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Selective Slow-Wave Sleep (SWS) Deprivation and SWS Rebound: Do We Need a Fixed SWS Amount per Night?

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Visually scored delta activity (stages 3 and 4, SWS) as well as computerized delta activity measures increase after total and selective sleep deprivation. It is, however, still controversial if SWS amount is only a function of prior waking duration, or if it is related to the structure of the previous sleep period (i.e., to the time spent in SWS). In order to clarify if the amount of SWS is crucial in determining SWS recovery, we selectively deprived SWS during two nights to assess the presence of a compensatory SWS rebound in the following recovery night. Ten normal males slept for 6 consecutive nights in the laboratory. After an adaptation and two baseline nights (BSL; BSL-A), selective SWS deprivation was accomplished for two consecutive nights (DEP-1; DEP-2), by means of an acoustic stimulation technique. A recovery (REC) night then followed. An almost complete selective SWS suppression during both deprivation nights was achieved. A significant increase of S4 and SWS in the REC as compared to the BSL-A paralleled a significant shortening of S3 and S4 latencies. S2 percentage significantly increased during both DEP nights with respect to the other experimental nights. There was no significant difference among nights with regard to total sleep time, percentage of REM sleep, stage 1, movement time, number of awakenings and number of movement arousals, indicating that the acoustic stimulation technique did not dramatically disrupt normal sleep continuity and architecture. These results indicate that SWS rebound after selective SWS deprivation can be ascribed to the loss of SWS accumulated during two consecutive nights, further supporting the idea that the delta sleep amount is more linked to SWS in the previous sleep periods than to the total sleep duration.

CURRENT CLAIM: SWS rebound after selective SWS deprivation can be ascribed to a loss of SWS, not to sleep curtailment/fragmentation.

Selective SWS deprivation studies have indicated that this experimental manipulation of sleep structure can enhance the SWS amount in the subsequent sleep period (Dijk et al., 1987; Dijk and Beersma, 1989; Gillberg et al., 1991; Gillberg and Akerstedt, 1994). In all these studies, an acoustic stimulation technique applied for a few hours during either diurnal or nocturnal sleep, allowed the authors to reduce but never to completely suppress the SWS amount, without increasing intra-sleep wakefulness. Nevertheless, even these "mild" manipulations of the SWS quantity led to a compensatory SWS rebound during the subsequent undisturbed sleep periods, suggesting that a more or less fixed amount of SWS is needed.

Up to now, longer SWS deprivation periods have been obtained only by using the *awakening method*. Bonnet (1986) performed two consecutive nights of SWS deprivation by fully awakening the participants every time they entered stage 3. This procedure allowed a complete suppression of stage 4 and a reduction of stage 3 percentage to 2-3%. However, the awakening method also provoked a marked increase of wakefulness and a decrease of REM sleep and of total sleep time (TST) during the experimental nights. More recently, Walsh et al. (1994) selectively deprived SWS for two consecutive nights by the same awakening method. In this case, SWS was virtually eliminated, but TST decreased and wake time increased in the deprivation nights. In both studies (Bonnet, 1986; Walsh et al., 1994) a clear SWS rebound in the recovery night was found; however, it is difficult to say how much of this rebound has to be ascribed to the selective SWS deprivation, and how much is due to the intrusion of wakefulness and to the shortening of TST in the deprivation nights.

In order to clarify whether the amount of sleep *per se* or the time spent in SWS is more important in determining SWS rebound, in the present study we selectively deprived SWS during two consecutive nights by means of an acoustic stimulation technique to assess the presence of a compensatory SWS rebound in the following recovery night. Great attention was paid to prevent participants from fully entering stage 3 by lightening their sleep, but also avoiding awakening them, and to keep sleep duration constant in all the experimental nights, in order to avoid its confounding influence on the recovery sleep structure.

METHODS

Participants

Ten normal right-handed male students (ages 20-30 years) were selected as paid volunteers for the study. All of them signed an informed consent before participating in the study. The requirements for inclusion were: normal sleep duration and schedule, no daytime nap habits, no excessive daytime sleepiness, no other sleep, medical or psychiatric disorder, as assessed by a one-week sleep log and by a clinical interview. Participants were required to avoid napping throughout the

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experiment; compliance was controlled by actigraphic recordings (AMI motion logger 16 K).

Procedures

The protocol of the study was reviewed and approved by the local Institutional Review Board. Participants slept for 6 consecutive nights in a sound-proof, temperature controlled room: 1) Adaptation; 2) Baseline (BSL); 3) Baseline with awakenings (BSL-A); 4) SWS Deprivation-1 (DEP-1); 5) SWS Deprivation-2 (DEP-2) and 6) Recovery (REC).

Every night, sleep recording started at about 11:30 p.m. and ended after 7.5 h of accumulated sleep. During nights #3-6 participants were awakened twice, and a psychophysiological test battery was administered (results will be presented elsewhere). The test battery lasted about 13 min. Performance was assessed with subjects laying down in bed in the dark. At the end of testing subjects were asked to go back to sleep.

During nights #4 and #5, two experimenters continuously monitored the EEG chart and delivered a tone (frequency: 1000 Hz; intensity: 40-110 dB), by pressing a button whenever at least 2 delta waves (0.5-3.5 Hz; >75 μV), determined by visual inspection, appeared in a 15-sec recording interval. Acoustic stimuli were administered through a loudspeaker placed about 40 cm above the subjects' head. Beginning from the lowest intensity, it was increased in steps of 5 dB if no response occurred (sleep stage shift, K-complex, EEG desynchronization, alpha burst, muscle tone increase, slow eye movements). In this manner, we prevented the subject from fully entering stage 3 by lightening his sleep, but avoiding awakening him.

Polygraphic Recordings

An Esaote Biomedica VEGA 24 polygraph set at a paper speed of 10 mm/s was used for polygraphic recordings. EEG (C3-A2 and C4-A1) was recorded with an AC time constant of 0.3 s. Submental EMG was recorded with a time constant of 0.03 s. Bipolar horizontal and vertical eye movements were recorded with a time constant of 1 s. Bipolar horizontal EOG was recorded from electrodes placed about 1 cm from the medial and lateral canthi of the dominant eye, and bipolar vertical EOG from electrodes located about 3 cm above and below the right eye pupil. Electrode impedance was kept below 5 KΩ. Left central EEG (C3-A2), EMG, and horizontal and vertical EOG were used to visually score sleep stages, according to the standard criteria (Rechtschaffen and Kales, 1968). With regard to delta sleep scoring, the amplitude criterion (>75 μV) expressed by Rechtschaffen and Kales was strictly followed.

Data Analysis

A one-way ANOVA with Night as a 5-level factor (BSL, BSL-A, DEP-1, DEP-2, REC) was carried out on the percentage and latency of each sleep stage (except for stages 3 and 4 -S3, S4-), Intra-Sleep Wakefulness (ISW), Movement Time (MT), Number of Awakenings (NA), number of Movement Arousals (MA), Total Sleep Time (TST), Total Bed Time (TBT) and Sleep Efficiency index (SE).

With regard to S3, S4 and SWS percentages, the ANOVA only included BSL-A and REC as levels of the Night factor. Neither deprivation night was considered in this analysis because S3, S4 and SWS (S3+S4) were virtually absent during DEP-1 and DEP-2 (see Results section). Furthermore, since S3, S4 and SWS percentage during BSL and BSL-A nights were statistically equivalent, we only considered the latter because both during the BSL-A and the REC two nocturnal experimental awakenings were scheduled (see Procedure section), at variance with BSL during which sleep was uninterrupted.

To further evaluate the effects of selective SWS deprivation on SWS redistribution during the recovery night, a two-way ANOVA Night (BSL-A, REC) by Cycle (1st, 2nd, 3rd, 4th) was carried out on S3, S4 and SWS percentages.

Finally, to evaluate the effects of experimental manipulation on REM sleep and on intra-sleep wake distribution, a two-way ANOVA Night (BSL, BSL-A, DEP-1, DEP-2, REC) by Cycle (1st, 2nd, 3rd, 4th) was carried out on REM percentage and on ISW duration.

ANOVAs were performed using the Greenhouse-Geisser correction. The Fisher PLSD test was used for *post hoc* comparisons of the means.

RESULTS

Table 1 shows means and standard deviations of each dependent variable during 5 out of the 6 experimental nights (adaptation was omitted).

The acoustic stimulation technique allowed us to achieve an almost complete selective SWS suppression during both the deprivation nights; in fact, the mean percentage of SWS was 0.29% and 0.65% in DEP-1 and DEP-2, respectively. During the DEP-1, 328.1 (±167.69) acoustic stimuli were delivered, with a mean intensity of 56.03 dB (±14.12). During the DEP-2, as a consequence of an increase of the arousal thresholds probably due to the greater SWS pressure, 739.8 (±314.8) acoustic stimuli were delivered, with a mean intensity of 72.1 dB (±11.45). The number of acoustic stimuli delivered was significantly increased in DEP-2 as compared to DEP-1 (Wilcoxon signed-rank test, Z=-2.8; p=.005) as well as their mean intensity (one-way ANOVA, $F_{1,9}$= 33.87; p=.0003).

In regard to sleep stage percentages, ANOVAs showed a significant increase of S4 ($F_{1,9}$=16.59; p=.003) and SWS ($F_{1,9}$=21.34; p=.001) in the REC as compared to the BSL-A, paralleled by a significant shortening of S3 ($F_{1,9}$=7.70; p=.02) and S4 latencies ($F_{1,8}$=11.37, p=.01). As a consequence of SWS deprivation, S2 percentage was significantly modified ($F_{4,36}$=17.91; p=.0001). In particular, post-hoc comparisons showed that S2 percentage was increased in the DEP-1 and DEP-2 with respect to the other experimental nights (DEP1 vs. BSL, p=.0006; DEP1 vs. BSL-A, p=.000005; DEP1 vs. REC, p=.0000002; DEP2 vs. BSL, p=.0006; DEP2 vs. BSL-A, p=.000004; DEP2 vs. REC, p=.0000002). On the other hand, as a consequence of SWS rebound, S2 was significantly decreased in the REC with respect to the BSL (p=.01).

ANOVAs also showed a significant main effect for SE ($F_{4,36}$=5.42; p=.002) and ISW ($F_{4,36}$= 2.68; p=.05). *Post-hoc* comparisons of the means showed that SE was significantly

Table 1
Visually Scored EEG Parameters During Each Experimental Night
Means and Standard Deviations (within brackets)

	NIGHT							
	1	2	3	4	5			Mean
Variable	BSL	BSL-A	DEP-1	DEP-2	REC	F	p	Differences
% Stage 1	7.06 (2.96)	9.59 (3.72)	8.52 (3.35)	6.81 (2.01)	7.26 (4.70)	1.72*	.17	--
% Stage 2	57.51 (5.07)	53.70 (7.01)	66.43 (4.49)	66.46 (7.38)	51.32 (7.51)	17.91*	.0001	3=4>1, 2, 5; 5<1
% Stage 3	6.43 (2.74)	5.74 (2.51)	0.29 (0.32)	0.60 (0.71)	5.96 (2.63)	.04**	.84	--
% Stage 4	6.32 (4.51)	5.69 (5.13)	0 (0)	0.05 (0.14)	11.01 (8.19)	16.59**	.003	5>2
% SWS	12.75 (5.86)	11.42 (6.12)	0.29 (0.32)	0.65 (0.79)	16.97 (8.90)	21.34**	.001	5>2
% REM	22.68 (3.36)	25.29 (4.29)	24.76 (5.70)	26.08 (7.06)	24.45 (3.07)	1.22*	.32	--
ISW	29.05 (30.45)	12.20 (8.45)	42.90 (60.23)	16.45 (16.34)	6.00 (5.81)	2.68*	.05	3>2, 4, 5
MT	6.70 (2.88)	4.95 (2.39)	6.85 (3.86)	6.15 (3.26)	5.40 (3.53)	1.93*	.13	--
MA	65.40 (34.19)	60.60 (27.69)	65.20 (26.38)	67.50 (24.03)	60.30 (34.09)	.49*	.74	--
NA	10.50 (5.42)	11.90 (8.09)	20.60 (23.65)	12.50 (9.16)	8.40 (4.79)	1.97*	.12	--
TBT	499 (33.65)	494 (22.57)	520 (34.66)	512 (29.28)	492 (25.33)	1.86*	.14	--
TST	457 (38.24)	462 (42.12)	451 (42.12)	478 (34.99)	468 (24.71)	1.75*	.16	--
SE (%)	91.66 (6.59)	89.64 (6.91)	85.04 (11.91)	91.24 (5.68)	93.72 (2.98)	5.42*	.002	5>2; 3<1, 2, 4, 5
S1 Latency	16.95 (18.84)	14.35 (13.13)	17.95 (17.46)	11.05 (9.13)	12.25 (10.47)	1.50*	.22	--
S2 Latency	19.95 (19.44)	18.15 (14.40)	21.30 (17.40)	14.45 (10.10)	16.80 (10.49)	1.14*	.35	--
S3 Latency	38.95 (21.69)	35.29 (15.67)	--	--	28.00 (11.85)	7.70**	.02	5<2
S4 Latency	44.31 (25.67)	41.67 (15.70)	--	--	33.83 (13.20)	11.37**	.01	5<2
REM Latency	92.5 (27.45)	101.9 (40.24)	141.5 (108.3)	119.7 (63.46)	98.1 (33.46)	1.44*	.24	--

SWS= Slow-Wave Sleep (stages 3+4); ISW= Intra-Sleep Wake; MT= Movement Time; MA= number of Movement Arousals; NA= Number of Awakenings; SE= Sleep Efficiency index (percentage of efficiency); TBT= Total Bed Time; TST=Total Sleep Time ISW, MT, TBT, TST and sleep stage latencies are expressed in minutes.
*Degrees of freedom= 4, 36 **Degrees of freedom= 1, 9

higher in the REC as compared to the BSL-A (p=.05); in addition, SE in DEP-1 was significantly lower than in all the other nights (DEP1 vs. BSL, p=.002; DEP1 vs. BSL-A, p=.03; DEP1 vs. DEP2, p=.003; DEP1 vs. REC, p=.0001). *Post-hoc* comparisons also indicated that ISW increased in DEP-1 as compared to all the other nights but BSL (DEP1 vs. BSL-A, p=.02; DEP1 vs. DEP2, p=.05; DEP1 vs. REC, p=.006). Since it was suspected that the increase of intra-sleep wake during DEP-1 was due to only one *outlier* subject (who proved to be very sensitive to acoustic stimulation, being awakened several times even by the faintest tones), a new ANOVA was performed disregarding his data. In this case, the main effect was no more significant ($F_{4,32}$=2.33; p=.08), however, showing a tendency toward significance. *Post-hoc* comparisons of the means indicated that ISW was significantly decreased during the REC as compared with both BSL (p=.02) and DEP-1 (p=.03) nights.

With regard to the other dependent variables, significant differences between nights were not found (see Table 1).

With respect to SWS redistribution during the REC night, ANOVA showed a significant Night by Cycle interaction only for S4 percentage ($F_{3,27}$=3.10; p=.05); *post-hoc* comparisons indicated that, in the first and third sleep cycle of REC, S4 percentage (mean percentage 1st cycle=29.2; 2nd=10.6; 3rd= 7.8; 4th=1.4), was significantly increased (p=.0002 and p=.03, respectively), as compared to the same cycles of the BSL-A night (mean percentage 1st cycle=16.1; 2nd=7.9; 3rd=.83; 4th=.26).

Finally, regarding ISW distribution among the sleep cycles of the 5 experimental nights, no main effect or interaction was significant. On the other hand, with regard to REM sleep, the main effect for the Cycle factor was significant ($F_{3,27}$=4.03; p=.02). *Post-hoc* comparisons showed that REM percentage during the first cycle (mean percentage=16.8), significantly differed from all the other cycles (mean percentage 2nd cycle= 24.3; 3rd=26.5; 4th=24.6; 1st vs. 2nd, p=.02; 1st vs. 3rd, p=.003; 1st vs. 4th, p=.02). The Night by Cycle interaction only approached significance ($F_{12,108}$=1.72; p=.07).

DISCUSSION

Selective SWS deprivation studies are very difficult to perform and often affected by methodological problems. From a methodological point of view, in a selective SWS deprivation study it is important to evaluate: a) the actual effectiveness of the deprivation procedure (i.e., the SWS amount in the experimental nights) and b) the intra-sleep wake amount and possible differences in total sleep time (TST) among the experimental nights. All these issues are strictly related. As a matter of fact, up to now the most successful SWS deprivation studies (Bonnet, 1986; Walsh et al., 1994), that adopted the awakening method to selectively deprive SWS, have also

reported a significant increase of intra-sleep wake and of light sleep (stage 1), and a concomitant reduction of REM sleep amount and of total sleep duration. In these cases, it is not unambiguous to ascribe the SWS rebound in the recovery night only to SWS manipulation. In fact, a shortening of TST and a fragmentation of the sleep period can be followed by an SWS increase during the recovery night (e.g., Bonnet, 1987).

In the present study an acoustic stimulation technique was used to deprive SWS because this method allowed us to avoid dramatic changes of sleep continuity. Actually, selective SWS deprivation for 2 consecutive nights was very effective, since the SWS percentage during both the deprivation nights was close to zero. In addition, a highly significant S4 and SWS rebound during the recovery night was found, accompanied by a shortening of S3 and S4 latency.

This SWS rebound cannot be considered a side-effect of an overall sleep curtailment. As a matter of fact, selective SWS deprivation did not affect TST. Furthermore, the exact amount of SWS denied during the deprivation nights was merely replaced by an increase of stage 2 (see Table 1). With regard to SWS redistribution during the recovery night, an increase of stage 4 percentage during each of the first four sleep cycles was present, even if the difference between REC and BSL-A night was significant only for the first and third cycle. This result is only in part surprising. In fact, several studies have found that the delta rebound after sleep deprivation of different durations is confined either to the first (e.g., Feinberg, Floyd et al., 1987; Feinberg et al., 1988), or to the first two NREM periods of recovery sleep (e.g., Lucidi et al., 1997). However, some of the earliest recovery models of delta sleep (Feinberg, 1974; Borbely, 1982) predicted increased delta across several NREM periods after total sleep loss. On the whole, our results on SWS rebound and redistribution seem to indicate that the effects of two consecutive nights of SWS deprivation on sleep structure of the recovery night can be very similar to those of total sleep deprivation, pointing out the importance of obtaining a more or less fixed amount of SWS per night. As a matter of fact, SWS in the recovery night increased in the present study by about one-third with respect to the baseline percentage (see Table 1); a similar size of SWS rebound has already been reported after sleep deprivations of different durations (e.g., Borbely et al., 1981; Rosa and Bonnet, 1985; Dijk et al., 1991).

It has been argued that SWS rebound after selective SWS deprivation could be due to sleep fragmentation and wake intrusion during the experimental nights more than to SWS suppression *per se* (e.g., Lucidi et al., 1997). This could be true for some early works on selective SWS deprivation (Agnew et al., 1967; Agnew and Webb, 1968; Moses et al., 1975; Moldofsky and Scarisbrick, 1976). However, more recently it has been clearly shown that even mild reductions of SWS quantity, obtained by means of acoustic stimulation without inducing a significant increase of wakefulness, are followed by a compensatory SWS rebound during the subsequent undisturbed sleep (Dijk et al., 1987; Dijk and Beersma, 1989; Gillberg and Akerstedt, 1994).

Although administering acoustic stimulation can disrupt the continuity of sleep with brief arousals, evidence of reduced sleep quality has not been found in all studies of experimentally induced fragmentation. As an example, in two different studies Badia and co-workers (Badia et al., 1985; Magee et al., 1987), found no evidence of increased daytime sleepiness as a consequence of requiring subjects to take a deep breath after stimuli presented as frequently as once every 4 minutes. Although full awakenings seldom occurred, responses were typically accompanied by indices of brief arousal (e.g., stage shifts, EMG increases, alpha bursts). In both studies experimental subjects appeared to have a 40-50% reduction of SWS; no other significant difference in sleep stage distribution as compared to control subjects was present. In these studies, however, sleep in the recovery night was not recorded.

Probably, fragmentation reduces daytime functioning only when sleep disturbance occurs at a greater rate. Bonnet (1987) compared three different types of periodic sleep disturbance. In this study, sleep was fragmented either by complete awakenings or body movements or EEG changes. The third condition, requiring only a change in ongoing EEG in response to the acoustic stimulation presented 2 min after the appearance of a well-defined spindle, K-complex or REM, is the most comparable with the selective SWS deprivation condition of the present study. The sleep disruption procedure caused a complete suppression of stage 4 and a marked reduction of stage 3, followed by a slight but significant S4 rebound during the recovery night. However, in the disruption nights a marked increase of wakefulness, of stage 1 percentage and of the number of awakenings, and a clear reduction of REM percentage was also found. In an already mentioned study, Walsh and co-workers (1994) compared the effects of a selective SWS deprivation by means of the awakening method with those of a control disruption procedure, consisting of awakening subjects out of stage 2 with at least two minutes of sleep between awakenings. In both experimental conditions a decrease in total sleep time and a significant increase in the number of awakenings, stage 1 and wake duration was found as compared to a control undisturbed condition. SWS was virtually eliminated during the SWS deprivation nights, leading to an SWS rebound during the recovery night. On the other hand, control disruption did not affect the SWS amount, however, causing a significant increase of SWS in the following night.

In summary, whenever an SWS rebound has been reported in a sleep fragmentation study, the sleep disruption procedure has always provoked, besides a SWS decrease, also a marked increase of wakefulness, of stage 1 and of the number of awakenings during the fragmentation nights. Although the procedure and the outcomes of some of the above-mentioned experimental fragmentation studies seem to be similar to those of the present study, with closer inspection it is clear this is not the case.

In the present study, great attention was paid to avoid full awakenings of the subjects by simply preventing them from entering stage 3. In other words, we delivered a tone whenever the EEG showed the first signs of synchronization, provoking a sort of continuous stage 2. In fact, two-thirds of both deprivation nights consisted of stage 2 (see Table 1). Only in a few cases was it necessary to arouse subjects who were

becoming insensitive to acoustic stimulation. However, all the indices of sleep continuity (stage 1 percentage, movement time, number of movement arousals and of full awakenings), did not significantly differ among the experimental nights, indicating that the selective SWS deprivation nights cannot be considered in the same way as fragmentation/disruption nights.

The increase of wakefulness during the first deprivation night was found only for one *outlier* subject. Disregarding his data, there is no significant difference among nights also with regard to intra-sleep wake duration. Finally, during the selective SWS deprivation nights there was no partial sleep deprivation, since total sleep time did not differ among nights. Therefore, it is reasonable to ascribe the effect of SWS rebound after selective SWS deprivation merely to the loss of SWS accumulated during two consecutive nights.

In conclusion, these results extend previous reports of an increased SWS amount after mild reductions of the SWS quantity by means of acoustic stimulation not affecting intra-sleep wake (Dijk et al., 1987; Dijk and Beersma, 1989; Gillberg and Akerstedt, 1994), further supporting the idea that the delta sleep amount is more linked to SWS in the previous sleep periods than to the total sleep duration.

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