They were awakened from sleep at 0700 hr. Following each of the four manipulations of the night sleep (including the condition with no sleep during the night) a recovery sleep was scheduled at 1100 hr and spontaneously terminated. In the following text the night sleep periods will be referred to as 823 (i.e. an 8-hr sleep started at 2300 hr, 43 and 25, respectively, and the day sleep periods as 811 (i.e. sleep started at 1100 hr after 8 hr of prior night sleep), 411, 211 and 011, respectively.

The sleep electroencephalogram (EEG) [Cz–Oz, and a bipolar electrooculogram (EOG)] was recorded with portable equipment (Oxford Medilog). The recordings were visually scored according to standard criteria (19) and were also subjected to spectral analysis (Fast Fourier Transform; FFT) (20) to yield spectral power in the delta band (0.5–3.9 Hz), i.e. the frequency band that has been shown to contain the main part of the homeostatic response (3). The EEG was sampled with 34.15 Hz and the spectral analyses were performed for 15-sec epochs, then averaged over 1-min periods. Detailed description of the method and rationale for the design can be found in our two earlier papers (5,6).

*Data analysis.* The first sleep cycle was assumed to begin with uninterrupted stage 1 sleep and to end when the first REM period (of at least 3 min) ended. In a few cases when a sleep-onset REM (SOREM) period occurred, the first cycle was assumed to begin when the SOREM period ended. A SOREM period was defined as a REM period that occurred up to 15 min (21) after sleep onset, without being preceded by SWS.

The delta power density for each minute (in the following referred to as SWA, slow-wave activity) was expressed in relation to the mean SWA of the first sleep cycle of the 823 condition for each subject (set to 100%). All further calculations will be based on these transformed data. SWA integrated over time is referred to as SWE (slow-wave energy). In addition to analyzing the changes as a function of time, we also related the changes to cycle duration, i.e. by using percent of cycle duration as the common time base (the duration of each cycle was set to 100%). For these analyses mean SWA was calculated in 5% bins of each cycle.

From the spectral analysis variables describing the dynamic changes within the first cycles were derived: 1) the slope of the linear regression line fitted to changes in SWA during the first 30 min of NREM; 2) time from sleep onset to the first maximum of the SWA (the maxima were found visually after smoothing the original data with five points); 3) time between the largest fall in SWA and the end of the NREM period (the largest fall was defined as the minimum of the first derivate of the smoothed data); and 4) the "plateau", i.e. the time from first maximum to the largest fall in SWA. The above measures, except the last, have been used earlier by Achermann (22), and in the present paper they serve as computer-derived counterparts to the timing and duration of visually scored SWS.

**TABLE 1.** *Summary of results from two-factor ANOVA of normalized (see text) SWA during the first and last 30 min of NREM and the first 10 min of REM from first sleep cycles*

| | | ANOVA: 823, 43, 25 and 011 | | | | | ANOVA: 011, 211, 411 and 811 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | *F* value | df | Epsilon | Level of significance | Significant pairwise comparison $p < 0.05$ | *F* value | df | Epsilon | Level of significance | Significant pairwise comparison $p < 0.05$ |
| First 30 min NREM | Conditions (C) | 10.95 | 3/21 | 0.55 | <0.01 | 011 > 823; 43; 25 | 25.53 | 3/21 | 0.56 | <0.001 | 011 > 211; 411; 811 |
| | Time (T) | 129.62 | 5/35 | 0.45 | <0.001 | | 48.15 | 5/35 | 0.46 | <0.001 | |
| | Interaction (C × T) | 2.97 | 15/105 | 0.29 | <0.05 | | 7.61 | 15/105 | 0.23 | <0.001 | |
| Last 30 min NREM | Conditions (C) | 4.32 | 3/21 | 0.71 | <0.05 | 823 < 43; 25; 011 | 8.28 | 3/21 | 0.75 | <0.01 | 011 > 411; 811 and 211 > 811 |
| | Time (T) | 6.81 | 5/35 | 0.32 | <0.02 | | 16.64 | 5/35 | 0.38 | <0.001 | |
| | Interaction (C × T) | 1.24 | 15/105 | 0.17 | ns | | 1.11 | 15/105 | 0.16 | ns | |
| First 10 min REM | Conditions (C) | 2.88 | 3/21 | 0.59 | ns | | 9.67 | 3/21 | 0.63 | <0.01 | 011 > 211; 411; 811 |
| | Time (T) | 4.36 | 1/7 | 1.00 | ns | | 0.44 | 1/7 | 1.00 | ns | |
| | Interaction (C × T) | 0.13 | 3/21 | 0.59 | ns | | 0.38 | 3/21 | 0.49 | ns | |

Analyses based on means of 5-min bins. Levels of significance after epsilon correction. Pairwise comparisons tested with the Newman–Keuls method.



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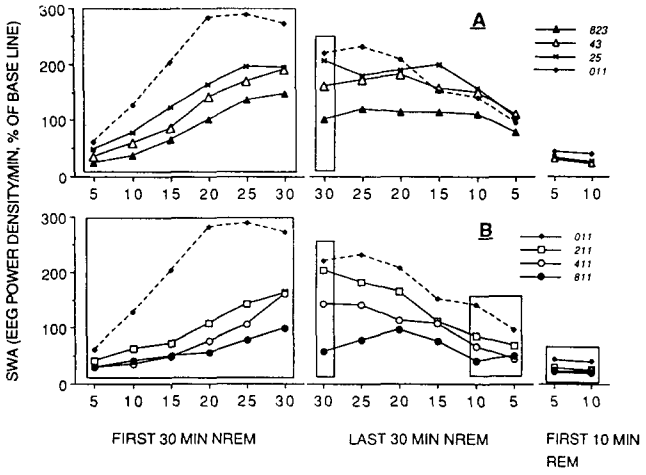


**FIG. 1.** SWA (power density/min) for the first and last 30 min of NREM and the first 10 min of REM, respectively, during the first sleep cycle. SWA expressed relative to mean SWA during the first cycle of the 823 sleep (=100). Means have been calculated for successive 5-min intervals. Top panel (A) shows data from the 823, 43, 25 and 011 conditions and bottom panel (B) from 011, 211, 411 and 811, respectively. Framed ares denote time points at which differences between conditions are significant (simple effects from ANOVA; see Table 1). Means of eight subjects.

To test if there were changes in SWA and visually scored SWS due to prior time awake also in the second sleep cycles, we subjected data from the first and second cycles of the 011, 211 and 823 conditions, respectively, to a two-way analysis of variance (ANOVA) for repeated measurements. By restricting the analysis to these three conditions and to the first 60 min of each cycle we could obtain data from the first and second cycles for all eight subjects. Results from the present experiment support the use of this procedure, since the homeostatic changes, at least during the first cycle, were shown to appear in the first half of the cycle (see below).

Statistical tests were performed on two combinations of conditions, the first combination being the four levels of sleep postponement (823, 43, 25 and 011) and the second being the four sleeps scheduled to start at 1100 hr (011, 211, 411 and 811). Parametric ANOVAs were used in all cases, except for one case where the baseline data were included where instead the Friedman nonparametric ANOVA was used. $F$ tests were performed after epsilon correction (two-way analyses) or using the conservative $F$ test with degrees of freedom $1/n - 1$ (one-way analyses) (23) for all parametric repeated measurements ANOVAs because the requirement of symmetrical variance–covariance matrices could not be met in the present data.

## RESULTS

Figure 1 (for ANOVA, see Table 1) depicts the SWA changes as a function of time for the first and last 30 min of NREM, as well as for the first 10 min of REM.

Data are given as means for successive 5-min periods. Both sets of data, i.e. from the sleeps that were progressively postponed (A) and from the sleeps that were started at 1100 hr (B), showed similar increases over the first 30 min. Most marked was the rapid increase during the 011 condition. Variation between conditions (simple effects) was significant for all time points during the first 30 min for both sets of data (framed area in Fig. 1). An inspection of Table 1 reveals significant interaction during the first 30 min for both sets of data, i.e. the conditions have different developments over time. Pairwise comparisons indicate that the 011 condition differed markedly from the other conditions, whereas there were no significant differences among the latter. During the last 30 min of NREM, we found overall decreasing SWA for all conditions, most apparent for the 11-hr sleeps. There were significant differences between conditions for both sets of data with 823 having a significantly lower level than the other three conditions for postponed sleeps and 011 having a significantly higher level than the other sleeps started at 1100 hr. In addition, the 211 sleep had a higher level of SWA than the 811 sleep. SWA did not change over time during the first 10 min of REM, but the 1100-hr sleeps differed between condi-

**TABLE 2.** *Summary of results from two-factor ANOVA of normalized (see text) SWA during the first sleep cycles*

| | ANOVA: 823, 43, 25 and 011 | | | | | ANOVA: 011, 211, 411 and 811 | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | $F$ value | df | Epsilon | Level of significance | Significant pairwise comparison $p < 0.05$ | $F$ value | df | Epsilon | Level of significance | Significant pairwise comparison $p < 0.05$ |
| Conditions (C) | 2.52 | 3/21 | 0.51 | ns | — | 10.93 | 3/21 | 0.49 | <0.01 | 011 > 211; 411; 811 |
| Time (%) (T) | 8.38 | 19/133 | 0.14 | <0.001 | — | 13.35 | 19/133 | 0.11 | <0.001 | — |
| Interaction (C × T) | 3.32 | 57/399 | 0.05 | <0.05 | — | 3.18 | 57/399 | 0.06 | <0.05 | — |

Duration of cycles set to 100%. Analysis based on means of 5% bins. Levels of significance after epsilon correction. Pairwise comparisons tested with the Newman–Keuls method.



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**TABLE 3.** *Means (and standard errors) of normalized FFT parameters and visually scored parameters during the first sleep cycles*

| | Condition | | | | | | | ANOVA: 823, 43, 25 and 011 | | | ANOVA: 011, 211, 411 and 811 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 823 | 43 | 25 | 011 | 211 | 411 | 811 | *F* values | Level of significance | Significant pairwise comparison $p < 0.05$ | *F* values | Level of significance | Significant pairwise comparison $p < 0.05$ |
| FFT Parameters | | | | | | | | | | | | | |
| SWE C1 | 10618 (1550) | 11715 (1086) | 11474 (758) | 16615 (1642) | 10709 (908) | 7871 (1435) | 6282 (1564) | 4.86 | ns | — | 18.62 | <0.01 | 011 > 211; 411; 811 and 211 > 811 |
| SWA C1 | 100 (0) | 120 (41) | 125.9 (34.8) | 162.3 (47.9) | 108.3 (31.9) | 96.6 (49.8) | 61.5 (40.5) | Friedman ANOVA used | <0.01 | — | 27.27 | <0.01 | 011 > 211; 411; 811 and 211 > 811 |
| SWE NREM | 10185 (1462) | 11226 (1089) | 10839 (837) | 15755 (1504) | 10023 (837) | 6519 (1433) | 5744 (1532) | 4.62 | ns | — | 18.97 | <0.01 | 011 > 211; 411; 811 |
| SWA NREM | 111.6 (3.4) | 144.4 (18.3) | 155.9 (11.4) | 197.3 (23) | 134.3 (12.0) | 101.0 (22.7) | 71.6 (15.3) | 8.94 | <0.02 | 011 > 823; 43 | 20.74 | <0.01 | 011 > 211; 411; 811 and 211 > 811 |
| SWE SWS | 7050 (1206) | 8314 (1121) | 9146 (1342) | 14942 (1179) | 7509 (887) | 4259 (802) | 2952 (1198) | 8.87 | <0.02 | 011 > 823; 43; 25 | 31.96 | <0.001 | 011 > 211; 411; 811 and 211 > 811 |
| SWA SWS | 189 (17.4) | 196.1 (17.3) | 212.7 (13.4) | 292.4 (30.1) | 226.2 (21) | 189.3 (26.9) | 181.1 (28.6) | 11.34 | <0.02 | 011 > 823; 43; 25 | 11.36 | <0.02 | 011 > 211; 411; 811 |
| Slope SWA first 30 min NREM | 5.4 (1.1) | 6.1 (0.9) | 6.0 (0.5) | 9.3 (1.0) | 4.6 (1.0) | 4.6 (0.9) | 2.6 (0.9) | 4.0 | ns | — | 14.84 | <0.01 | 011 > 211; 411; 811 |
| Time to first maximum (min) | 40.0 (6.8) | 33.4 (3.3) | 29.5 (2.1) | 26.9 (4.7) | 36.3 (3.9) | 39 (4.2) | 41.5 (4.2) | 1.81 | ns | — | 2.19 | ns | — |
| Largest fall, time from end NREM (min) | 27.3 (9.8) | 17.4 (9.6) | 8.1 (2.7) | 15.8 (6.2) | 15.8 (6.2) | 15.1 (3.8) | 18.4 (5.6) | 1.64 | ns | — | 0.10 | ns | — |
| Time from first maximum to largest fall | 21.6 (3.7) | 30.25 (6.4) | 31.9 (6.2) | 35.9 (7.3) | 25.9 (5.8) | 12.9 (2.5) | 7.8 (2.5) | 1.14 | ns | — | 7.42 | <0.05 | 011 > 411; 811 |
| Visually scored parameters | | | | | | | | | | | | | |
| SWS latency | 32.2 (6.9) | 27.1 (4.6) | 17.56 (2.0) | 13.1 (1.9) | 24.6 (2.7) | 28.1 (3.4) | 42.1 (3.7) | 3.43 | ns | — | 15.2 | <0.01 | 011 > 411; 811 and 211 > 811 and 411 > 811 |

tions, mainly through higher levels during the 011 condition.

The above analysis of the first sleep cycle does not cover the entire cycle but leaves a gap in the middle. As an alternative approach we analyzed SWA relative to cycle duration. The data are presented in Fig. 2 and the statistical tests in Table 2.

Both sets of data vary significantly with time in cycle. Conditions varied significantly for the 1100 hr day sleeps (B) and, as shown by the pairwise comparisons, this was due mainly to the 011 condition. There were no differences between conditions for the sleep postponement data (A). The interaction between condition and time in cycle was significant for both sets of data. As shown in Fig. 2, the significant differences between conditions (simple effects; framed areas in Fig. 2) ap-



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**TABLE 3.** *Continued.*

| | Condition | | | | | | | ANOVA: 823, 43, 25 and 011 | | | ANOVA: 011, 211, 411 and 811 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 823 | 43 | 25 | 011 | 211 | 411 | 811 | *F* values | Level of signif-icance | Significant pairwise comparison $p < 0.05$ | *F* values | Level of signif-icance | Significant pairwise comparison $p < 0.05$ |
| SWS period (min) | 37.3 (6.3) | 42.4 (6.4) | 43 (7.9) | 51.1 (4.9) | 33.2 (3.4) | 22.5 (2.6) | 16.3 (3.5) | 1.6 | ns | — | 19.8 | <0.01 | 011 > 211; 411; 811 and 211 > 811 |
| End of SWS | 69.6 (10.8) | 69.5 (7.1) | 60.6 (7.9) | 64.2 (6.1) | 57.8 (5.1) | 50.6 (4.5) | 58.4 (4.7) | 0.34 | ns | — | 1.4 | ns | — |
| Second stage 1+2 period (min) | 23.2 (6.1) | 16.1 (8.4) | 10.5 (3.1) | 20.9 (7.6) | 17.7 (2.9) | 17.2 (3.1) | 15.1 (5.2) | 1.27 | ns | — | 0.24 | ns | — |
| REM latency (min) | 92.75 (15.0) | 85.6 (13.6) | 71.1 (6.7) | 85.1 (11.1) | 75.5 (3.4) | 67.8 (5.7) | 73.4 (5.8) | 0.61 | ns | — | 1.06 | ns | — |
| REM period (min) | 16.5 (4.1) | 19.3 (3.9) | 23.8 (8.8) | 20.8 (1.9) | 27.1 (5.9) | 20.7 (5.1) | 24.9 (5.1) | 0.35 | ns | — | 0.49 | ns | — |
| Duration | 109.2 (16.6) | 104.9 (14.9) | 94.9 (7.9) | 105.9 (10.6) | 102.6 (8.8) | 88.4 (8.6) | 98.4 (6.5) | 0.20 | ns | — | 0.69 | ns | — |

Summary of one-factor ANOVAs for repeated measures. Levels of significance after the conservative *F*-test suggested by Winer (df for all tests 1/7). Pairwise comparisons tested with the Scheffé test. The Friedman nonparametric ANOVA was used for the test that included the baseline data (=100).

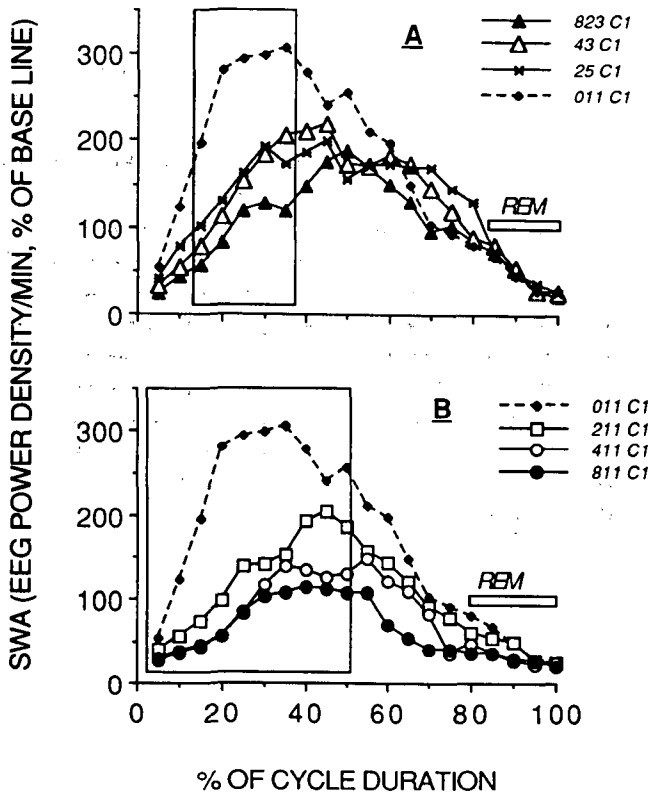


**FIG. 2.** SWA (power density/min) expressed relative to mean SWA during the first cycle of the 823 sleep (=100) and plotted against relative cycle duration. Means have been calculated for successive 5% time intervals. Top panel (A) shows data from the 823, 43, 25 and 011 conditions and bottom panel (B) from 011, 211, 411 and 811, respectively. Framed areas denote time points at which differences between conditions are significant (simple effects from ANOVA; see Table 2). Means of eight subjects.

pear in the first half of the cycle, most prominent for the 1100 hr sleeps. Once again, the main effect derives from the 011 condition.

Whether one studies the changes of power as a function of time or as a function of cycle duration, some common features appear: There was for all conditions a significant initial increase and a significant decrease at the end of the cycle. The first sleep cycle of the 011 sleep differed markedly from the other conditions: SWA increased earlier and reached higher levels.

Table 3 incorporates detailed information on first cycles. SWE and SWA were analyzed for the entire cycle, for NREM and for SWS, respectively. SWE within the first cycle did not change significantly as sleep was postponed, while there was a significant increase in SWA. The results for NREM were similar. When the analysis was restricted to SWS, both SWE and SWA increased with postponement of sleep. Pairwise comparisons show that the high levels during the 011 condition were the main explanation to the significant ANOVAs.

The 1100 hr sleeps differed more clearly among themselves: not only did the 011 condition differ significantly from all the other conditions but also the 211 condition differed from the 811 condition on several variables. Especially strong was the effect for SWE during SWS, i.e. if the *F* value may be taken as an indicator of the size of the effect.

The slope of SWA during the first 30 min differed significantly only for the 1100-hr sleeps. Neither the timing of the first maximum of SWA nor that of the



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largest fall in SWA varied significantly. The time between the first maximum and the largest fall increased significantly with prior sleep restriction for the 1100-hr sleeps.

Finally, Table 3 reveals that the only significant changes in the visually scored parameters were a clear decrease in SWS latency and a clear increase in SWS duration as a function of prior sleep restriction during the 1100-hr sleeps.

In addition to the above analyses we also calculated intra-individual correlations between the duration of the first cycle and SWS latency (as a measure of need for recovery). The mean of these correlation coefficients was 0.21, with the individual coefficients ranging from −0.27 to 0.95. The mean of the correlation coefficients did not differ significantly from zero ($t$ test after transforming the individual coefficients to Fisher's $Z$).

Data on SWA during SWS and duration of SWS from Table 3 are plotted in Fig. 3. Although both variables have the same general trends over conditions, it is apparent that SWA increases more steeply at the highest levels of prior sleep loss.

*Second cycles.* The ANOVA (see Table 4) demonstrated significant variations over conditions as well as between first and second cycles. Pairwise comparisons between first and second cycles (i.e. the first 60 min of each cycle) showed that the first cycle had significantly higher SWA than the second cycle for both the 823 and the 011 conditions. Furthermore, the second cycle of the 011 condition had significantly higher SWA than that of the 823 condition. In fact, SWA was twice as high. The results for SWS during the first 60 min followed a similar pattern, although the only significant pairwise comparison was between the first and second cycles for the 011 condition.

## DISCUSSION

The pattern of the first sleep cycle was clearly related to the prior sleep/wake history. There was an initial increase in SWA during the first cycle, and as sleep was progressively postponed until 1100 hr (i.e. 28 hr) this increase was markedly steeper than for all other sleeps. Toward the end of the cycle there was the expected decrease in SWA, most obvious for the day sleeps. Again, the 28-hr prior waking time induced the strongest effects. Also, when SWA was analyzed relative to cycle duration, similar results emerged: The differences in the dynamics due to the prior sleep/wake conditions were mainly confined to the first half of the cycle, and the most profound changes appeared after 28 hr of continuous waking.

Visually scored SWS increased and SWS latency decreased significantly during the 1100-hr sleeps as prior night-time sleep decreased. Interestingly, the duration of SWS increased mainly by an earlier onset of SWS while the termination remained unchanged. The FFT-derived measures of the onset and duration of SWA (the slope of the first 30 min, the duration of the "plateau" and the timing of the largest fall of SWA) show the same pattern as that of SWS latency, duration of SWS and the timing of the end of SWS. Also, for the other measures the homeostatic responses were pronounced during the 1100-hr sleeps. The differences between conditions were not solely explained by the 011 condition, but there were also significant differences between other conditions, indicating a graded dose–response relationship. SWA and SWE increased for the entire cycle, for the NREM period and for SWS, respectively.

It is obvious from Table 3 that successive postponement of the night sleep from 2300 hr to 0500 hr produced moderate homeostatic responses within the first cycle. Only when sleep was postponed to 1100 hr the following morning did clear responses emerge. The homeostatic effects were mainly accomplished by an increase in intensity, i.e. in SWA. Furthermore, these changes appeared most clearly when the period of study was restricted to visually scored SWS. Neither visually scored SWS latency nor SWS duration varied significantly, even when the 011 data were included in the analyses.

An interesting question is why the night sleeps were less affected by prior waking than the day sleeps. Beersma et al. (24) have shown that an increased REM pressure suppresses NREM intensity. Such an increase in REM pressure might be expected as sleep is postponed toward the morning hours (11,12). In the present data there were, however, no clear signs of increased REM pressure, e.g. decreases in REM latency or increases in REM duration. In addition, recent data have shown that homeostatic responses dominate the first hours of sleep also when sleep is scheduled at different times of day (9) or when the circadian phase is advanced by the influence of bright light (25). A probable explanation for the lack of homeostatic responses, as sleep was postponed from 23 hr to 5 hr (and for some variables was postponed until 11 hr) may be that they differ comparatively little in preceding homeostatic pressure. It has been shown that SWS increases linearly, or at least more steeply, with prior waking periods up to approximately 16 hr, after which the increase levels off (7,18). Also, the "two-process model" (26) would suggest a similar relation between prior waking and SWA. Sleep postponement in the present experiment involved 16–28 hr of prior waking, whereas the day sleeps were preceded by a much wider range of prior waking, from 4 (the 811 condition) to 28 hr. It thus



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**TABLE 4.** *Normalized (see text) SWA and SWS (min) during the first 60 min of the first and second cycles, respectively*

| Condition variable | First sleep cycle | Second sleep cycle | Source | *F* value | df | Epsilon | Level of significance |
|---|---|---|---|---|---|---|---|
| SWA 823[a] | 130.6 (13.3) | 70.2 (7.1) | Between conditions | 26.64 | 2/14 | 0.56 | <0.001 |
| SWA 211 | 133.2 (11.8) | 97.5 (19.9) | Between cycles | 41.19 | 1/7 | 1.00 | <0.0001 |
| SWA 011[b] | 225.8 (21.1) | 139.8[c] (21.0) | Interaction | 2.03 | 2/14 | 0.88 | ns |
| SWS 823 | 20.1 (5.8) | 8.8 (3.0) | Between conditions | 8.12 | 2/14 | 0.70 | <0.02 |
| SWS 211 | 20.8 (2.7) | 10.7 (4.2) | Between cycles | 25.10 | 1/7 | 1.00 | <0.02 |
| SWS 011[a] | 36.1 (3.6) | 20.8 (4.1) | Interaction | 0.38 | 2/14 | 1.00 | ns |

Means, standard errors (in parentheses) and results from two-factor ANOVA. Levels of significance after epsilon correction.
[a,b] Significant ($p < 0.05$; $p < 0.01$; Newman–Keuls test) pairwise comparison between first and second cycle.
[c] Significant ($p < 0.05$; Newman–Keuls test) pairwise comparison with second cycle of the 823 condition.

seems reasonable to expect more differentiated responses for the latter set of conditions.

It is true for the day sleeps, and to some extent for the night sleeps, that the homeostatic pressure accomplished increases both in the duration and the intensity of SWS. In spite of this increased pressure all available NREM time was not used for SWS. Instead, there was for all conditions a period of stage 1+2 sleep and, derived from the FFT data, a corresponding period between the largest fall in SWA and the start of REM sleep. It seems as if SWS (or the period with intense SWA) was interrupted after a certain time, because neither the cessation of SWS not the timing of the largest fall of SWA varied between conditions, and not directly by the occurrence of REM. Furthermore, SWS ceased at that time even if the need for SWS or SWE was not satisfied. Otherwise there would have been no "spill over" of SWE to the second cycle, as was the case for the 011 sleep.

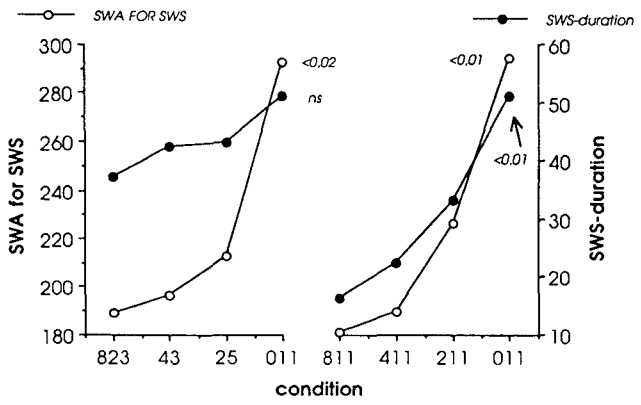


**FIG. 3.** Mean SWA during SWS and duration of SWS (min) during the first cycle over conditions. Left panel shows the 823, 43, 25 and 011 conditions and right panel the 811, 411, 211 and 011 conditions, respectively (p values from the ANOVAs in Table 3). Means of eight subjects.

The homeostatic responses to sleep loss seem to follow a typical sequence of events: at the lower levels of prior sleep loss the increases in SWS duration dominate, whereas the intensity changes dominate at the highest level. It is tempting to once again speculate that there is a maximum time allotted to SWS activity within the first sleep cycle. When this time is used up, the intensity of SWS has to increase in order to continue the homeostatic response. However, in the present experiment, even the considerable increase in SWS intensity after 28 hr of prior waking was not sufficient to accommodate the entire recovery within the first sleep cycle. And because the first cycle did not change systematically in duration, there was an increased homeostatic activity also in the second sleep cycle. In that respect our results differ from those of Feinberg et al. (8,10).

In conclusion, the temporal occurrence of REM sleep and the duration of the first cycle remained unaffected in spite of prior waking periods ranging from 4 hr to 28 hr. Both the duration and intensity of SWS activity were clearly and systematically affected by increased prior waking. The homeostatic response was to a large extent accomplished during the first sleep cycle. When prior waking exceeded that of normal sleep/wake conditions with 12 hr there was, however, a significant "spill over" to the second cycle. The present data suggest that this "spill over" occurs because the first sleep cycle, for some reason, is limited in the amount of SWS activity (both duration and intensity) that it can accommodate. Furthermore, the limit in duration is reached before the start of REM sleep and, hence, does not seem to be directly determined by the latter.

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