Aircraft noise effects on sleep: A systematic comparison of EEG awakenings and automatically detected cardiac arousals

Mathias Basner[1*], Uwe Müller[1], Eva-Maria Elmenhorst[1], Barbara Griefahn[2]

1 German Aerospace Center (DLR), Institute of Aerospace Medicine, 51170 Köln, Germany

2 Institute for Occupational Physiology at Dortmund University, Ardeystr. 67, 44139 Dortmund, Germany

*corresponding author: e-mail: mathias.basner@dlr.de

ABSTRACT

OBJECTIVES: Polysomnography is the gold standard for investigating noise effects on sleep, but data collection and analysis are sumptuous and expensive. We recently developed an automatic algorithm for the identification of cardiac activations associated with cortical arousals, which uses heart rate information derived from a single electrocardiogram (ECG) channel (Basner et al. 2007a). We hypothesized that cardiac arousals can be used as estimates for EEG awakenings.

METHODS: Polysomnographic EEG awakenings and automatically detected cardiac activations were systematically compared using laboratory data of 112 subjects (47 male, mean ± SD age 37.9 ± 13 years), 985 nights and 23,855 aircraft noise events (ANEs).

RESULTS: The overall agreement was higher in control (81.9 %) compared to noise nights (76.4 %). However, if corrected for chance expected agreement according to Landis and Koch (1977), agreement was higher in noise (κ=0.60) compared to control nights (κ=0.33), representing “moderate to substantial” and “fair” agreement respectively. The probability of automatically detected cardiac arousals increased monotonously with increasing maximum sound pressure levels of ANEs, exceeding the probability of EEG awakenings by up to 18.1 %. If spontaneous reactions were taken into account, exposure-response curves were practically identical for EEG awakenings and cardiac arousals.

CONCLUSIONS: Automatically detected cardiac arousals can be used as estimates for EEG awakenings. This inexpensive, objective, and non-invasive method facilitates large scale field studies on the effects of traffic noise on sleep. More investigations are needed to further validate the ECG algorithm in the field and to investigate inter-individual differences in its ability to predict EEG awakenings.

INTRODUCTION

There is no doubt that noise in general and aircraft noise specifically disturb sleep (Muzet 2007). These disturbances can be characterized by changes in the composition of sleep stages and by a fragmentation of sleep. Although there is still debate, awakenings are usually considered an adequate indicator of noise induced sleep disturbances (Basner et al. 2006; Griefahn et al. 2008; Ollerhead et al. 1992).

Polysomnography, i.e. the simultaneous recording of the electroencephalogram (EEG), the electrooculogram (EOG), and the electromyogram (EMG) remains the gold standard for measuring and evaluating sleep. According to specific conventions (Iber et al. 2007; Rechtschaffen et al. 1968), the night is divided into 30-s epochs. Depending on EEG frequency and amplitude, specific patterns in the EEG, muscle tone in the EMG, and the occurrence of slow or rapid eye movements in the EOG, different stages of sleep are assigned to each epoch. Shorter activations in the EEG

and EMG, so-called *arousals*, can be detected with the polysomnogram (Bonnet et al. 2007; Iber et al. 2007). These arousals are usually accompanied by activations of the autonomic nervous system (Basner et al. 2007a; Sforza et al. 2004).

However, polysomnography also has some disadvantages. EEG, EOG, and EMG electrodes and wires are somewhat invasive and may therefore influence sleep. The instrumentation of subjects is cumbersome and cannot be done by the subjects themselves. Finally, sleep stage classification requires trained personnel, and is known to be associated with high inter- and intra-observer variability (Drinnan et al. 1998; Loredo et al. 1999). Hence, only a few polysomnographical noise effects studies with relatively small sample sizes have been conducted in the past (see Basner & Samel (2005) for a short review).

It is absolutely legitimate to use methods other than polysomnography (e.g. questionnaires, actigraphy, or signalled awakenings) in order to gather information on the effects of noise on sleep. However, one has to be aware that these alternative methods never cover all aspects of sleep. Relevant changes in sleep structure may be overlooked, while changes may be indicated without a relevant correlate in the CNS. Nevertheless, it would be highly desirable to have a method with low methodological expense (self administered by the subjects, automatic objective analysis) and high validity compared to the gold standard polysomnography.

We recently developed an ECG-based algorithm for the automatic identification of cardiac activations associated with cortical arousal (Basner et al. 2007a). This algorithm uses beat-to-beat information derived from a single ECG channel to automatically calculate the beginning and the end of increases in heart rate (so-called cardiac activations) that are likely to be associated with cortical arousals including EEG awakenings. Repeated noise induced autonomic activations may play a key role in the genesis of hypertension and associated cardiovascular diseases. However, as of today there is no generally accepted convention on what exactly constitutes a cardiac arousal, e.g., how strong a heart rate increase must be in order to be classified as a relevant cardiac activation that may be associated with short-term or long-term clinical consequences. Recent findings of a carefully designed experiment by Guilleminault et al. (2006) support the thesis that EEG arousals are a prerequisite for the detrimental effects of sleep fragmentation on daytime functioning. Therefore, it seemed reasonable to determine the relevance of cardiac activations depending on whether they are accompanied by cortical arousals or not. In this study, polysomnographical EEG awakenings and automatically detected cardiac activations were systematically compared based on 985 laboratory nights of 112 subjects.

METHODS

Subjects and Protocol

We investigated 128 subjects in a laboratory study on the effects of aircraft noise on sleep that was conducted between 1999 and 2004 at the Institute of Aerospace Medicine at the German Aerospace Center (DLR) and is described in detail elsewhere (Basner & Samel 2005). In each of 16 study phases, 8 subjects were investigated simultaneously with polysomnography. They slept in separate bedrooms in the underground sleep facility of the DLR-Institute of Aerospace Medicine for 13 consecutive nights. Here, ANEs with maximum SPLs of 45, 50, 55, 60, 65, 70, 75, or 80 dB(A) were played back via loudspeakers during nights 3-11. In one of the 11 nights, always the same ANE was repeatedly played back 4, 8, 16, 32, 64, or 128 times per night. Night 1 served as adaptation, night 2 as baseline, and nights 12 and

13 as recovery. Lights were turned off at 11 pm and on again at 7 am. 16 subjects served as a control group. They were not exposed to aircraft noise and excluded from this analysis. The experimental group consisted of 112 subjects (47 male, mean ± SD age 37.9 ± 13 years).

ECG-analysis

The ECG was derived from the chest wall (derivation Einthoven II) and sampled at 1000 Hz. R-waves were automatically detected with a software developed in a LabVIEW^{TM} environment (Samel et al. 1997). The time of each heartbeat was stored in a separate line of an ASCII file together with the heart rate (beats per minute, bpm) derived from the interval to the preceding beat. An ECG-based algorithm was used to automatically detect cardiac activations associated with cortical arousal as defined by the American Sleep Disorders Association in 1992 (Bonnet et al. 1992). The algorithm itself is extensively described elsewhere (Basner et al. 2007a). Briefly, a median heart rate is calculated for a ± 90 s interval relative to the momentary heart beat. In a Bayesian approach and comparable to five sequential diagnostic tests, differences of five consecutive heartbeats to the median heart rate are used to determine cardiac activations, i.e. whether the sequence of heart rate differences is associated with a cortical arousal. The ECG-algorithm software stored start and end times of cardiac activations in separate files for each night.

Event-related analysis

An event-related analysis establishes a direct temporal association between the occurrence of an ANE and the reaction of the investigated subject (Basner et al. 2006; Ollerhead et al. 1992; Passchier-Vermeer et al. 2002). This analysis was only possible because during the study electrophysiological signals were synchronously sampled with a trigger signal indicating noise on- and offset. Probability of reactions additionally caused by aircraft noise ($P_{ADDITIONAL}$) and of aircraft noise induced reactions ($P_{INDUCED}$) were calculated according to Brink et al. (2006). We set the screening interval for both EEG awakenings and cardiac activations to 60 s (or 2 polysomnographical epochs), as this maximized both $P_{ADDITIONAL}$ and $P_{INDUCED}$ for EEG awakenings.

EEG awakenings were defined as sleep stage changes from any other sleep stage to stage *Wake*. An epoch was defined as the first epoch under the influence of noise if the ANE started within ± 15 s relative to the start of the epoch. By design, the ANEs started exactly at the beginning of the epoch in 85 % of all noise events. The first noise epoch and the epoch following it were screened for an EEG awakening. The same 60 s interval, but now relative to the onset of the ANE, was screened for the onset of a cardiac activation as defined by Basner et al. (2007a). An ANE was excluded from the analysis if the subject was already awake in the epoch preceding the first noise epoch. Therefore, noise events outside of SPT, i.e. before sleep onset or after final awakening, were also excluded from the analysis. Finally, the ANE was only included if the 10-s interval preceding noise onset was free of cardiac activations. Spontaneous reaction probability was determined in noise-free baseline nights. Here, elapsed time after sleep onset was determined for each noise event onset, and the respective interval in the baseline night of the same subject was screened for spontaneous reactions with the above mentioned methodology.

It was one objective of the study to demonstrate that a valid cardiac activation analysis is possible without or with minimal human supervision. Therefore, ECG raw data were deliberately not visually inspected prior to data analysis. Rather, an automatic

algorithm was used to investigate heart beats within a -100 s to +150 s interval relative to noise onset. Heart beats with heart rates below 28 or above 105 were considered invalid. A prior analysis of the data had shown that less than 0.2 % of heartbeats were below 28 bpm or above 105 bpm. The ANE was excluded form the analysis if more than 10 % of heartbeats in the interval were considered invalid. The ANE was also excluded if a single heart beat was lower than 10 bpm, indicating an ECG signal loss of 6 s or longer, or if there was no ECG signal at all.

RESULTS

From 30,580 ANEs originally planned for playback, 23,855 ANEs (78.0 %) were used for the final analysis. 5,054 ANEs (16.5 %) were excluded because the ANEs occurred outside of SPT, because the subject was awake prior to noise onset, or because the 10 s interval prior to noise onset overlapped with a cardiac activation. Only 1,671 ANEs (5.5 %) were excluded due to subject withdrawal or equipment failure. Spontaneous reaction probability estimations were based on 23,937 *virtual*-ANEs.

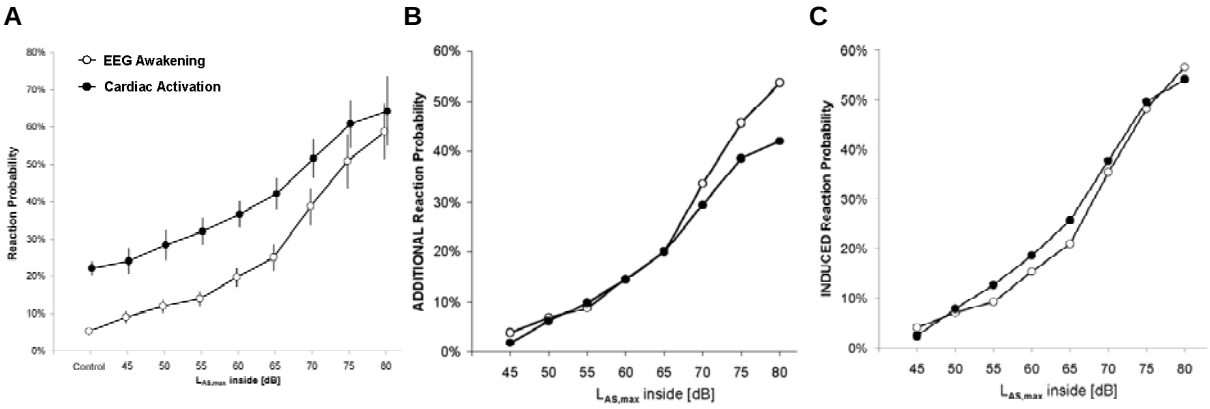

Figure 1: Exposure-response relationships (ERLs). **A** ERLs for EEG awakenings and cardiac activations depending on maximum sound pressure level $L_{AS,max}$ inside the bedroom. The spontaneous reaction probability (labelled "Control") was determined in noise-free baseline nights. Point estimates and 95 % confidence intervals are shown. **B** ERLs for reactions additional to spontaneous reactions are shown. They were calculated according to equation (1). **C** ERLs for noise induced reactions are shown. They were calculated according to equation (2).

The probability of EEG awakenings and cardiac activations depending on maximum SPL (measured at the sleeper's ear) as well as spontaneous reaction probabilities are shown in Figure 1 A. Both EEG awakenings and cardiac activations increased monotonously with increasing SPLs, but absolute cardiac activation probability was higher for all maximum SPLs compared to EEG awakenings. The difference in probabilities (cardiac activations minus EEG awakenings) decreased with increasing SPLs. Based on the 60 s screening interval spontaneous reaction probabilities were about fourfold higher for cardiac activations (22.2 %) compared to EEG awakenings (5.2 %).

The results of an agreement analysis between EEG awakenings and cardiac activations across all subjects and nights and based on single ANEs showed that the overall agreement was higher in control (81.9 %) compared to noise nights (76.4 %). However, if corrected for chance expected agreement according to Landis and Koch (1977), agreement was higher in noise (κ=0.60) compared to control nights (κ=0.33), representing "moderate to substantial" and "fair" agreement, respectively.

Reaction probabilities $P_{ADDITIONAL}$ and $P_{INDUCED}$ depending on maximum SPL are shown for EEG awakenings and cardiac activations in Figure 1 B and C. For both $P_{ADDITIONAL}$ and $P_{INDUCED}$, the observed exposure-response curves for EEG awaken-

ings and cardiac activations almost ran on top of each other. $P_{ADDITIONAL}$ tended to be lower for cardiac activations compared to EEG awakenings for maximum SPLs ≥ 70 dB(A) (Figure 1 B), whereas $P_{INDUCED}$ for cardiac activations marginally surpassed $P_{INDUCED}$ for EEG awakenings for all SPLs except for 45 dB(A) and 80 dB(A) ANEs (Figure 1 C).

DISCUSSION

In this study, polysomnographically determined EEG awakenings and automatically detected cardiac activations were systematically compared in noise-free control nights and nights with exposure to aircraft noise using event-related analysis.

It was shown that both EEG awakenings and cardiac activations increased monotonously with increasing maximum SPLs, but probability of cardiac activations was higher compared to EEG awakenings in all exposure conditions and the control nights. The higher prevalence of cardiac activations was expected, as the ECG algorithm was designed to detect cardiac activations associated with cortical arousals (Basner et al. 2007a). The latter are defined as activations of the central nervous system lasting 3 s or longer (Bonnet et al. 1992; Iber et al. 2007), and they are therefore more frequent and less specific than EEG awakenings. Bonnet and Arand (2007) observed 10.6-21.9 spontaneous EEG arousals per h total sleep time (TST) in healthy subjects, compared to 2.9-7.3 awakenings per h TST (both frequencies increased with age).

The difference in reaction probabilities (cardiac activations minus EEG awakenings) decreased with increasing SPLs, i.e. - compared to EEG awakenings – the ECG algorithm was more sensitive to detect noise induced alterations at low noise levels, corroborating recent findings of Basner et al. (2007b). Low SPLs seem to be loud enough to increase the number of EEG arousals (and associated cardiac activations), but most of these arousals also seem to be too brief to be scored as an EEG awakening as well. The higher sensitivity of the ECG algorithm may therefore be advantageous in situations with low exposure levels or chronic noise exposure with habituation. At noise levels above 65 dB(A), and especially at 80 dB(A), ECG and EEG exposure-response curves converged. Here, most of the CNS activations seemed to be long enough to be scored both as cardiac activations and EEG awakenings. The small increase in the probability of cardiac activations at 80 dB(A) compared to 75 dB(A) suggests a ceiling effect, but exposure nights with noise levels greater than 80 dB(A) would be needed to confirm this impression.

If spontaneous reaction probability is taken into account according to Brink et al. (2006), exposure-response curves were practically identical for EEG awakenings and cardiac activations (Figure 1 B and C). If (additionally) induced EEG awakenings are considered the primary outcome of a noise effects study, it may therefore be possible to replace polysomnography with a single channel ECG in order to achieve reliable estimates of the frequency of noise induced EEG awakenings by automatically detected cardiac activations.

CONCLUSION

In conclusion, using data of 112 subjects and 23,855 ANEs with maximum SPLs between 45 and 80 dB(A), this investigation showed that the probability of automatically detected cardiac activations increased monotonously with increasing SPLs of ANEs. Furthermore, exposure-response curves for reactions (additionally) induced by aircraft noise were practically identical for EEG awakenings and cardiac activations.

The latter could therefore be used as estimates for EEG awakenings. Replacing polysomnography with a single channel ECG would be advantageous in several ways. In contrast to the EEG, ECG electrodes could be attached unsupervised and without problems by the investigated subjects themselves. This would facilitate large scale field studies with low methodological expense but high validity of results, as long as (additionally) induced EEG awakenings are the primary endpoint of the investigation. The method could be combined with other low maintenance methods like actigraphy in order to further increase the validity and scope of the results. Furthermore, the ECG is analyzed automatically and objectively by the ECG algorithm and therefore more reliable, less time consuming and cheaper than polysomnography. However, using a single channel ECG necessarily leads to data reduction and polysomnography therefore remains the gold standard for investigating noise effects on sleep. More analyses and studies are needed to further validate the ECG algorithm in the field and to investigate inter-individual differences in its ability to predict EEG awakenings.

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