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The effects of sleep loss on task performance and the electroencephalogram in young and elderly subjects

F.T.Y. Smulders^{a,*}, J.L. Kenemans^b, L.M. Jonkman^b, A. Kok^a

^aDepartment of Psychology, University of Amsterdam, Amsterdam, The Netherlands ^bDepartment of Psychopharmacology, University of Utrecht, Utrecht, The Netherlands

Abstract

The effects of 28-h sleep loss on performance, reaction time (RT) distribution functions, and spectral composition of the EEG were evaluated in three choice-RT tasks for young (N = 12, aged 18-24 years) and old (N = 12, aged 62-73 years) subjects. Manipulations of stimulus degradation, stimulus-response compatibility, and interstimulus interval variability were to affect encoding, response selection, and motor adjustment stages, respectively. In order to discriminate between independent variables that were presumed to be computational or energetical in nature, effects on EEG spectra and RT-distributions were studied. Spectra of the EEG indicated higher cortical arousal levels for the elderly than for the young. The most dramatic effect of sleep loss on performance was a marked increase in the number of omitted responses. This effect was smaller for the elderly than for the young. The results suggest that the detrimental effects of sleep loss are smaller in the elderly, which is consistent with an inverted-U relationship between arousal and performance. The age effects on the processing stages were mainly limited to response selection. © 1997 Elsevier Science B.V.

Keywords: Aging; Arousal; Performance; Reaction time distribution; Spectral analysis; Electroencephalogram; Sleep loss

^{*} Corresponding author, Maastricht University, Dept. of Psychology, P.O. Box 616, 6200 MD Maastricht, The Netherlands.

1. Introduction

If arousal is considered as an unidimensional construct, there is both evidence for a relative under-arousal and for an over-arousal in the nervous system of the elderly. Various types of evidence on this issue can be discerned. In a review of the literature, Prinz et al. (1990) reported that the spectral composition of the electroencephalogram (EEG) often shows a relative abundance of lower frequencies in the elderly in comparison to young subjects, suggesting a lowered cortical arousal. In most of these clinical studies, the subjects were in a relaxed waking state. In contrast, in other studies, where subjects were engaged in a cognitive task, or were continuously monitored to avoid drowsiness (Duffy et al., 1993), the EEG showed a relative abundance of higher frequencies in the elderly in comparison to young subjects. Secondly, some evidence of a decrease in arousal or excitability is provided by studies showing a reduction in the amplitude of the P300 component of the event-related brain potential (ERP). The elderly show a smaller enhancement of the P300 component of the ERP to novel and unpredictable stimuli as compared to frequent stimuli (Smith et al., 1980; Donchin et al., 1986; Looren de Jong et al., 1989). The latter result can be interpreted in terms of a reduced cortical response to task-relevant stimuli in the elderly (Kok & Zeef, 1991). Thirdly, it has been found that plasma epinephrine levels and free fatty acids are raised in the elderly (Woodruff, 1985), which is a sign of over-arousal. Finally, in response to flash stimuli, ERPs between 90 and 200 ms were found to be larger in the elderly (Dustman & Snyder, 1981), which could be indicative of a loss of inhibition or an increase in arousal as a phasic response to input.

As an alternative to using physiological measures as relatively direct indices of arousal, one may consider task performance, and study interactions between effects of aging and variables that are presumed to affect arousal. The interpretation can then be guided by the inverted-U relation between arousal and performance (Yerkes & Dodson, 1908; Hockey, 1984). Again, however, the results appear to be inconclusive. For example, Davies & Davies (1975) found that speed in a proof reading task was facilitated by noise in an older group, but not in a young group. Presuming the inverted-U relation, this would argue for the old being less aroused than the young. In contrast, Falk & Klein (1978) found that noise impaired performance on a critical flicker frequency task in elderly subjects.

It is possible that these inconsistencies arise because there exist various energetical mechanisms that have specific effects on physiological measures and that are not sensitive to aging in equivalent ways. For instance, Broadbent (1971) proposed, in addition to a lower-arousal mechanism, an upper-arousal mechanism with a controlling and coordinating function. Pribram & McGuinness (1975) proposed a similar distinction, but labelled the coordinating mechanism as 'effort', and split the lower-arousal mechanism into 'arousal', referring to a phasic response to input, and 'activation', referring to a tonic readiness to respond. Whereas these notions mainly concerned physiological responses, Sanders (1983) related them to a computational stage model of information processing based on performance measures. The resulting cognitive-energetical model allowed for a performance-based investigation of effects of sleep loss and other stressors on energetical mechanisms and computational stages (Frowein et al., 1981; Sanders et al., 1982). In this model, an inverted-U relation between energetical supply and performance can still be found: too high or too low levels of arousal or activation should degrade performance if they are not adjusted by the investment of effort.

The present study was to investigate age effects on the multiple energetical mechanisms and computational processing stages in the cognitive-energetical model. To this end, we studied the daytime sequelae of 28-hour sleep deprivation in old and young subjects. In three choice-reaction time tasks, stimulus degradation, stimulus-response (S-R) compatibility, and interstimulus-interval (ISI) variability were manipulated. These task variables were chosen because the additive factors method (AFM; Sternberg, 1969) suggests that they selectively affect the information processing stages of stimulus encoding, response selection, and motor adjustment, respectively (Sanders, 1980; Sanders, 1983). Effects of aging and sleep loss on these stages can be revealed with the use of the AFM by evaluation of the interactions with the proper task variables. In previous studies, sleep loss interacted with stimulus degradation and time uncertainty, most notably near the end of the task, but it was additive with S-R compatibility (Frowein et al., 1981; Sanders et al., 1982). This indicates that sleep loss affects both perceptual and motor processing (encoding and motor adjustment), but not the central processing stage (response selection). In general, if old age is accompanied by a reduction in energetical supply, it will add to these sleep loss effects. Inversely, if old age is accompanied by an increase in energetical supply, it is predicted to counteract the effects of sleep loss.

Sanders (1983) proposed two ways to distinguish between direct effects on computational stages and indirect effects on energetical mechanisms. First, any detrimental effects on energetical mechanisms should increase near the end of the task, whereas direct effects on computational stages should remain fairly constant across a block of trials. Therefore, we will use the time spent on the task as an additional independent variable.

Secondly, it was proposed that computational and energetical effects can be distinguished through their effects on RT-distributions. Specifically, in contrast to computational effects, energetical effects should be largest at the upper end of the RT-distribution, and smaller, if not absent, at the lower end (Sanders & Hoogenboom, 1970). We studied the effects of age, sleep loss and task variables on the distribution of RTs across a block of trials. Effects on RT-distributions will be interpreted in two steps. First, it will be established if supposedly energetical variables (sleep loss, time-on-task) and computational (task-) variables had indeed these specific effects on RT-distributions. If so, the next step will be to use the nature of age effects on RT-distributions as an indication of age effects on computational versus energetical mechanisms.

In addition, the spectral composition of the background EEG was analyzed. Although the precise mechanisms that are involved in the generation of the EEG remain unknown, it is generally assumed that the EEG is strongly influenced by the activity in subcortical structures, like the locus ceruleus, through the projections of various neurotransmitter systems (Lopes da Silva, 1991). These neural mechanisms are also involved in the regulation of tonic and phasic fluctuations in cortical arousal and alertness (see Robbins & Everitt (1995) and Steriade & McCarley (1990) for reviews). Traditionally, a low arousal state is inferred from the relative dominance of large-amplitude low-frequency waves, and higher arousal states from increasing high-frequency low-amplitude waves. Accordingly, beta activity (13-30 Hz) is related to a state of high arousal, theta activity (4-8 Hz) to a state of drowsiness, and delta activity (0.5-4 Hz) to deep sleep (Ray, 1990). These EEG changes have been primarily observed in the analysis of long-epoch EEG during sleep and wakefulness. In the present study, however, the EEG was recorded during the tasks on a trial-by-trial basis in order to directly relate it to variations in task performance. It may be speculated that the EEG will be affected mainly by energetical variables. Effects on the EEG will be interpreted along the same lines as effects on RT-distributions: first, it will be established if supposedly energetical and computational variables can be distinguished by their effects on the EEG. If so, the next step will be to use the nature of age effects on the EEG as an indication of age effects on computational versus energetical mechanisms.

2. Methods

2.1. Subjects

A group of elderly (12 males, aged 62–73 years, mean = 66.8) was recruited by means of an advertisement in a local newspaper. A group of young subjects (12 males, aged 18–24 years, mean = 20.7) consisted of students at the University of Amsterdam; they received course credits in addition to the 50 Dutch guilders that all participants received. All subjects reported to be healthy and to have normal or corrected-to-normal vision. They were screened for normal sleeping behavior via a questionnaire. Average habitual sleep duration was 458 and 417 min per night, for young and elderly subjects, respectively (F(1, 22) = 2.8; NS). The night preceding the normal sleep session (see below), these durations were 449 and 433 min respectively (F(1, 22) < 1, NS).

2.2. Procedure

All subjects participated in three sessions. During the first session, the tasks were practiced. The second and third were experimental sessions held after normal sleep or sleep loss, 1 and 2 weeks after the practice session. The order of sleep and sleep loss sessions was counterbalanced across subjects. In the normal sleep condition, subjects slept at home. In the sleep-loss condition, subjects reported at the laboratory at 21:00 h and were kept awake for the night and the next morning. During this time, they could play games, watch video's or read. In both conditions, the experiment started at noon. Subjects were instructed to abstain from alcohol and coffee starting the evening preceding the experiment, and not to take any day-time

naps on the day and morning preceding the experiment. On each day two subjects were tested. After each 15-min task block, one subject could rest while the other was being tested.

2.3. Design and stimuli

The subject sat alone in a dimly lit room at a distance of 80 cm from a monitor. Three tasks were presented, each in two blocks, corresponding to three factors with two levels each. In the stimulus-degradation task, subjects had to respond to the digit '2', '3', '4', or '5' by pressing one of four buttons. These digits were mapped onto the left hand middle and index finger and the right hand index and middle finger, respectively. Each digit consisted of a dot pattern surrounded by a frame also consisting of dots. Each dot comprised 6×6 pixels on the screen. Digits were degraded by placing 12 dots from the frame at random positions in the field within the frame on positions not occupied by the 14–17 dots of the digit. There were seven degraded versions of each digit. The size of the frame was 23 mm horizontally and 29 mm vertically. Presentation time was 400 ms and the interval between the offset and onset of stimuli varied randomly between 2590 and 3090 ms, following a rectangular distribution. Responses within 2000 ms were taken into account. A central fixation cross was presented during this interval. Intact and degraded stimuli were varied between blocks of 317 trials.

In the S–R compatibility task, a '2' or a '3' was presented at the left or the right of a central fixation cross on the screen. Subjects had to respond with the hand at the side of the digit in the compatible condition, and with the other hand in the incompatible condition. For the left hand, the '2' required a middle finger response and the '3' required an index finger response; for the right hand, the mapping was opposite. The size of the digits was 4.8 mm horizontally and 7.3 mm vertically, and the distance from the fixation cross was 32 mm. A raster of the same size as the digits was presented simultaneously at the contralateral position. S–R compatibility was varied between blocks. The presentation time, number of trials, interval between stimulus onsets, and maximum response time were the same as in the stimulus degradation task.

In the ISI variability task, subjects had to respond with the left index finger upon presentation of the digit '3' and with the right index finger upon the digit '4'. ISI variability was manipulated by either keeping the ISI fixed at 3020 ms for a block of trials, or varying it randomly between 1520 ms and 5680 ms, following a rectangular distribution. Responses within 1160 ms were taken into account. The stimuli were presented in the center of the screen and subtended 9.4 by 18.0 mm. The blocks contained 277 (fixed ISI) or 236 (variable ISI) trials. The stimuli were placed in a frame that remained visible throughout blocks.

The order of tasks (3) and task blocks (2) was counterbalanced across subjects and sessions. Subjects were instructed to react fast and accurately. Training consisted of 100 trials for each task block, which was repeated until the percentage of errors was below 10. Preceding the ISI variability blocks, subjects were instructed to try to anticipate the next trial.

2.4. Physiological recording

The electroencephalogram (EEG) was recorded from Fz, Cz, Pz, and Oz (Jasper, 1958) by means of tin electrodes attached to an electro-cap, referenced to linked earlobes. Bipolar vertical and horizontal electro-oculograms (EOGs) were recorded with tin cup electrodes placed above and below the left eye, and just lateral to the outer canthus of each eye. A ground electrode was placed on the forehead. Electrode impedance was kept below 8 k Ω . The EEG was amplified with a time constant of 5.0 s and a 35-Hz low pass filter, and digitized at 100 Hz for 1.28 s, starting 200 ms before stimulus presentation. Recordings of the EEG on paper were used to ensure that the subjects had not fallen asleep.

2.5. Data reduction

First, incorrect and omitted response-trials were excluded from the database. The RTs of the remaining trials were used for the distributional analyses. Vincentized group reaction time distributions were computed following the procedure proposed by Ratcliff (1979).

Secondly, correct trials with an RT longer or shorter than the mean plus or minus 2.5 standard deviations were excluded from the database. Trials with artifacts (saturation of the AD-converter or an EEG-amplitude greater than 100 μ V) were removed. The remaining trials were used to compute mean RT, and their EEG was used in a spectral analysis. Ocular artifact in the EEG was corrected for by regression analysis in the frequency domain (Woestenburg et al., 1983). For the spectral analysis, the 128-point time series from Fz, Cz, Pz and Oz were submitted to a fast Fourier transform after the average per condition (ERP) was removed. The periodograms were averaged across trials, and the power was computed for the delta (0.8–3.9 Hz), theta (4.7–7.8 Hz), alpha (8.6–11.7 Hz), sigma (12.5–14.1 Hz), beta1 (14.8–21.9 Hz) and beta2 (22.7–29.7 Hz) bands. These values were log-transformed (ln) to normalize their distributions (Gasser et al., 1982).

3. Results

In this section, abbreviations will be used for the independent variables: SL (sleep loss), SD (stimulus degradation), SRC (stimulus-response compatibility), ISI (variability of interstimulus interval), TOTL (time-on-task-linear trend), and TOTQ (time-on-task- quadratic trend). Reported F-values were significant at 5% with (1,22) df, unless stated otherwise.

3.1. Reaction times

Fig. 1 displays the effects on mean RT in the three tasks. In the stimulus degradation task, aging, degradation, and sleep loss led to a slowing of RT (F = 21.2, 189.2, and 5.9, respectively), but these factors did not interact. The effect of degradation decreased near the end of the task (SD × TOTL: F = 11.3).

In the S-R compatibility task, old age, S-R incompatibility and sleep loss led to a slowing of RT (F = 26.5, 94.6, and 7.8). The effect of S-R compatibility was larger in old than in young subjects (F = 7.7). The effects of sleep loss increased during the task (SL × TOTL: F = 5.9).



Fig. 1. The effects of sleep loss and time-on-task on mean reaction time for young and old subjects, as a function of stimulus degradation (upper panel), S-R compatibility (middle panel), and ISI variability (lower panel).

In the ISI variability task, old age, ISI variability and sleep loss all led to a slowing of RT (F = 13.9, 24.8, and 14.2), but these factors did not interact. RT increased during the task (TOTL: F = 30.7), especially from the first to the second period (TOTQ: F = 4.5) and in the sleep loss condition (SL × TOTL: F = 12.9). Furthermore, there was a second order interaction between age, time-on-task and ISI variability (Age × ISI × TOTL: F = 5.7). Fig. 1 suggests that this effect reflects a tendency for the advantage of fixed ISI over variable ISI to increase from the first to the last task period in the young group, while this advantage seems to decrease in the old group.

3.2. Errors

Fig. 2 displays the effects on errors in the three tasks. In the stimulus degradation task, sleep loss and degradation led to an increase in errors (F = 7.0 and 62.8). The degradation effect was larger in the elderly than in the young (Age × SL: F = 5.1), but the elderly did not make more errors on the whole (Age: F < 1). The sleep loss effect increased during the task (SL × TOTL: F = 9.3). Fig. 2 suggests that for young subjects, there was a steep increase in errors from the first to the second task period in the intact/sleep loss condition, with a decrease from the second to the third task period. In the degraded/sleep loss condition, this effect of time-on-task was inverted. In the old, a similar tendency can be seen. These effects are reflected in the following interactions: SD × TOTQ (F =8.4), superseded by SL × SD × TOTQ (F = 8.6).

In the S-R compatibility task, sleep loss and S-R incompatibility led to an increase in errors (F = 4.9 and 30.3), and the elderly made less errors than the young (F = 5.6).

In the ISI variability task, sleep loss and ISI variability led to an increase errors (F = 16.5 and 7.6). More errors were made at the end than at the beginning of the task (F = 5.9). The latter effect was modulated by sleep loss, ISI variability and age group (Age × SL × ISI × TOTL: F = 11.0). Fig. 2 suggests that in the young, ISI variability effects increased in normal sleep conditions, but decreased in sleep loss conditions, while in the old this pattern was just opposite.

3.3. Omissions

Fig. 3 displays the effects on response-omissions in the three tasks. Sleep loss led to a general increase in omissions (F = 17.5). This effect was stronger towards the end of the task (SL × TOTL: F = 22.1), and for degraded stimuli presented to young subjects (Age × SL × SD: F = 5.5).

In the S-R compatibility task, sleep loss led to an increase of omissions (F = 13.3), especially towards the end of the task (SL × TOTL: F = 12.3). This effect tended to be stronger in the young than in the old (Age × SL × TOTL: F = 3.8, 0.05 < P < 0.10).



Fig. 2. The effects of sleep loss and time-on-task on commission-error percentages for young and old subjects, as a function of stimulus degradation (upper panel), S-R compatibility (middle panel), and ISI variability (lower panel).

In the ISI variability task, like in the S-R compatibility task, sleep loss led to an increase of omissions (F = 20.4), especially towards the end of the task (SL × -TOTL: F = 16.6), an effect that tended to be stronger in the young than in the old (Age × SL × TOTL: F = 3.1, 0.05 < P < 0.10).

3.4. RT-distributions

Fig. 4a-c displays the group reaction time distributions for 20% quantiles in the stimulus degradation task, the S-R compatibility task, and the ISI variability task, respectively. For the figures, subjects from both age groups were taken together



Fig. 3. The effects of sleep loss and time-on-task on the percentage of omission-errors for young and old subjects, as a function of stimulus degradation (upper panel), S-R compatibility (middle panel), and ISI variability (lower panel).



Fig. 4. The effects of stimulus degradation (a), S-R compatibility (b), ISI variability (c), and sleep loss on the group reaction time distributions during the first (ToT1) and third (ToT3) 5-min period of the task.

because the analysis of age effects did not reveal significant differences, and only the data from the first and last 5-min period were selected because effects of time-on-task were essentially linear. For a statistical evaluation of effect sizes for responses at the upper and the lower end of the distribution, the mean reaction times of the first and fifth quantile were contrasted in a separate ANOVA, in which a factor labeled 'SPEED' was added to the within-subjects design.

In the stimulus degradation task, the effects of sleep loss, degradation, and time-on-task were larger for relatively slow than for relatively fast responses (F = 11.2, 24.1, and 6.7). The effects of time-on-task were particularly large at the upper end of the distribution for intact stimuli, especially after sleep loss (SD × TOTL × SPEED: F = 6.9; SL × SD × TOTL × SPEED: F = 6.3).

In the S-R compatibility task, the effect of sleep loss was larger at the upper than at the lower end of the distribution, especially near the end of the task (SL × SPEED: F = 8.6; SL × TOT × SPEED: F = 10.8). The effect S-R compatibility was also larger at the upper end of the distribution (SRC × SPEED: F = 81.3).

In the ISI variability task, the effects of sleep loss and time-on-task were larger at the upper than at the lower end of the distribution (SL × SPEED: F = 28.5; TOT × SPEED: F = 32.7). These two effects mutually strengthened each other, especially in the constant-ISI condition (SL × TOT × SPEED: F = 15.3; SL × ISI × TOT × SPEED: F = 9.0). Table 1, row six, gives a gross overview of the most important effects on RT distributions.

3.5. Frequency spectra of the EEG

MANOVAs were conducted for each spectral band. Only if the multivariate effect of 'leads' (Fz, Cz, Pz, Oz) was significant at 5%, post-hoc univariate tests were conducted for each lead; in the interest of space, the multivarate effects are not reported in the text. Figs. 5-7 display the effects on power spectra in different tasks, and Table 1 gives a gross overview of the most important effects. In these figures, data were pooled across levels of the task variable, because task effects were minimal, except in the ISI variability task (see below). Furthermore, the spectra on the Cz and Pz locations are not shown because the effects on Cz, if significant, were similar to the effects on Fz, and any effects on Pz resembled the effects on Oz.

3.5.1. Stimulus degradation task

Fig. 5 displays effects on power spectra in the stimulus degradation task. There was more delta power in young than in old subjects (Age: F = 6.6), and more at the end than at the beginning of the task (TOTL: F = 8.1).

Theta power increased during the task (TOTL: F = 8.1). There was a second-order interaction between age, sleep loss and leads. Post hoc tests indicated that theta power was larger in young than in old subjects at all leads (F = 11.84, 9.6, 9.3, and 7.6 at Fz, Cz, Pz, and Oz), and theta power was increased after sleep loss, but only at Fz and Cz (F = 13.3 and 6.5). Effects of age and sleep loss did not interact at any lead (all *F*-values < 1).

Alpha power increased during the task (TOTL: F = 17.1). Alpha power at Oz was decreased in the elderly (F = 4.7), and alpha at Fz was increased after sleep loss (F = 6.4). Sigma power increased during the task (TOTL: F = 34.7). Sigma power at

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Stimulus degradation (C)	S-R compatibility (C)	ISI variability (C/E)	Sleep loss (E)	Time-on-task (E)	Age (C/E)
EEG-delta EEG-delta EEG-alpha EEG-sigma EEG-betal RT-distribution × TOT	(young f. old [) f	÷€	← ← ← ← ← ←	← ← ← ← ← ← ← ←	→→ 〕
Main effects of independent variables on the on mean R.T. The independent variables wer power in a specific frequency band of the EE mean R.T at the end of the task. For instance it was smaller for old than for young subject up), and the effect of sleep loss on mean RT	frequency spectrum of the e presumed to be compute G. a larger effect on relati e, the EEG power in the d s (arrow down); the effect increased near the end o	EEG, on the distribution ational (C) or energetical vely slow than on fast rest clta band was larger in va of stimulus degradation w f the task (arrow up). We	of reaction times, a (E) in nature. An u onses in the RT dir riable-ISI than in cc as larger at the upj ak or unsteady effe	nd their interaction pward arrow indicates stribution, or an incr onstant-ISI condition per end of the RT-di cts are given in pare	with time-on-task tes an increase of ease of effects on is (arrow up), but istribution (arrow intheses.

Table 1 A gross overview of main effects Fz and Cz was increased after sleep loss (F = 12.0 and 8.0). Betal power increased during the task (TOTL: F = 22.2). Betal power at Fz and Cz was increased after sleep loss (F = 17.1 and 11.7). Beta2 power increased slightly with time spent on the task (TOTL: F = 16.5).



Fig. 5. The effects of sleep loss, time-on-task (three 5-min periods: ToT1, ToT2, ToT3), and electrode position on EEG power spectra (bands: D, delta power; T, theta; A, alpha; S, sigma; B1, beta1; B2, beta2; see text) in two age groups in the stimulus degradation task.



Fig. 6. The effects of sleep loss, time-on-task (three 5-min periods: ToT1, ToT2, ToT3), and electrode position on EEG power spectra (bands: D, delta power; T, theta; A, alpha; S, sigma; B1, beta1; B2, beta2; see text) in two age groups in the S-R compatibility task.

3.5.2. S-R compatibility task

Fig. 6 displays the effects on power spectra in the S-R compatibility task. Delta power was larger in young than in old subjects (F = 9.6). It increased during the task, especially in sleep-deprived young subjects (Age × SL × TOTL: F = 8.1). Theta power was increased in the young (F = 9.4). Theta power at Fz, Cz, and Pz was increased after sleep loss (F = 37.1, 12.8, and 5.6).

Alpha increased during the task (TOTL: F = 18.2). Sleep loss increased alpha power (F = 7.2). At Oz, the increase in alpha power after sleep loss was more pronounced for compatible than for incompatible blocks (F = 5.9). As in the stimulus degradation task, sleep loss increased alpha at Fz (F = 13.7).

Sigma power increased during the task (TOTL: F = 20.3). There was a significant interaction between S-R compatibility and age on Fz and Oz (F = 5.7 at Fz and Oz). In the old, sigma power was smaller in the incompatible condition than in the compatible condition, whereas this difference was negligible, or opposite in the young. Sleep loss led to a increase in sigma power at Fz (F = 5.1).

Betal power increased during the task (TOTL: F = 29.0), especially in young subjects after sleep loss (Age × SL × TOTL: F = 5.6). Betal power at Fz and Cz was increased after sleep loss (F = 10.4 and 4.9). Beta2 power increased during the task (TOTL: F = 23.8).

3.5.3. ISI variability task

Fig. 7 displays the effects on power spectra in the ISI variability task. Delta power was larger in young than in old subjects (F = 8.1). It increased during the task (TOTL: F = 17.2). Furthermore, it was larger in variable-ISI than in fixed-ISI conditions (F = 19.7). Theta power tended to be larger in variable-ISI conditions (F = 4.2, 0.05 < P < 0.10). Theta power was increased in the young and by time spent on the task (F = 10.1 and 13.7). Theta power at Fz and Cz was increased by sleep loss (F = 19.7 and 7.9).

Alpha power increased during the task (TOTL: F = 15.5), except in young subjects in the normal sleep/variable ISI condition (Age × SL × ISI × TOTL: F = 4.5). Sleep loss increased alpha power at Fz and Cz (F = 11.4 and 4.4). Sigma power increased with time spent on the task (TOTL: F = 27.3). Sleep loss increased sigma power at Fz and Cz (F = 11.9 and 4.8).

Beta1 power increased with time spent on the task (TOTL: F = 27.3). This effect was largest at Fz (F = 21.6), but also significant at the other leads (F = 26.2, 9.0, and 7.5, at Cz, Pz, and Oz). Sleep loss increased beta1 power at Fz and Cz (F = 18.9 and 9.2). Beta2 power was increased by sleep loss (F = 10.8). This effect was largest at Fz and Oz (F = 12.9 and 6.3) but also significant at the other leads (F = 11.0 and 5.8, at Cz and Pz). At Cz, the effect of sleep loss was modified by ISI variability (SL × ISI: F = 4.3).

3.5.4. In summary: effects on frequency spectra

Table 1 gives an gross overview of effects on frequency spectra of the EEG. In general, the effect of old age was to reduce the power in the delta, theta, and alpha (SD task only) bands, effects that were rather widespread across the scalp. Sleep loss increased power in the theta, alpha, sigma, and beta1 bands; these effects were mostly predominant at, and often limited to, Fz and Cz. Generally, in all frequency bands and at all scalp sites, the power was larger near the end than at the beginning of the task. All these effects of supposedly energetical variables were relatively independent of the tasks content. At the same time, task variables by themselves had hardly any effect on power spectra, the remarkable exception being an increase in delta power in variable-ISI blocks.

4. Discussion

In the present study, the effects of sleep loss on multiple dependent variables in two age groups were compared. The background EEG was presumed to reflect variations in cortical arousal in a relatively direct manner. Behavioral measures were to index performance and the loci of experimental effects in a stage model.



Fig. 7. The effects of sleep loss, time-on-task (three 5-min periods: ToT1, ToT2, ToT3), and electrode position on EEG power spectra (bands: D, delta power; T, theta; A, alpha; S, sigma; B1, beta1; B2, beta2; see text) in two age groups in the ISI variability task.

Finally, the distributions of reaction times, and the course of effect sizes during the task might serve as criteria for the computational vs. energetical nature of the independent variables.

4.1. Independent variables: computational or energetical in nature?

In principle, there were three ways in which the computational or energetical nature of the independent variables could be manifested. We expected the effects of energetical, but not computational variables to be largest at the upper end of the distribution of reaction times. Secondly, it was expected that energetical variables have larger effect sizes near the end of the task than at the beginning. Both expectations derive from the assumption that the effect of effort, compensating for a suboptimal state, varies across trials, to disappear more often near the end of the task. The spectra of the background EEG were used to provide for a more direct and independent index of any effects on cortical arousal. As to this index, one would not expect substantial effects of computational variables.

As expected, the effects of sleep loss and time-on-task were larger for relatively slow than for relatively fast responses. However, also the effects of typical computational variables like stimulus degradation and S-R compatibility were larger for slow than for fast responses. Thus, we did not find evidence for qualitatively different effects of energetical and computational variables on RT distributions. Only the effects of subject age and ISI-variability were equivalent for fast and slow responses. Although this finding may be interesting by itself, it does not provide a strong argument for either an energetical or computational nature of age effects, in view of the other effects on RT-distributions.

As expected, the effect on mean RT of a typical energetical variable like sleep loss generally increased near the end of the task. The effects of task variables were, in general, not increased near the end of the task. The decrease of effect of stimulus degradation was probably due to residual practice effects that remained in spite of the extensive training received by the subjects. Thus, the dependence of effect sizes on the time spent on the task matched our presuppositions as to the nature of the task and state variables.

As to the effects on the power spectrum of the EEG, the general effect of sleep loss was a marked increase in the theta band at Fz and Cz. In young subjects, frontal theta activity even gained dominance over neighboring bands (Figs. 5–7). The power in higher frequencies (alpha, sigma and beta1) was also increased after sleep loss, but these effects were relatively small. In the beta2 band, sleep loss did not have any effect. Thus, after sleep loss relatively low EEG frequencies were enhanced, which is usually associated with a lowered arousal state. Generally, near the end of the task, the power in all EEG frequencies was increased, not allowing for an interpretation as to the direction of changes in arousal during the task. The effects of old age were opposite to the effects of sleep loss: old subjects showed less EEG power in the lower frequencies (delta, theta and, less markedly, alpha), while in the higher frequencies there were no appreciable differences from young subjects. This observation provided a first clue that old subjects were more aroused than young subjects, at least during the task. Other dependent variables should reveal if this had any implications for performance after sleep loss.

In conclusion, the power spectrum of the EEG was not substantially affected by stimulus degradation and S-R compatibility, but it was very sensitive to sleep loss and time-on-task. This pattern of results is compatible with the notions that: (a) stimulus degradation and S-R compatibility are computational in nature and that, (b) the background EEG selectively indexes energetical mechanisms. In retrospect, the decrease in delta power in fixed-ISI conditions is not surprising: ISI variability, and the analogous experimental manipulations of time-uncertainty and foreperiod duration, may be considered as energetical variables if they exert their effect upon reaction time through the allowance for presetting stages via an energetical mechanism. The observed decrease in delta power in fixed-ISI conditions may well be attributed to the maintenance of an alert state that is associated with the active anticipation of the stimulus. Our analysis of effects on the RT distributions did not indicate a difference between supposedly computational and energetical variables. Thus, the background EEG may be a better indicant of the involvement of energetical mechanisms in the effect of a given factor than the effect of the factor on the shape of the RT-distribution. The validity of using an increase of effect sizes during the task as an indicant of an energetical involvement is probably somewhere in between.

4.2. Sleep loss effects on performance of young and elderly subjects

Subject age, sleep loss and task variables affected mean reaction times by main effects in the expected direction. It was hypothesized that sleep loss effects would interact with stimulus degradation and ISI variability, but no interactions with task variables were observed. This could be taken as evidence that sleep loss and task variables did not have an effect on a common processing stage. However, it cannot be ruled out that they did indeed affect a common stage, but the sleep loss effects were too small to enable a positive interaction. Before accepting that sleep loss and task variables did not affect a common stage, it should be evaluated if the other effects of sleep loss are indifferent to the task content too.

Other effects of sleep loss conformed to expectations: sleep loss effects generally increased during the task, and they were larger at the higher end of the RT-distribution. However, the most dramatic effect of sleep loss on performance was observed in the number of omitted responses. Omissions of response in these choice reaction tasks may well reflect the same mechanisms as lapses of attention in vigilance tasks, which are known to increase markedly after sleep loss (Williams et al., 1965; Wilkinson, 1969; Johnson, 1982). In normal sleep conditions, omissions were practically absent. After sleep loss, the number of omissions was also small during the first 5-min period, but it increased in later task periods. This increase tended to be larger in young than in old subjects in two tasks (S–R compatibility, ISI variability), suggesting a higher sensitivity of young subjects to sleep loss. Further support for this supposition was found in the stimulus degradation task. After sleep loss, young subjects omitted more responses to degraded than to intact stimuli. The latter effect is an example of sleep loss amplifying the negative impact of stimulus degradation on performance, an effect that was expected for mean RT (Sanders et al., 1982). In terms of the stage model an omitted response is probably best described as a failure to complete a stage at all. It is not clear why processing degraded stimuli did not lead to an extra delay due to sleep loss (RT), but only to an extra increase in omissions¹. It could be considered an effect of blocked presentation of intact and degraded stimuli. In the more demanding block with degraded stimuli, energetical supply could have fallen short more often. But then, there were no indications that stimulus degradation affected the background EEG. Possibly, for young subjects, after sleep loss a certain level of energetical supply (arousal) was sufficient for intact but not for degraded stimuli.

In conclusion, the most interesting effect of sleep loss on performance was an increase of the number of omissions. This increase was more marked near the end of the task, agreeing well with the usual finding of an incremental incidence of lapses of attention after sleep loss. In correct-response trials, the effects of sleep loss on performance were not very large, virtually eliminating the possibility of localizing sleep loss' influence on any particular information processing stage. Apparently, the subjects' ability to cope with the effects of sleep loss tended to be an all-or-none phenomenon.

4.3. Age effects on energetical mechanisms

The results indicated that young subjects experienced stronger effects of sleep loss (omissions), and that they were generally less aroused than old subjects during the task (EEG power spectra). Thus, the adverse effects of a decrease in arousal, resulting from sleep loss, were stronger as the initial level of arousal was lower (in young as opposed to old subjects). These findings are compatible with an inverted-U relationship between arousal and performance, if all effects occur on the ascending left part of the function (Hockey, 1984). That the baseline arousal level in young subjects is at this part of the function in these tasks is suggested by the fact that their performance is improved by the increase of arousal associated with caffeine intake (Lorist et al., 1994).

Based on the present data, it is difficult to further distinguish between age effects on the two mechanims involved in the 'lower arousal' mechanism: 'arousal' and 'activation' as used by Pribram & McGuinness (1975) and Sanders (1983). Only the interaction between age and stimulus degradation after sleep loss (omissions, see Fig. 3) could be taken as an indication of an age effect on 'arousal' rather than 'activation', because it concerns input processes. Although there were marked

¹ It could be argued that the occurence of omission-errors is actually an artifact caused by the combination of very long reaction times, and a limited sampling duration of responses in this computer-paced task. However, the plots of the distribution functions (Fig. 4) reveal that this is very unlikely: it can be seen that the maxima in the RT-distributions well preceded the end of response-sampling (after 2 s in the stimulus degradation and S-R compatibility task, and after 1.16 s in the ISI variability task).

differences (mainly in scalp distribution) between sleep loss and age effects on the EEG, the exact relation between EEG spectra and more specific energetical mechanisms has not yet been established, so that no strong conclusions can be drawn.

4.4. Age effects on computational stages

The effects of age on processing stages, based on mean RT results, were observed to be selective. Age effects were augmented in S-R incompatible conditions, but were independent of stimulus degradation and ISI variability. This indicates a selective age effect on the response selection stage, which remained robust after sleep loss. The effects on commission-errors did not invalidate the interaction between effects of age and S-R compatibility on RT. The additivity of age and stimulus degradation on RT, however, was accompanied by an interaction on errors: the elderly made slightly more errors with increasing degradation than the young. The effects of ISI variability on errors were small and equivalent in both age groups. In all, reaction time results suggest that the effect of aging on computational stages was mainly confined to response selection.

4.5. Conclusions

The present study provides evidence that the elderly suffer less from the adverse effects of sleep loss than the young. These results are in line with the findngs of Brendel et al. (1990), who also found smaller effects of sleep loss on mood and performance in the elderly. Furthermore, the present results suggest that EEG spectra are sensitive to energetical, but not computational effects. The finding that relatively low EEG frequencies are reduced in the elderly suggests that their arousal levels are higher than those of young subjects. These results agree with the general finding that in the elderly, in comparison to the young, low EEG frequencies are reduced when subjects are engaged in a cognitive task (Prinz et al., 1990), or when they are carefully monitored to avoid drowsiness (Duffy et al., 1993). The effects of aging on computational processing stages are mainly limited to the central stage of response selection.

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References

Brendel, D.H., Reynolds, C.F.I., Jennings, J.R., Hoch, C.C., Monk, T.H., Berman, S.R., Hall, F.T., Buysse, D.J., & Kupfer, D.J. (1990). Sleep stage physiology, mood and vigilance responses to total sleep deprivation in healthy 80-year-olds and 20-year-olds. *Psychophysiology*, 27, 677-685. Broadbent, D.E. (1971). Decision and Stress. London: Academic Press.

- Davies, A.D.M., & Davies, D.R. (1975). The effects of noise and time of day upon age differences in performance at two checking tasks. *Ergonomics*, 4, 321-336.
- Donchin, E., Miller, G.A., & Farwell, L.A. (1986). The endogenous components of the event-related potential-a diagnostic tool? In D.F. Swaab, E. Fliers, M. Mirmiran, W.A. Van Gool & F. van Haaren (Eds.). Progress in Brain Research, (pp. 87-103). Amsterdam: Elsevier.
- Duffy, F.H., McAnulty, G.B., & Albert, M.S. (1993). The pattern of age-related differences in electrophysiological activity of healthy males and females. *Neurobiology of Aging*, 14, 73– 84.
- Dustman, R.E., & Snyder, E.W. (1981). Life-span-changes in visually evoked potentials at central scalp. Neurobiology of Aging, 2, 303-308.
- Falk, J.L., & Klein, D.W. (1978). Stimulus persistence in CFF: overarousal or underactivation. Experimental Aging Research, 4, 109–123.
- Frowein, H.W., Reitsma, D., & Aquarius, C. (1981). Effects of two counteracting stresses on the reaction process. In J. Long & A. Baddeley (Eds.). Attention and Performance, Vol. IX, (pp. 575–589). Hillsdale, NJ: Lawrence Erlbaum.
- Gasser, T., Bacher, P., & Möcks, J. (1982). Transformations towards the normal distribution of broad band spectral parameters of the EEG. *Electroencephalography and Clinical Neurophysiology*, 61, 181–193.
- Hockey, G.R.J. (1984). Varieties of attentional state: the effects of environment. In D.R. Davies & R. Parasuraman (Eds.). Varieties of Attention, (pp. 449-483). New York, NY: Academic.
- Jasper, H.H. (1958). The ten twenty electrode system of the international federation. Electroencephalography & Clinical Neurophysiology, 10, 371-375.
- Johnson, L.C. (1982). Sleep deprivation and performance. In W.B. Webb (Ed.). *Biological Rhythms*, *Sleep and Performance*, (pp. 111-141). New York, NY: John Wiley.
- Kok, A., & Zeef, E.J. (1991). Arousal and effort: a review and theoretical synthesis of studies of age-related changes in event-related potentials. In C.H.M. Brunia, G. Mulder, & M.N. Verbaten (Eds.). Event-Related Brain Research, (Electroencephalography and Clinical Neurophysiology, Supplement 42, pp. 324-341). Amsterdam: Elsevier.
- Looren de Jong, H., Kok, A., & Van Rooy, J.C.G.M. (1989). Stimulus probability and motor response in young an old subjects: an ERP study. *Biological Psychology*, 29, 125-148.
- Lopes da Silva, F. (1991). Neural mechanisms underlying brain waves: from neural membranes to networks. *Electroencephalography and Clinical Neurophysiology*, 79, 81–93.
- Lorist, M.L., Snel, J., & Kok, A. (1994). The influence of caffeine on information processing stages in well rested and fatigued subjects. *Psychopharmacology*, 113, 411-421.
- Pribram, K.H., & McGuinness, D. (1975). Arousal, activation, and effort in the control of attention. *Psychological Review*, 82, 116–149.
- Prinz, P.N., Dustman, R.E., & Emmerson, R. (1990). Electrophysiology and aging. In J.E. Birren & K.W. Schaie (Eds.). *Handbook of the Psychology of Aging*, 3rd edn., (pp. 135-149). New York, NY: Academic Press.
- Ratcliff, R. (1979). Group reaction time distributions and analysis of distribution statistics. Psychological Bulletin, 86, 446-461.
- Ray, W.J. (1990). The electrocortical system. In J.T. Cacioppo & L.G. Tassinary (Eds.). Principles of Psychophysiology, (pp. 385-412). Cambridge: Cambridge University Press.
- Robbins, T.W., & Everitt, B.J. (1995). Arousal systems and attention. In M.S. Gazzaniga (Ed.). The Cognitive Neurosciences, (pp. 703-720). The M.I.T. Press, Cambridge, Massachusetts.
- Sanders, A.F. (1980). Stage analysis of reaction processes. In G.E. Stelmach & J. Requin (Eds.). *Tutorials in Motor Behavior*, (pp. 331–354). Amsterdam: North Holland.
- Sanders, A.F. (1983). Towards a model of stress and human performance. Acta Psychologica, 53, 61-97.
- Sanders, A.F., & Hoogenboom, W. (1970). On the effects of continuous active work on performance. *Acta Psychologica*, 33, 414–431.

- Sanders, A.F., Wijnen, J.C.L., & van Arkel, A.E. (1982). An additive factor analysis of the effects of sleep-loss on reaction processes. Acta Psychologica, 51, 41-49.
- Smith, D.B.D., Michalewsky, H.J., Brent, G.A., & Thompson L.W. (1980). Auditory averaged evoked potentials and aging: factors of stimulus, task and topography. *Biological Psychology*, 11, 135–151.
- Steriade, M., & McCarley, R.W. (1990). Brainstem Control of Wakefulness and Sleep. New York, NY: Plenum Press.
- Sternberg, S. (1969). The discovery of processing stages: extensions of Donders' method. Acta Psychologica, 30, 276-315.
- Wilkinson, R.T. (1969). Some factors influencing the effects of environmental stresses upon performance. *Psychological Bulletin*, 72, 260-272.
- Williams, H.L., Kearney, O.F., & Lubin, A. (1965) Signal uncertainty and sleep loss. Journal of Experimental Psychology, 66, 401-407.
- Woestenburg, J.C., Verbaten, M.N., & Slangen, J.L. (1983). The removal of eye-movement artefact from the EEG by regression analysis in the frequency domain. *Biological Psychology*, 16, 127–147.
- Woodruff, D.S. (1985). Arousal, sleep and aging. In: J.E. Birren & K.W. Schaie (Eds.), Handbook of the Psychology of Aging, 2nd edn., (pp. 261–295). New York, NY: Van Nostrand Reinhold.
- Yerkes, R.M., & Dodson, J.D. (1908). The relation of strength of stimulus to rapidity of habit formation. Journal of Comparative Neurology and Psychology, 18, 459-482.