EEG Arousal 

EEG Arousals in Normal Sleep: Variations Induced by Total and Selective 

Slow-wave Sleep Deprivation

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Study Objectives: Aim of the present study was to assess changes in arousal rates after selective slow-wave (SWS) and total sleep deprivations.

Design: Two-way mixed design comparing the arousal index (AI), as expressed by the number of EEG arousals divided by sleep duration, in totally or selectively sleep deprived subjects.

Setting: Sleep laboratory.

Patients or Participants: Nineteen normal male subjects [mean age=23.3 years (S.E.M.=0.55)].

Interventions: AI was measured in baseline nights and after selective SWS (N=10) and total sleep deprivation (N=9).

Measurements and Results: During the baseline nights AI values changed across sleep stages as follows: stage 1 > stage 2 and REM > SWS, but did not present any significant variations as a function of time elapsed from sleep onset. The recovery after deprivation showed a reduction in EEG arousals, more pronounced after total sleep deprivation; this decrease affected NREM but not REM sleep. During the baseline nights AI showed a close-to-significance negative correlation with REM duration, while during the recovery nights a significant positive relation with stage 1 duration was found.

Conclusions: The present results suggest that recuperative processes after sleep deprivation are also associated with a higher sleep continuity as defined by the reduction of EEG arousals.

Key words: EEG arousal; sleep deprivation; SWS deprivation; ASDA rules

INTRODUCTION

SPONTANEOUS AROUSAL IS A PHYSIOLOGICAL COMPONENT OF NORMAL SLEEP AND SO IT WAS INCLUDED IN RECHTSCHAFFEN & KALES CRITERIA.1 It was defined as “any increase in EMG or any channel which is accompanied by a change in pattern on any additional channel”; its scoring is possible only if it is immediately preceded and followed by epochs of sleep. However, Rechtschaffen & Kales guidelines state that arousal is not considered as epoch scoring. In the past few years the relation between arousals and inadequate sleep recuperative benefits has been studied within experimentally disturbed sleep, with the hypothesis that a higher frequency of arousals (and, therefore, fragmentation of sleep) reduces the restorative benefit of sleep and results in a greater impairment of daytime performance.2,3 Interest in this issue is mainly due to the common association between arousals and sleep-related breathing disorders.4

In 1992 a task force of the American Sleep Disorders Association (ASDA) developed scoring rules for these phasic EEG changes resulting in fragmented sleep,5 independently of the Rechtschaffen & Kales scoring system.1 Due to clinical interest in determining their frequency in normal sleep, EEG arousals, scored following ASDA rules, have been quantified across the ages in normal subjects.6,7 Both studies converged in showing a linear increase of EEG arousals with age.

Little is known of the sensitivity of this index to the increased need of recuperation that follows total or selective sleep deprivations in normal subjects. In our opinion, recovery sleep after sleep deprivation should be characterized by a lower fragmentation indexed by a decrease in EEG arousals. The aim of the present study is therefore to assess changes in arousal rates after selective SWS and total sleep deprivations. These two paradigms are characterized by a recuperative increase of SWS in the nights that follow sleep deprivation as compared to baseline nights.8,9 Since the need for recuperation, as expressed by SWS rebound, is higher after total sleep deprivation, a more pronounced decrease of EEG arousal is also expected.

MATERIAL AND METHODS

Subjects

Nineteen normal male subjects were selected as paid volunteers for the study [mean age=23.3 years (S.E.M.=0.55); age range=20-30 years]. Subjects were selected if they usually went to sleep between 23:00 and 24:00 and if they usually slept seven to eight hours per night. Other requirements for inclusion were: 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 unaware of the purpose of the experiment and signed an informed consent; their rights were protected through the entire course of the experiment.

Procedure

The data for this study come from two separate, previously published studies.8,9 We decided to retrospectively combine these two samples since they are characterized by a different recuperative increase of SWS in the nights that follow sleep deprivation (i.e., SWS rebound is higher after total sleep deprivation than...
Subjects were recorded in a sound-proof, temperature-controlled room (22° C). Ten of them participated in a SWS deprivation study, sleeping six consecutive nights: 1) Adaptation; 2) Baseline 1 (BSL-1); 3) Baseline 2 (BSL-2); 4) SWS Deprivation-1 (DEP-1); 5) SWS Deprivation-2 (DEP-2); 6) Recovery (REC). SWS deprivation was performed during nights #4 and #5 by delivering a tone (frequency: 1000 Hz; intensity: 40-110 dB-spl) whenever at least two delta waves (0.5-3.5 Hz; >75 µV) appeared in a 15-second recording interval. This technique allowed us to set SWS amount near to zero in both the deprivation nights.

Nine subjects participated in a total deprivation study: 1) Adaptation (AD); 2) Baseline (BSL); 3) Recovery (REC). A 40-hour schedule of sleep deprivation began after morning awakening from the BSL night.

**Sleep Recording**

A VEGA 24 (Esaote Biomedica, Firenze) 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.

Bipolar horizontal and vertical eye movements were recorded with a time constant of 1 second. 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.

Submental EMG was recorded with a time constant of 0.03 seconds as a standard measure of electromyographic activity during sleep. Electrode impedance was kept below 5 KOhms.

Left central EEG (C3-A2), EMG, and horizontal and vertical EOG were used to visually score sleep stages, according to the standard criteria. With regard to SWS scoring, the amplitude criterion (>75 µV) was strictly followed.

**EEG Arousal Index**

EEG arousals were scored following ASDA rules; the only modification made to these rules was a shorter interval (1.5 seconds instead of 3 seconds) of EEG changes. This criterion allows increasing the time resolution of the analysis. Therefore, we scored as an arousal any shift in the EEG frequency to alpha or theta for at least 1.5 seconds irrespective of changes in submental EMG during NREM sleep but accompanied by a 1.5 seconds increase in EMG amplitude during REM sleep.

Since the aim of the study was to assess changes in arousal rates after selective SWS and total sleep deprivations, we scored the arousal index (AI) during the BSL and REC nights of the total sleep deprivation, and BSL-2(1) and REC nights of the SWS deprivation. AI was expressed by the ratio between number of arousals divided by sleep duration.

**RESULTS**

**Polysomnography**

Table 1 reports the main polysomnographic variables during the baseline and recovery nights of selective and total sleep deprivations. Recovery after slow-wave sleep deprivation was characterized by increases of only SWS duration (also a decrease

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Figure 1—Means (S.E.M.) of arousal index (# of arousals/hour) as a function of sleep stage of the baseline nights (N=19).

(1) BSL-2 (selective SWS deprivation) and BSL (total sleep deprivation) did not show any significant difference with respect to the amount of stage 1, 2 SWS and REM Sleep.
in SWS latency) and sleep efficiency, while total sleep deprivation caused broader changes in the recovery nights: a decrease of stage 1, stage 2 and sleep latency; an increase of SWS (practically doubled, from about 11% to over 22%), and of the sleep efficiency index. Finally, Intra-Sleep Wake [ISW(2)] significantly decreased after both total and selective sleep deprivations.

The sleep stage data from the selective slow-wave sleep deprivation nights have been reported previously 8 and so a summary will be provided here. Total sleep time, stage 1 and REM sleep amount, stage 1 and REM sleep latency, number of movement time and of arousal (defined according to R & K rules), did not show any significant difference as compared to the other nights. The only significant difference was an increased ISW and a decreased Sleep Efficiency (SE%), but these effects were only due to a single outlayer subject. Therefore, 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 (and these did not differ from the baseline ones), indicating that the selective SWS deprival nights can not be considered in the same way as fragmentation/disruption nights. More details (e.g., number of experimenter induced arousals or percentages of sleep stages) on these SWS deprivation nights have been reported elsewhere. 8

Finally, the polysomnographic variables of the baseline nights were also compared, since they were respectively the third one within the SWS deprivation condition and the second one within the total deprivation condition, and they could be differently contaminated by poor sleep quality during the adaptation night. In fact, the comparisons between stage 1, stage 2, SWS and REM amounts of the two baseline nights showed no significant difference [stage 1: F(1,17)=1.02, p=0.33; stage 2: F(1,17)<1; SWS: F(1,17)<1; REM: F(1,17)<1].

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Table 1—Means and S.E.M. (expressed in minutes) of sleep parameters during the baseline (BSL) and the recovery night (REC) of the selective SWS (N=10) and total sleep deprivation (N=9) paradigms. The table also reports the one-way ANOVA results of the within-subjects comparisons between the nights. It should be noticed that BSL of SWS deprivation paradigm is the second baseline night, that is the night preceding the two consecutive SWS deprivation nights. SWS=Slow-Wave Sleep (stages 3+4); ISW=Intra-Sleep Wake; SE=Sleep Efficiency index (percentage of efficiency); TST=Total Sleep Time.

<table>
<thead>
<tr>
<th>Sleep Parameter</th>
<th>BSL Mean (S.E.M.)</th>
<th>REC Mean (S.E.M.)</th>
<th>F(1,9)</th>
<th>p</th>
<th>BSL Mean (S.E.M.)</th>
<th>REC Mean (S.E.M.)</th>
<th>F(1,8)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1 latency (min)</td>
<td>14.35 (4.15)</td>
<td>12.25 (3.31)</td>
<td>0.73</td>
<td>0.42</td>
<td>15.67 (5.23)</td>
<td>2.50 (0.61)</td>
<td>8.41</td>
<td>0.02</td>
</tr>
<tr>
<td>Stage 2 latency (min)</td>
<td>18.15 (4.55)</td>
<td>16.80 (3.32)</td>
<td>0.23</td>
<td>0.65</td>
<td>19.11 (5.57)</td>
<td>4.06 (0.81)</td>
<td>9.44</td>
<td>0.01</td>
</tr>
<tr>
<td>SWS latency (min)</td>
<td>35.25 (4.96)</td>
<td>28.00 (3.75)</td>
<td>7.70</td>
<td>0.02</td>
<td>45.94 (14.51)</td>
<td>12.89 (2.09)</td>
<td>6.13</td>
<td>0.04</td>
</tr>
<tr>
<td>REM latency (min)</td>
<td>101.90 (12.72)</td>
<td>98.10 (10.58)</td>
<td>0.41</td>
<td>0.54</td>
<td>85.83 (9.47)</td>
<td>94.94 (17.54)</td>
<td>0.35</td>
<td>0.57</td>
</tr>
<tr>
<td>Stage 1 (min)</td>
<td>44.85 (6.05)</td>
<td>34.95 (7.88)</td>
<td>2.51</td>
<td>0.15</td>
<td>36.72 (5.13)</td>
<td>19.83 (2.79)</td>
<td>15.42</td>
<td>0.004</td>
</tr>
<tr>
<td>Stage 2 (min)</td>
<td>248.00 (10.85)</td>
<td>240.10 (11.47)</td>
<td>0.95</td>
<td>0.35</td>
<td>256.00 (8.59)</td>
<td>228.83 (13.29)</td>
<td>7.53</td>
<td>0.02</td>
</tr>
<tr>
<td>SWS (min)</td>
<td>52.20 (8.50)</td>
<td>78.65 (12.95)</td>
<td>21.74</td>
<td>0.001</td>
<td>49.22 (8.58)</td>
<td>99.00 (14.13)</td>
<td>23.70</td>
<td>0.001</td>
</tr>
<tr>
<td>REM (min)</td>
<td>116.75 (6.19)</td>
<td>114.40 (4.77)</td>
<td>0.17</td>
<td>0.69</td>
<td>107.67 (8.26)</td>
<td>99.90 (6.21)</td>
<td>0.82</td>
<td>0.39</td>
</tr>
<tr>
<td>ISW (min)</td>
<td>12.20 (2.67)</td>
<td>6.00 (1.84)</td>
<td>6.48</td>
<td>0.03</td>
<td>17.33 (2.52)</td>
<td>9.33 (1.42)</td>
<td>5.88</td>
<td>0.04</td>
</tr>
<tr>
<td>TST (min)</td>
<td>461.80 (7.05)</td>
<td>468.10 (7.81)</td>
<td>0.59</td>
<td>0.46</td>
<td>449.60 (7.10)</td>
<td>447.60 (5.90)</td>
<td>0.21</td>
<td>0.65</td>
</tr>
<tr>
<td>SE (%)</td>
<td>89.64 (2.19)</td>
<td>93.72 (0.94)</td>
<td>7.02</td>
<td>0.03</td>
<td>92.54 (1.54)</td>
<td>97.24 (0.29)</td>
<td>9.33</td>
<td>0.01</td>
</tr>
</tbody>
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(2) Intra-Sleep Wake was defined as the total duration of epochs scored as wake according to R & K rules.
Arousal Index (AI)

Figure 1 reports mean AI during each sleep stage of the baseline nights. A two-way mixed design ANOVA [Deprivation (SWS vs. total) x Stage (1 vs. 2 vs. SWS vs. REM)] yielded significant differences only for Stage (F(1,17)=19.49; p=0.0004), indicating higher arousal rates during stage 1 as compared to stage 2 (p=0.0002, with Duncan’s Multiple Range Test), SWS (p=0.0004) and REM sleep (p=0.0001), while SWS was significantly lower than stage 2 (p=0.02) and REM sleep (p=0.05). These differences do not vary between the two different subjects’ groups.

With regard to time elapsed from sleep onset, Figure 2 reports mean AI during the first seven hours of the baseline nights. The two-way mixed design ANOVA [Deprivation (SWS vs. total) x Hour] showed no significant main effect or interaction for these
The effect of total and selective SWS deprivation was assessed by a two-way mixed design ANOVA [Deprivation (SWS vs. total) x Night (baseline vs. recovery)] on the mean AI. Results showed significant effects for Night (F(1,17)=19.49; p=0.0004) with a decrease from the baseline [M=12.30 (0.84)] to the recovery night [M=9.97 (0.91)], and for the Deprivation x Night interaction (F(1,17)=4.18; p=0.05). This interaction, depicted in Figure 3, showed that the decrease during the recovery night was greater after total (p=0.006, with Duncan’s Multiple Range Test) than after selective sleep deprivation (p=0.10).

This reduction of AI during recovery nights was mainly attributable to NREM as compared to REM sleep. In fact, a three-way mixed design ANOVA [Deprivation (SWS vs. total) x Night (baseline vs. recovery) x Stage (NREM vs. REM sleep)] showed a Night x Stage interaction (F(1,17)=4.89; p=0.04) pointing to the decrease of arousals in the recovery night that affects only NREM sleep (Figure 4): while the comparison between baseline and recovery nights was significant for NREM sleep (p=0.04, with Duncan’s Multiple Range Test), it was not for REM sleep (p=0.74). The analysis also confirmed the significant effects for Night (F(1,17)=10.52; p=0.005) and the Deprivation x Night interaction (F(1,17)=5.28; p=0.03), indicating respectively lower arousal rates during the recovery nights (BSL=12.13, REC=10.34), and a greater decrease of AI after total sleep deprivation (Total deprivation: BSL=12.29, REC=9.23; SWS deprivation: BSL=11.96, REC=11.44).

Finally, to clarify whether the decrease of AI during NREM stages of the recovery nights is simply associated with the increased amount of SWS which is characterized by lower arousal rates, product-moment correlations between AI and sleep stage amounts were calculated separately for the baseline and recovery nights. As detailed in Table 2, during the baseline there was a negative correlation approaching significance only for REM sleep, while during the recovery night the positive correlation was significant for stage 1. The correlations between SWS amount and AI explained very small percentages of variance and were not significant (Figure 5).

**DISCUSSION**

The main finding of the present study is that EEG arousal frequency decreases as a consequence of sleep deprivation. The size of this reduction is larger after total sleep deprivation as compared to selective SWS deprivation. In other words, there is an inverse relationship between sleep recuperative processes following sleep deprivation (greater after total sleep deprivation) and arousal rates. During the recovery nights this affected only NREM sleep stages, while the arousal index remained unchanged during REM sleep. Similar changes were found in sleep stage...
amount between baseline and recovery nights, as reported in Table 1. Both deprivations affected only NREM stage duration and latency, without any significant difference on latency and duration of REM sleep. A similar dissociation between changes of arousal indices during NREM sleep but not during REM sleep has been previously described also with regard to age variations.7 Furthermore, changes of EEG arousal during NREM sleep are not a simple by-product of the increased amount of SWS which, in turn, is characterized by lower AI values, because the reduction of AI during NREM sleep of the recovery nights was positively correlated only with stage 1 and there is no correlation with the SWS amount.

It should be noticed that recovery nights of the present study are also characterized by a concomitant decrease of REM phasic activity, that is rapid eye movements,9,10 and by a negative relation between decrease of rapid eye movements (REMs) and the increase of SWS amount.9,10 The decrease of phasic REM activity deserves some comments, since it has been shown that REMs are a reliable and a sensible index of recuperative variations of Slow-Wave Sleep.9,10,11-14 It could be interesting to assess if they also correlate to EEG arousals. The size of this reduction is larger after total sleep deprivation as compared to selective SWS deprivation.8,10 Furthermore, changes of EEG arousals do not correlate either with tonic (REM duration and latency) and phasic (rapid eye movements) features of REM sleep. With the caution of inferential techniques and scoring system for sleep stages of human subjects. Los Angeles, CA: Brain Information Service, Brain Information Institute, University of California, 1968.

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