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Effect of cognitive arousal on sleep latency, somatic and cortical arousal following partial sleep deprivation

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SUMMARY Emerging research has shown that sleepiness, defined as the tendency to fall asleep, is not only determined by sleep pressure and time of day, but also by physiological and cognitive arousal. In this study we evaluated (i) the impact of experimentally induced cognitive arousal on electroencephalogram (EEG) defined sleep latency, and subjective, somatic and cortical arousal, and (ii) whether experimentally induced cognitive arousal enhances performance on a driving simulator test. Twelve healthy sleepers each spent three nights and the following day in the sleep laboratory: an adaptation, a cognitive arousal and a neutral testing day. In the cognitive arousal condition, a visit of a television camera crew took place and subjects were asked to be interviewed. On each testing day, a 5-min heart rate recording, subjective sleepiness and arousal scales, Multiple Sleep Latency Test and a 25-min driving simulator task were scheduled three times at 2-h intervals. Experimentally induced cognitive arousal resulted in significant increases in objective sleep latency. Significantly elevated levels of subjective and somatic arousal – as indexed by a subjective arousal scale and heart rate – were also evidenced following cognitive arousal induction. A marginally significant trend for increased cortical arousal, measured by EEG beta activity, was also found. No effects were found on driving simulator performance. These findings support the concept of cognitive arousal as a significant component in determining sleep latency. In addition, it was illustrated that cognitively induced arousal can provoke increases in somatic and possibly even cortical arousal in normal sleepers. However, this was not accompanied by an enhanced ability to perform adequately on a driving simulator test.

KEYWORDS arousal, beta electroencephalogram activity, heart rate variability, multiple sleep latency test, sleepiness, stress

INTRODUCTION

Arousal and sleepiness in insomnia patients

Emerging research supports the concept of sleepiness, defined as the tendency to fall asleep, as resulting from a combination of not only sleep pressure – the amount and quality of sleep before and the time awake – and circadian rhythm (Borbély, 1982), and also physiologic and cognitive arousal. The concept of arousal as a major component in determining sleep tendency largely originated in studies on the development and perpetuation of insomnia. Although patients with primary insomnia have reduced sleep time and sleep quality at night, they tend to have longer sleep latencies (Bonnet and Arand, 1995; Stepanski *et al.*, 1988) and enter more slowly into slow wave sleep than healthy sleepers (Hevery *et al.*, 2002). These findings have been related to the increased levels of various forms of arousal observed in insomnia patients: elevated levels of somatic arousal, e.g. measured as metabolic rate (Bonnet and Arand, 1995), cortical arousal, e.g. measured as electroencephalogram (EEG) beta activity (Perlis *et al.*, 2001a), nocturnal sympathetic arousal, as shown by elevations of norepinephrine (Irwin *et al.*, 2003) and increased low frequency spectral power in heart rate variability (HRV) (Bonnet and Arand, 1998a), and

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cognitive arousal, as suggested by the association between stress-related intrusive thoughts and sleep complaints (Hall *et al*., 2000). These observations led to the hyperarousal hypothesis on primary insomnia (Bonnet and Arand, 1997; Harvey, 2002; Perlis *et al*., 2001b), suggesting that increased levels of cortical, somatic and/or cognitive arousal underlie the insomnia symptoms and prevent insomniacs to acquire adequate sleep. This implies that sleep tendency is indeed determined by sleep drive as well as arousal at least in the insomnia population.

Arousal and sleepiness in normal sleepers

There is also other data to support the concept of arousal as a component of sleepiness beyond pathological states and to include it in general models of sleepiness. Experimental studies have established the role of physiologic arousal in determining sleep latency in normal sleepers. Indeed, experimentally induced increases in physiologic arousal – walking as compared with television viewing and the administration of caffeine versus placebo – were found to increase sleep latencies on the Multiple Sleep Latency Test (MSLT) in good sleepers (Bonnet and Arand, 1998b, 2003). The role of cognitive arousal in determining sleepiness within a general population was also extensively studied in correlational studies. Multivariate modelling indicated that psychological distress – considered as an indicator of chronic psychophysiologic arousal – was significantly associated with prolonged sleep latencies as measured by the MSLT (Kronholm *et al*., 1995). Similarly, structural equation modelling suggested a substantial relationship between cognitive arousal symptoms, such as worrying, and sleep onset latencies at night in a sample of the general population (E. De Valck and R. Cluydts, unpublished data). In contrast, experimental research on the effect of cognitive arousal in normal sleepers is scarce and inconclusive. Gross and Borkovec (1982) were the first to report on such an experiment. They found significantly longer sleep onset latencies when subjects were given a speech task on a specific topic before taking a nap in comparison with not getting this task. However, only one nap opportunity was used. In a study by Hall *et al*. (2004), acute stress induced by a speech task resulted in poorer sleep maintenance in normal sleepers. Tang and Harvey (2004) specifically addressed the effect of a similar acute stressor on sleep latency. Unexpectedly, in their first experiment, cognitive arousal induction resulted in shorter objective sleep onset latencies compared with a neutral condition, while in their second experiment, no differences in objective sleep latency were found between a cognitive arousal group (speech threat), a physiologic arousal group (caffeine) and a placebo group. The absence of an expected increase in objective sleep latency following cognitive arousal could be the result of methodological limitations. That is, only one nap opportunity was used to measure sleep onset latency by means of actigraphy, a method shown to have shortcomings in the assessment of sleep onset latency (Pollak *et al*., 2001) and to be less reliable in specific patient groups, including insomniacs (Sadeh and Acebo, 2002). Concerning the impact of cognitively induced arousal on subjective sleepiness, animated video series have been found to result in lower self-rating scores of sleepiness when compared with landscape video series, intended to induce lower levels of cognitive arousal (Hayashi *et al*., 1998).

The different components of an arousal response

A subjective, somatic and cortical component of the arousal response can be distinguished. These responses can be induced both physiologically and cognitively. Here, we focus on cognitively induced arousal. Subjective arousal responses have been observed in response to public speaking (Carrillo *et al*., 2001; Schwerdtfeger, 2004). However, they do not always correspond well with indicators of somatic arousal (Schwerdtfeger, 2004) and are considerably influenced by factors such as gender (Carrillo *et al*., 2001). In the present study, heart rate (HR) and HRV were measured as indicators of somatic arousal, while cortical arousal was assessed by EEG beta activity. HR has been found to be a useful indicator of somatic arousal in response to experimentally induced cognitive-emotional stress (Lundberg *et al*., 2002; Ohsuga *et al*., 2001). In addition, HRV, the variations between consecutive heartbeats, can be used as a quantitative marker of the autonomic nervous system (Camm *et al*., 1996). Spectral analysis of HRV offers both an indicator of parasympathetic activity, derived from the power in the high frequency band (0.15–0.4 Hz), and an indicator of sympathetic activity, inferred from the ratio of low to high frequency power, as power in the low frequency band (0.04–0.15 Hz) represents the combination of parasympathetic and sympathetic activity. Cognitively induced arousal, by performing a psychological test in a competitive setting (Delaney and Brodie, 2000; Siska, 2002) or by a challenging speech task (Hughes and Stoney, 2000), has produced significant increases in sympathetic activity and decreases in parasympathetic activity, as indicated by HRV spectral analysis parameters. Hall *et al*. (2004) found that acute cognitive stress provoked by a speech task was accompanied by analogous changes in HRV during sleep. No data was found on the effect of cognitive stress on HR and HRV pre- and peri-sleep onset. Increases in other parameters of somatic arousal, such as electrodermal activity, have also been observed in response to a public speech task (Schwerdtfeger, 2004). Finally, EEG beta activity has been proposed as an important indicator of cortical arousal, especially in the context of insomnia (Perlis *et al*., 2001b). In patients with sleep-onset insomnia, elevated levels of EEG beta activity have been observed (Freedman, 1986). Yet, it is unknown whether cognitive stress in normal sleepers can provoke heightened levels of cortical arousal around sleep onset too. During wake, EEG beta activity has been related to cognitive function – e.g. attention and perception – in normal subjects (e.g. Lopes Da Silva, 1991).

The ability to stay awake versus the ability to fall asleep

Another issue within the current study considers the impact of cognitively induced arousal on the ability to stay awake as opposed to the ability to fall asleep. Previous research suggests a partial discrepancy between the sensitivity of the MSLT and the Maintenance of Wakefulness Test (MWT) to various sleepiness/alertness manipulations (Sangal *et al*., 1992). We hypothesize that while both the MSLT and MWT measure sleep latency resulting from the combination of sleep drive and arousal, induced arousal has a differential impact on both tests and this accounts for the discrepancy. Specifically, in tests designed to assess the ability to stay awake, arousing factors are already intrinsically present, e.g. sitting up in the MWT, instead of lying down in the MSLT. Previous research showed that the instruction to stay awake and the upright position contribute to increased physiologic arousal and longer sleep latencies in the MWT (Bonnet and Arand, 2001). In turn, a lower starting level of arousal in the MSLT than in the MWT is hypothesized to leave more opportunity for extra arousal inductions to show an effect on the MSLT. This hypothesis was in accordance with the findings of a survey study indicating increases in sleep onset latency at bedtime – a situation of low external stimulation – as cognitive arousal symptoms increased (E. De Valck and R. Cluydts, unpublished data). Conversely, in situations of higher external stimulation, such as while driving a car, cognitive arousal symptoms did not influence sleep tendency. Regarding physiologic arousal however, Bonnet and Arand (1999) showed that increasing levels of physical activity resulted in longer sleep latencies both on the MSLT and the MWT. More research is needed to gain further insight into this matter.

Study aims and hypotheses

Traditionally, cognitive arousal is induced by giving a speech task. In the current study, we opted for a speech task in the form of a television interview. This was intended to ensure subjects not gaining insight into our study objectives with subjects trying to comply with our expectations, avoiding so-called demand characteristics. Indeed, a traditional speech task without a clear objective to the subjects is expected to raise more questions on the study aims in the subjects than the television interview, as in the past our laboratory has already participated in documentary films. This cognitive arousal manipulation could also be present during the whole session without compromising the credibility of the manipulation. It enabled measuring the impact of cognitively induced arousal on objective sleep latency as measured with the MSLT, with three two-hourly sleep latency tests. We expect the level of cognitive arousal induced by the arrival of the camera crew before the start of the first sleep latency test to be highest as this involves the first confrontation with the camera crew. Besides that, the actual television interview of the subject before the start of the last sleep latency test is expected to result in a higher level of cognitively induced arousal than the announcement of the oncoming interview before the second sleep latency test, as this considers only anticipation to a stressful situation, instead of the active participation in such a situation.

It was hypothesized that cognitively induced arousal would give rise to an acute insomnia-like state with increased sleep latencies on the MSLT and elevated levels of somatic and cortical arousal, as assessed by HR/HRV and EEG beta activity respectively. We used a driving simulator test, previously validated as being sensitive to sleep deprivation (De Valck and Cluydts, 2001) and physiologic arousal induced by caffeine (De Valck *et al*., 2003), as a measure of the ability to remain alert under monotonous conditions. The effect of cognitively induced arousal on driving simulator performance was expected to be small or non-existent, as this test provides more external stimulation than the MSLT.

METHODS

Subjects

Subjects were recruited via the electronic newsletter of the Free University of Brussels. Eight males and six females between 20 and 35 years of age (mean = 27, SD = 5.5) were enrolled. Prior to laboratory study, all subjects completed sleep diaries for a period of 1 week. They were informed of the protocol – except for the visit of a television camera crew in the arousal condition of which they were debriefed at the end of the last testing day – gave their informed consent, and were paid for their participation. Inclusion criteria were a regular sleep–wake schedule, no sleep complaints, a good general health, a habitual caffeine usage not exceeding 4 units per day, and having a driving license. Two male subjects were excluded: one suffered from serious sleep onset problems as indicated in his sleep diary, the other got ill during the second testing day. All remaining subjects had a Pittsburgh Sleep Quality Index (Buysse *et al*., 1989) between 0 and 5, indicating that they were moderate to good sleepers. They reported average sleep-onset latency below 20 min – except for one subject with an average sleep onset latency of 31.5 min – they were no habitual short or long sleepers (average total sleep time > 6 h and < 9 h), and their sleeping period was oriented towards the night-time. The study was approved by the Ethics Committee at the Free University of Brussels.

Subjects were asked to maintain a regular sleep–wake schedule and to consume a maximum of 4 units of alcohol and caffeine per day during the course of the study. Compliance with these instructions was checked by self-report. The days before the testing days they were instructed to abstain from caffeine and alcohol from 16:00 hours.

Experimental design

Following an adaptation night with 8 h time in bed, there were two experimental conditions with 3 h time in bed: an arousal and a neutral condition, with and without the visit of a television camera crew respectively. Each subject participated in the three testing days – an adaptation, an arousal and a neutral testing day – with an interval of 4–7 days. The two experimental conditions were counterbalanced.

Procedure

Subjects arrived at the laboratory at 22:00 hours on evenings preceding the testing days. They participated in the study in groups of two individuals. In the evening recreational activities such as watching television, reading, conversation or playing round games were allowed. On the first evening a 25-min test practice session on the driving simulator was performed. Subjects went to their rooms and lights were switched off at 23:00 hours on adaptation nights and at 04:00 hours on experimental nights. In the morning, subjects were awakened at 07:00 hours, after which they had a shower and a caffeine-free breakfast. Again, free time could be spent at choice with recreational activities, such as reading and conversation. Subjects did not leave the laboratory during the day and physical activity was kept to a minimum. HR was measured three times – at 08:45, 10:45 and 12:45 hours – during 5 min while subjects were sitting down. Sleepiness and arousal scales were completed following the HR measurements at 08:50, 10:50 and 12:50 hours. The MSLT took place at 09:00, 11:00 and 13:00 hours, each time followed by a 25-min driving simulator tests. HR was measured during naps. A caffeine-free lunch was scheduled at 12:00 hours.

In the arousal condition, a research assistant arrived at 07:50 hours, announced the visit of a television camera crew and asked the subjects if they were willing to participate in a documentary film on sleep and related issues. The camera crew arrived at 08:20 hours and filmed the subjects while EEG electrodes were being attached and while they were waiting for the first HR measurement at 08:45 hours. During the MSLT naps and the driving simulator tests, the camera crew waited in a separate room. At 10:00 hours, after subjects had completed the first test block – HR, sleepiness and arousal scales, MSLT nap and driving simulator test – an interview with a sleep expert took place in the laboratory. At 10:35 hours the subjects were asked whether they agreed being interviewed on their motivation and experience as participants of sleep research, and interview questions were shortly discussed. Following the second test block starting at 10:45 hours and the lunch at 12:00 hours, an interview of the subjects took place. The camera crew left at 12:45 hours – at the start of the third test block – explaining that they would probably have to return after the last test block to film some events for a second time because of some technical problems. Subjects were informed on the true objective of the visit of the television camera crew at the end of the third testing day. They were also asked whether they believed the camera crew was making a documentary film and that they had given genuine interviews. An overview of the experimental procedure can be found in Table 1.

Table 1 Overview of the experimental procedure

Time (hours)	*Activity*
22:00	Subjects arrive at the laboratory. Adaptation night: 25-min practice session on the driving simulator Free time
23:00	Bedtime on adaptation night
04:00	Bedtime on experimental night
07:00	Subjects are awakened. Shower and breakfast Free time
07:50	**Announcement of the visit of a television camera crew**
08:20	**Arrival of a television camera crew** **Subjects are filmed while EEG electrodes are attached and while waiting.**
08:45	5′ heart rate recording while sitting
08:50	SSS and POMS
09:00	Sleep latency test
09:30	25′ driving simulator test
10:00	**Interview with sleep expert**
10:35	**Request for interview with subjects and discussion of interview questions**
10:45	5′ heart rate recording while sitting
10:50	SSS and POMS
11:00	Sleep latency test
11:30	25′ driving simulator test
12:00	Lunch
12:25	**Interview with subjects**
12:45	5′ heart rate recording while sitting
12:50	SSS and POMS
13:00	Sleep latency test
13:30	25′ driving simulator test
14:00	Third testing day: debriefing of subjects on true objective of visit of camera crew Subjects are discharged. In case of extreme sleepiness, a nap is recommended.

Activities in bold only took place in the arousal condition.
MSLT, Multiple Sleep Latency Test; POMS, Profile of Mood States; SSS, Stanford Sleepiness Scale.

Measurements

Multiple Sleep Latency Test

The MSLT was conducted according to the standard protocol (Carskadon *et al*., 1986), except that there were only three two-hourly sleep latency tests. EEG recordings were scored in 30-s epochs using Rechtschaffen and Kales (1968) criteria. Subjects were awakened after three consecutive 30-s epochs of stage 1 sleep or one epoch of any other sleep stage.

Subjective sleepiness and arousal

The Fatigue subscale of the Profile of Mood States (POMS) and the Stanford Sleepiness Scale (SSS) (Hoddes *et al*., 1973) were used to measure subjective sleepiness. Subjective arousal was assessed by the Tension subscale of the POMS (MacNair *et al*., 1971). In both the SSS and the POMS, higher scores were indicative of a higher intensity of the construct measured.

HR and HRV

For 5 min while sitting at the start of each test block and during the sleep latency tests, heart interbeat intervals were registered at a rate of 500 samples per second with a Polar S810 device (Polar Electro OY, Kempelee, Finland). This device uses band pass filtering and adaptive threshold methods for QRS detection. The inaccuracy of the R–R measurement has been reported to be < 3 ms, which is sufficient for HRV analysis (Kinnunen and Heikkilä, 1998). The smoothness prior-based approach was used to remove the low frequency trend component in the R–R series. After collection, R–R intervals deviating more than 200 ms from the mean R–R interval in the selected detrended segment were removed. The HRV analysis software V1.1 (Biomedical Signal Analysis Group, 2002) was used to assess mean HR, the power in the high frequency band (HF; 0.15–0.4 Hz) – expressed in normalized units – and the ratio LF/HF, with LF referring to the power in the low frequency band (0.04–0.15 Hz), using spectral analysis of the R–R interval, based on the fast Fourier transform. These parameters will be reported for 3-min periods (starting 1 min after the beginning of the recording) while subjects were sitting and for 5-min periods (starting 1 min after the beginning of the recording) during the sleep latency tests. Shorter periods, with a minimum of 3 min, were selected during the sleep latency tests when subjects fell asleep in < 6 min. This was to prevent including HR data from subjects falling asleep, as the initiation of stage 1 sleep is known to result in decreased HR (Carrington *et al*., 2003; Trinder *et al*., 2001).

Electroencephalogram

The EEG was recorded during sleep latency tests (C3-A2, O2-A1) at a sampling rate of 256 Hz using the Procomp+ device and analysed with Biograph V2.1 software (Thoughttechnology Ltd and Mind Media BV, 2000). The first and last 5-min artefact-free and movement-free segments after removal of the first minute of the recording of each sleep latency test were selected. These were subjected to finite impulse response routines. Absolute power (mV^2) in the theta (4–8 Hz), alpha (8–12 Hz), sigma (12–14 Hz), beta-1 (14–20 Hz) and beta-2 (20–35 Hz) bandwidths was computed for both EEG sites.

Driving simulator performance

The computer program Drivesim 3.00 of the York driving simulator (York Computer Technologies, Kingston, Ontario, Canada) was used to assess driving performance. A detailed description of the driving simulator device and the driving task – except for the duration of the driving task being 25 min in the current study instead of 45 min – can be found in De Valck *et al*. (2003).

Three variables were analysed to assess driving performance (i) lane drifting, the standard deviation of the road position in centimeter, (ii) speed deviation, the mean deviation from the posted speed limit in kilometer per hour, (iii) accident liability, being 1 if the car left the road or hit another vehicle at least once during the drive, 0 if this was not the case. Data of the first 5 min of each drive were not included in the analyses, to allow adaptation to the driving task to develop.

Statistical analysis

The MSLT sleep latencies, EEG absolute power in the specified bandwidths, HR, HRV (LF/HF ratio and HF power), subjective sleepiness and arousal scale scores (POMS and SSS), and driving simulator performance (lane drifting and speed deviation) were analysed using repeated measures ANOVA in which condition (cognitively induced arousal, neutral) and time of day (first, second and third test block) were extracted as within-subject variables. When appropriate, further comparisons were carried out using Scheffé *post hoc* tests. The significant effects found for the subjective rating scales (POMS and SSS) were checked using Friedman ANOVA, the nonparametric alternative to repeated measures ANOVA, as it considered ordinal data. The dichotomous variable accident liability was analysed by a Cochran *Q*-test. To evaluate the relationships between somatic, cortical and subjective arousal and objective sleep latency, correlations were calculated between HR, HRV, EEG absolute beta power, scores on the subjective arousal scale, and objective sleep latency. These parameters were averaged across the three measurements of the arousal condition. All reported results refer to differences at the 0.05 significance level, except where noted otherwise.

RESULTS

Cognitive arousal induction

All subjects were filmed and believed that the camera crew was making a documentary film, including genuine interviews. Two subjects refused being interviewed, but they were present during the interview of the other subject in their group.

Multiple sleep latency test

A significant main effect for condition was found [$F(1,11) = 8.78$, $P < 0.02$], indicating that mean sleep latency in the cognitive arousal condition (mean = 15.54, SE = 1.30) was longer than in the neutral condition (mean = 11.50, SE = 1.05). Neither a main effect of time of day, nor an interaction effect of condition and time of day was observed. Sleep latency data across the day under arousal and neutral conditions are plotted in Fig. 1.

Mean sleep latency following the adaptation night was 15.43 min (SE = 1.34).

Subjective sleepiness and arousal

Considering subjective sleepiness (SSS and subscale fatigue of the POMS), there was only a significant main effect for time of day for the scores on the SSS [$F(2,22) = 18.47$, $P < 0.0001$].

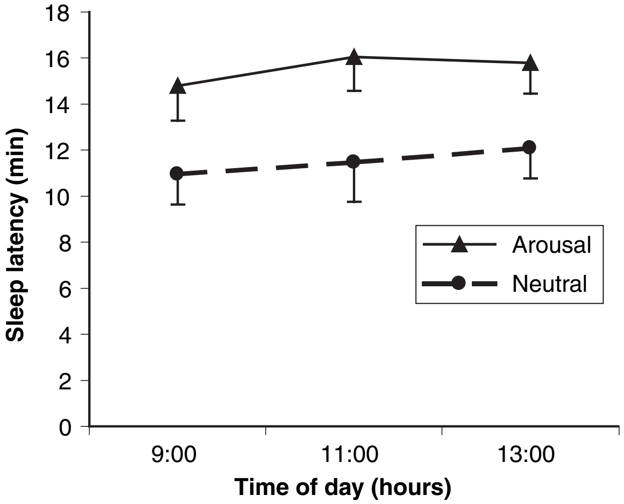

Figure 1. Sleep latencies across the day under cognitive arousal and neutral conditions. Vertical bars denote standard errors.

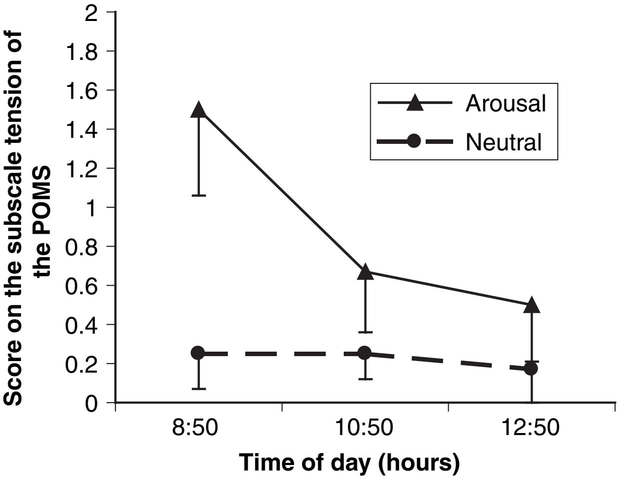

Figure 2. Mean scores on the subscale Tension of the POMS across the day in the cognitive arousal and neutral condition. Vertical bars denote standard errors.

Subjective sleepiness levels were highest at the first test (mean = 3.33, SE = 0.39) ($P < 0.05$), decreased at the second test (mean = 2.71, SE = 0.37) ($P < 0.05$), and were lowest at the third test of the day (mean = 2.21, SE = 0.29) ($P < 0.05$). No other main or interaction effects on subjective sleepiness were observed.

A significant main effect of time of day was observed for the scores on the subscale Vigor of the POMS [$F(2,22) = 6.34$, $P < 0.01$]. Vigor scores were higher during the last testing (mean = 6.79, SE = 1.18) as compared with the first (mean = 5.04, SE = 0.98) ($P < 0.01$) and the second testing of the day (mean = 5.54, SE = 1.24) ($P < 0.10$).

Subjective arousal, as assessed by the subscale Tension of the POMS, was significantly higher in the arousal (mean = 0.89, SE = 0.32) versus the neutral condition (mean = 0.22, SE = 0.14), suggesting that the arousal manipulation was successful [$F(1,11) = 9.43$, $P < 0.05$]. In addition, a main effect of time of day was found [$F(2,22) = 6.92$, $P < 0.01$]. Subjective arousal scores were higher at the first testing than at the second ($P < 0.05$) and third testing of the day ($P < 0.01$). A significant condition by time of day interaction effect [$F(2,22) = 9.47$, $P < 0.01$], suggested that the time of day effect described, was only present in the arousal condition. Figure 2 illustrates these findings.

All significant effects found, were confirmed by nonparametric Friedman ANOVA.

HR and HRV

For nine of the 12 subjects, the HR recordings while sitting could be used (three technical failures). This revealed a significant main effect of condition for mean HR [$F(1,8) = 6.99$, $P < 0.05$], suggesting elevated HR in the arousal condition (mean = 78.31, SE = 4.05) as opposed to the neutral condition (mean = 74.28, SE = 3.80). Additionally, a significant main effect of time of day on mean HR was found [$F(2,16) = 9.37$, $P < 0.01$], indicating that HR was lower in the second testing as compared with the first ($P < 0.06$) and the third testing of the day ($P < 0.01$). There was no significant interaction effect between condition and time of day. Figure 3 illustrates the main effects of condition and time of day on mean HR. None of the HRV parameters yielded significant main or interaction effects.

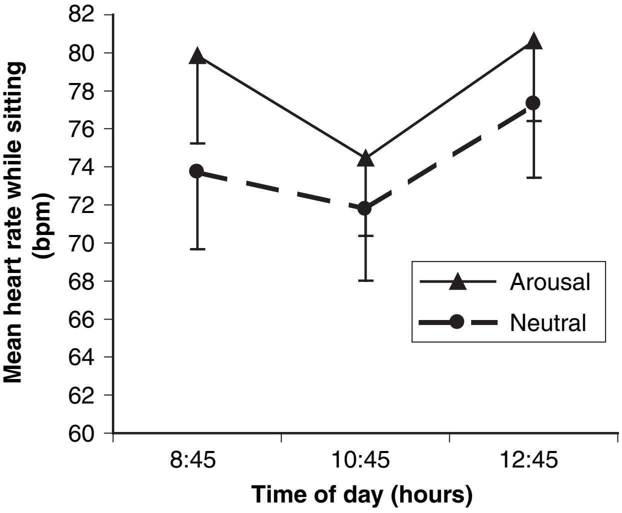

Figure 3. Mean heart rate while sitting across the day in the cognitive arousal and neutral condition. Vertical bars denote standard errors.

The HR recordings at the beginning of the MSLT naps were only complete for three of the 12 subjects, so no statistical analysis could be performed on these data.

Electroencephalogram

Within the first 5-min segments of the MSLT naps, there was a trend for the absolute beta-2 power (20–35 Hz) at C3A2 to be higher in the cognitive arousal condition (mean = 202.31, SE = 73.43) as opposed to the neutral condition (mean = 167.90, SE = 56.94) [$F(1,11) = 3.27$, $P < 0.10$]. This was accompanied with a decrease in absolute theta power in the arousal condition (mean = 93.85, SE = 22.18) as compared with the neutral condition (mean = 107.34, SE = 22.55) [$F(1,11) = 5.78$, $P < 0.05$]. There was no main effect of time of day or a significant interaction effect for beta-2 and theta power. No significant effects were found for the absolute power in any of the other bandwidths.

A similar trend for absolute beta-2 power (20–35 Hz) at C3A2 was observed within the last 5-min segments of the MSLT naps, with higher levels of beta-2 power in the cognitive arousal condition (mean = 148.59, SE = 25.04) in comparison with the neutral condition (mean = 117.75, SE = 15.04) [$F(1,11) = 4.03$, $P < 0.07$]. No other main or interaction effects were found for beta-2 power or the absolute power in the other bandwidths.

A graphical presentation of the beta-2 power in the first and last 5-min segment of the MSLT naps in the arousal and neutral condition is presented in Fig. 4.

HR, HRV, EEG beta power, subjective arousal and MSLT

None of the correlations between HR, HRV, EEG absolute beta power, scores on the subjective arousal scale, and objective sleep latency were significant, with the exception of the mutual correlations between HR and HRV parameters.

Driving simulator performance

Driving performance parameters lane drifting and speed deviation yielded no main or interaction effects of condition and time of day. In addition, there were no significant differences in accident liability between the experimental conditions, as indicated by the Cochran Q-test [$Q(5) = 3.33$, NS]. Accident frequency and means and standard errors for lane drifting and speed deviation in the neutral and arousal condition are given in Table 2.

To exclude the possibility that arousal only had an effect on driving performance in the beginning of the task and faded during the remaining of the drive, data of minute 1 to minute 6 of the driving task were analysed. This analysis showed that there was no effect of cognitively induced arousal in the beginning of the task either [$F(1,11) = 1.84$, NS for lane drifting; $F(1,11) = 1.95$, NS for speed deviation].

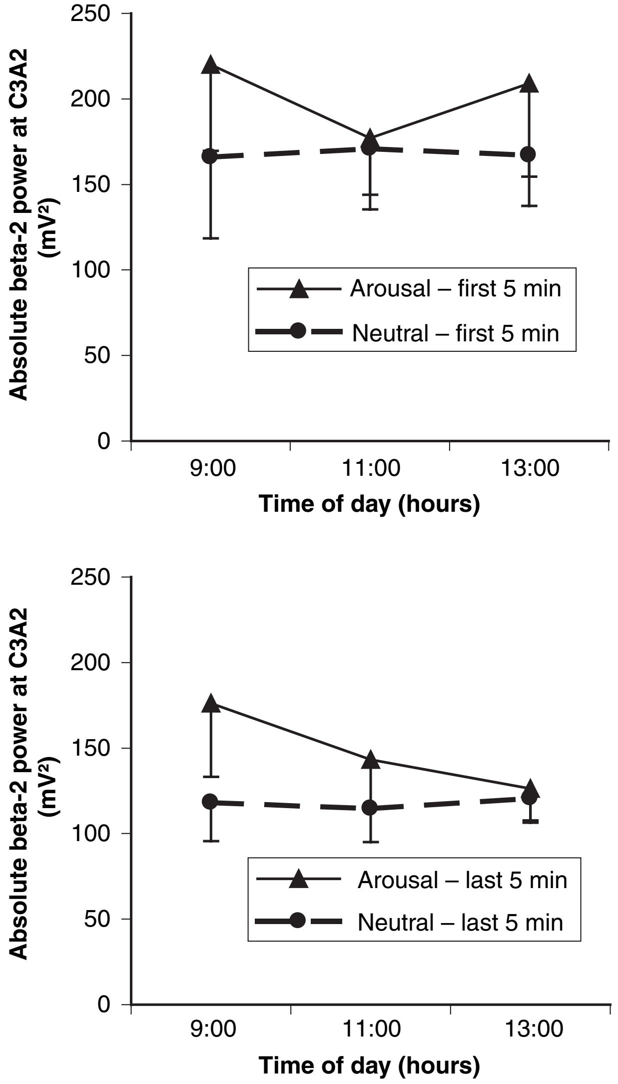

Figure 4. Absolute beta-2 power at C3A2 during the first (top) and the last (bottom) 5 min of the MSLT naps across the day under cognitive arousal and neutral conditions. Vertical bars denote standard errors.

Table 2 Mean values and standard errors for lane drifting and speed deviation and accident frequency in the arousal and neutral condition

	Lane drifting (cm)		*Speed deviation (km h^{-1})*		
Condition	*Mean*	*SE*	*Mean*	*SE*	*Accident frequency (%)*
Arousal	36.42	4.74	5.03	1.21	6
Neutral	35.94	4.26	5.64	1.64	11

Accident frequency is the percentage of sessions in which at least one accident occurred.

DISCUSSION

Experimentally induced cognitive arousal resulted in statistically significant increases in sleep latency as measured by the MSLT. During the visit of the television camera crew, subjects took on average 4 min more to fall asleep than in the neutral condition. The magnitude of this change approximates that seen in normal subjects being partially sleep deprived in the sleep laboratory following physiologic arousal – brief walk (sleep latency = 7.9 minutes) versus watching television (sleep latency = 2.9 minutes) (Bonnet and Arand, 1998b).

Although the study was not designed to assess the impact of partial sleep deprivation on objective sleepiness, the findings suggest there was an increase in sleep latency (4 min) in response to the sleep deprivation in the neutral condition as compared with the adaptation night. Equal changes in sleep latency have been observed after sleep restriction to 3–5 h as compared with 8 h sleep at night by other researchers (Devoto *et al*., 1999). However, the average sleep latency in absolute terms after 8 h time in bed (15.43 min) and after 3 h time in bed (11.50 min) was elevated in comparison with previous studies (Bonnet and Arand, 2001; Drake *et al*., 2001). We consider it unlikely that these elevated sleep latencies are a consequence of insomnia problems in our subjects because in the sleep diaries no insomnia complaints are reported. Possibly, the data indicate a higher – although not pathologically high – baseline sleep latency in general in our subjects.

The results on the subjective arousal scale suggested that the subjects indeed experienced the situation in the arousal condition as stressful. Moreover, there were indications that this subjective arousal was accompanied by cortical and somatic components of arousal. There was a marginally significant trend towards increased EEG levels of beta-2 (20–35 Hz) power while trying to fall asleep during the MSLT naps in the cognitive arousal condition. This trend seemed to be maintained throughout the naps, as it was found during both the first and the last 5-min segment of the naps. Elevated levels of beta-2 power have also been observed in patients with primary insomnia as opposed to secondary insomnia and good sleeper controls, although these patients also showed increases in beta-1 (14–20 Hz) and gamma activity (35–45 Hz) [Perlis *et al*., 2001a]. In insomnia patients these findings have raised the hypothesis that insomniacs suffer from the inability to disengage cognitive processes around sleep onset (Perlis *et al*., 2001b). The current study suggests that this inability can be present in normal subjects too in the context of acute stress. However, it should be stressed that the findings on cortical arousal considered only marginally significant trends, so replication of these findings is needed.

Elevated levels of somatic arousal were indicated by increases in mean HR during sitting in the cognitive arousal condition. The observed average increase in HR (4.0 bpm) was larger than that seen in normal sleepers following a walk as opposed to TV viewing (2.4 bpm) in an experiment on the effect of physiologic arousal on sleep latency (Bonnet and Arand, 1998b). However, a rise in HR of this size could be expected as subjects engaged in stressful cognitive tasks have been found previously to experience HR increases of 6 bpm (Ohsuga *et al*., 2001) to 8.6 bpm (Lundberg *et al*., 2002). In contrary to what was expected, we did not find a significant impact of cognitively induced arousal on HRV while subjects were sitting. As the other somatic and the cortical arousal parameters suggested stress was successfully induced in our subjects, we consider it likely that the absence of an effect on HRV was because of a methodological limitation. Namely, it has been shown that even slight movements – not affecting HR – can substantially increase the low-frequency components of spectral analysis of HRV (Fortrat *et al*., 1999). Such minor movements might have obscured the effects on HRV in our study. Furthermore, HR data during naps could not be analysed because of the large amount of missing data. It seems that the polar device used is not applicable for HR and HRV measurement during sleep, although it is a useful and cost-effective tool for HR and HRV assessment of subjects who are sitting up quietly.

Taken together, the present research findings seem to imply that cognitively induced arousal was able to induce an acute insomnia-like state in normal sleepers, with increased sleep onset latencies and subjective, somatic and cortical arousal. In patients with primary insomnia, sleep-onset problems have been found to be accompanied by elevated levels of subjective, cortical and somatic arousal too. Present findings imply a possible causal role of cognitive arousal in insomnia, instead of cognitive arousal merely being epiphenomenal to sleeplessness. Obviously, this should not be misinterpreted as excluding alternative causal paths in chronic insomnia. For instance, Perlis *et al*. (2001b) suggested that in chronic insomnia cortical arousal might occur simply as a conditioned phenomenon in the absence of a situational stressor.

More generally, these findings support the concept of cognitive arousal as a main component of objective sleepiness. This seems to apply for the passive involvement in a stressful environment (filming of the subjects prior to the first nap test), the anticipation to a stressful situation (announcement of an oncoming interview of the subjects prior to the second nap test), and the active involvement in a stressful situation (giving an interview for television prior to the third nap test).

We chose to use the MSLT to assess the effects on objective sleep latency, as this represents the best validated and a reliable measure of sleepiness (e.g. Carskadon and Dement, 1982). Whereas the finding of a significant effect of physiologic arousal on the MSLT resulted in a recommendation to carefully monitor the activity level of subjects prior to MSLT evaluations (Bonnet and Arand, 1998b), the present findings are more difficult to be translated into practical guidelines. Indeed, controlling for cognitive arousing elements is less evident, because such elements are not always visible to or under control of the experimenter. At a minimum, present findings should encourage researchers to be aware of the impact of cognitive arousal on MSLT testing. While it is recognized that a minimum of four naps is recommended within the standard procedure of the MSLT, the present study used only three naps at 2-h intervals, as the cognitive arousal manipulation, the visit of the television camera crew, did not allow extending the testing procedure for another 2 h. However, this is not a major shortcoming as Zwyghuizen-Doorenbos *et al*. (1988) found high test–retest reliability for MSLT procedures with three or more naps.

Whereas cognitively induced arousal had clear and substantial effects on the ability to fall asleep, no impact was found on driving simulator performance. This supports the hypothesis that cognitively induced arousal only impairs the ability to fall asleep in situations of low external stimulation, while it does not enhance the ability to stay awake in situations of higher external stimulation. At first glance, this conclusion appears contradictory to experiments showing that physiologic arousal both decreases the ability to fall asleep (MSLT) and increases the ability to stay awake (MWT) (Bonnet and Arand, 1998b, 1999). It could be suggested that this discrepancy results from the use of different, non-equivalent tests of the ability to stay awake. Indeed, subjects are more active during the driving simulator test than during the MWT. In line with our hypothesis, higher starting levels of arousal within the individual during the simulator test when compared with the MWT, leave less opportunity for the extra arousal induction to show its effect. However, a previous study showed that the impact of arousal induced by the administration of caffeine could be evidenced on the driving simulator test (De Valck and Cluydts, 2001). Alternatively, it may be that different forms of arousal are induced and responsible for the inconsistent findings. That is, physiologically induced arousal and not cognitively induced arousal has an impact on the ability to stay awake. In this context, the suggestion could be offered that physiologic and cognitive arousal have a qualitatively different influence on our sleep–wake regulation mechanisms.

Another important issue within the study of arousal influences on sleepiness, considers the interrelation of the different forms of arousal, e.g. subjective, somatic and cortical arousal. It remains to be established whether these various forms of arousal are fundamentally different or if they are largely intercorrelated or even interchangeable. Contrary to previous studies (Bonnet and Arand, 1999, 2000), we did not find significant correlations between subjective, somatic and cortical arousal and objective sleep onset latency. This could simply be related to the relatively small sample size, providing less power to illustrate potential effects. However, Bonnet and Arand (1999, 2000) did find consistent relationships between HR and nap latency with only 12–14 subjects following physiologic arousal. The divergent finding of the current study could be explained by induced physiologic arousal having a more profound impact than induced cognitive arousal, making the relationship between the different aspects of arousal and sleep latency less clear in the case of induced cognitive arousal. As discussed, however, the magnitude of change in sleep latency and HR in our study was comparable or even larger than that found in studies of physiologic arousal. A third possible explanation relates to the hypothesis of the multidimensionality of arousal. This hypothesis suggests that relatively distinct forms of arousal can be distinguished because different response mechanisms are involved in the production of an aroused state (Perlis *et al.*, 2001b; Tang and Harvey, 2004). Potentially, important individual differences exist in the way the various response systems differentially react to cognitively induced arousal. This might explain the inconsistent pattern of subjective, cortical and somatic responses to the arousal induction with some subjects for instance responding with somatic arousal and others predominantly with cortical arousal.

In conclusion, the present findings indicate the need for recognizing the role of cognitively induced arousal in determining sleepiness as measured with the MSLT and in theoretical models of sleepiness. It also shows that cognitively induced arousal in itself can provoke increases in somatic arousal as evidenced by elevated HR, and a trend towards higher EEG beta activity was found. Finally, the current study proposes that cognitively induced arousal does not enhance the ability to stay awake and perform adequately, as assessed by a driving simulator task. Future research should consider how the level of external stimulation in a sleepiness test determines the likelihood of observing an impact of cognitively induced arousal on sleep tendency. Also, more research is needed on the impact of cognitively versus physiologically induced arousal on the different response systems of arousal.

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