Cardiovascular and Autonomic Response to Environmental Noise During Sleep in Night Shift Workers

Norman Carter PhD,[1] Robyn Henderson MSc,[2] Saroj Lal MSc,[3] Michael Hart MApplSc,[4] Sharon Booth BA, PGDipPsy,[5] and Stephen Hunyor MD[6]

[1]University of Sydney; [2]None—Formerly Royal North Shore Hospital, Sydney; [3]University of Technology, Sydney; [4]Consultant—Formerly Royal North Shore Hospital, Sydney; [5]Royal Prince Alfred Hospital, Sydney; [6]Royal North Shore Hospital, Sydney

Summary: Polysomnograph, beat-by-beat heart rate and blood pressure were monitored in night shift workers exposed to environmental and laboratory noise events during day sleep.

The study was carried out in a sleep laboratory.

Subjects were nine young, healthy female night shift workers.

Recorded noises from trucks, civilian aircraft, low altitude military aircraft and tones were presented at 55, 65, and 75 L_{Amax}.

Sleep stage, heart rate, and systolic and diastolic blood pressures before and immediately after onset of noise events were compared. Spectral analyses of heart rate and blood pressure variabilities were used to compare sympathetic and parasympathetic nervous tone in 10-min. intervals containing noise and quiet.

Heart rate was responsive to noise level but not noise type. Blood pressure increased primarily to sounds of sudden onset. Noise-induced awakening and alpha EEG responses were related to BP increase. Increase in HR was greatest when subjects were awakened by noise or already awake. Spectral analysis of BP variabilities indicated increased sympathetic vascular tone due to noise. Similar analyses of HR data indicated no noise effect. No habituation to noise was apparent over three consecutive sleep sessions.

It was concluded that over the range of noise levels used, heart rate responds to noise level during sleep; blood pressure to sounds of sudden onset. Spectral analysis of blood pressure variabilities is a sensitive measure of autonomic nervous response to environmental noise and should also be studied in subjects sleeping at home.

Key words: Sleep; shift work; environmental noise; autonomic nervous system; cardiovascular system

INTRODUCTION

SLEEP IS KNOWN TO BE A STATE OF REDUCED SYMPATHETIC AUTONOMIC TONE ACCOMPANIED BY DECREASED HEART RATE AND BLOOD PRESSURE.[1]. Therefore, sleep could exert a cardio-protective effect by re-setting baroreceptor sensitivity and providing respite for the cardiovascular system, while chronic sleep disturbance due to noise could have the opposite effect.[2] Persistent blood pressure (BP) rises of as little as 5 mm Hg have implications for individuals' long-term cardiovascular health.[3]

Laboratory and field studies have shown increases in minute-by-minute average heart rate (HR), and finger pulse amplitude responses to aircraft flyover and motor vehicle passbys during sleep. These responses tend not to habituate.[4,5]

Elevated BP regularly accompanies episodes of sleep apnea. Diurnal BP is also increased, but is reversed by successful treatments of sleep apnea.[6,7] To find out whether the increase in BP was due to repeated arousals or to other factors, arousals and BP responses were studied using auditory stimuli and normal subjects sleeping in a laboratory. Systolic blood pressure (SBP) increase occurred even when there were no concurrent electroencephalographic (EEG) changes.[8,9,10] These experimental results suggest that chronic exposure to environmental noise during sleep could contribute to permanent increases in BP in otherwise healthy individuals.

It has also been found that increases in muscle sympathetic nervous activity followed presentations of "door knocking" sounds and 125 ms, 800 Hz tones during light sleep.[10,11,12] However, the use of artificial sounds with instantaneous rise times in studies of beat-by-beat cardiovascular responses and neurographic studies does not justify generalization to familiar environmental sounds with relatively gradual onset, such as civilian aircraft flyovers and road traffic. Also, other data[13] indicate that autonomic nervous system activity has regional differences, so that neurographic measures of peripheral sympathetic nervous activity may not be representative of such activity in the heart.

Frequency analyses of HR and BP variabilities have shown that power spectral densities of certain spectral components reflect modulation of cardiovascular systems by sympathetic and parasympathetic nervous tone,[14,15,16,17] and have confirmed that sleep is a period of reduced sympathetic, and enhanced vagal, tone with variations across sleep stages.[1,18,19] This suggests that the method may be useful for non-invasively studying the effects of environmental noise on autonomic balance during sleep.

Aims and Research Plan

The physiological effects of noise on night shift workers is of particular interest because of their requirement to sleep during the day, when environmental noise is greatest. The present study aimed to extend previous research in this area by comparing the effects of four types of noise events (civilian aircraft landing, truck passbys, tones, and low-flying military aircraft) during

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Address correspondence to: Dr. Norman Carter, 25 Calga Street, East Roseville 2069, Australia; Tel: 62 02 9417 4709; Fax: 62 02 9417 4772; E-mail: nlcarter@dot.net.au

sleep on beat-by-beat BP and HR, and sympathetic and parasympathetic nervous system tone in night shift workers.

METHODS

Noise Recording, Replay, and Monitoring

Military aircraft overflight. Noise from a low-flying military aircraft was provided by a DAT tape recording of noise on the ground generated by high velocity, low-level Tornado aircraft passing overhead. Recordings were replayed on an AIWA HHB 1 PRO recorder into Marconi Stepped Attenuators Type TF262, Technics Type SU-7300 power amplifier, and two Digitor 40W loudspeakers (Dick Smith, Sydney), placed on the ceiling in diagonally opposite corners of the sleep room.

Trucks. Digital stereophonic recordings of truck passbys were made at night to ensure low background noise levels. Four recordings were made of trucks travelling downhill, uphill, braking, and driving at steady speed on level terrain. Instrumentation included two Bruel and Kjaer (B&K) 1/2" microphones Type 4155 on B&K Type 2231 sound level meters, calibrated with a B&K Type 4220 pistonphone. Both channels were fed into an AIWA HHB 1 PRO digital recorder.

The truck noises were subsequently re-recorded from the AIWA DAT tape to audiovisual tape and replayed, using a National Panasonic Type AG 6800 video cassette recorder, through Marconi Stepped Attenuators Type TF262 to a mains powered mixer (Type A-8400), power amplifier (Technics Type SU-7300), and two Linz Type 8066A loudspeakers in the sleep room. The loudspeakers were placed on the floor with centers two meters apart, and two meters from the near side of the bed, equidistant from the subjects' head (pillow) position.

Civilian aircraft landings. Four noises of Boeing 747 aircraft landing at Sydney (Kingsford Smith) Airport were recorded under the flight paths less than one kilometer from runway threshold. The recordings were made using Bruel and Kjaer Type 4155 1/2" microphones, B & K sound level meters Type 2231, and AIWA "HHB 1 PRO" digital tape recorders. These recordings were subsequently transferred to cassette tape, and replayed from a TEAC TR A-360 cassette recorder into the sleep room. The two channels were replayed through separate Marconi Stepped Attenuators Type TF262, and the Technics Type SU-7300 power amplifier to two Digitor 40W speakers (Dick Smith, Sydney) on the ceiling in diagonally opposite corners of the sleep room.

5.0s 1000 Hz tones. The tones were generated by a NAL oscillator and recorded with a TEAC TR A-360 cassette recorder and replayed using the same attenuators, amplifier, and Linz Type 8066A loudspeakers as for truck noises.

Noise Monitoring

All noises presented to subjects were monitored. A Bruel and Kjaer (B & K) Type 4155 1/2" microphone, connected to a B & K Type 2639 pre-amplifier and battery-powered "KT" microphone power supply, was placed in the sleep room 60 cm above the subject's head position. The output of this microphone was fed via a sound attenuating duct to the control room and to two B & K Type 2120 frequency analyzers. The AC output of one frequency analyzer was input to a calibrated B & K level recorder (Type 2307). The paper display of the level recorder enabled the experimenter to visually monitor noise levels at the subject's head position during the experiment, and provided a "hard copy" of the time histories and A-weighted noise levels generated during each session. The DC output of the other frequency analyzer was fed to an A/D board mounted in a IBM PC compatible 486 computer, where it was digitized and stored simultaneously with the sleep polygraph. This system enabled the experimenters to record precisely the time of day of the noise peaks, as well as their time relation to the sleep polygraph and finally to subjects' beat-by-beat BP and HR.

The noise monitoring system was calibrated by placing a B & K Type 4220 pistonphone on the microphone, and adjusting the frequency analyzers to register 124 dB (linear scale).

Physiological Monitoring

Sleep monitoring and recording included eye movement (electroculogram or EOG, 10/20 system electrode placements ROC/A1, LOC/A2), submental muscular tension (electromyogram or EMG), and vertex EEG (electrodes at C3/A2). Signals from the electrodes were amplified by Neomedix Type NT810 AC/DC "head stage" amplifiers. These amplifiers incorporated RF transformers to achieve Class A patient isolation. Outputs were fed (simultaneously with the A-weighted noise level) to a Burr Brown A/D board, mounted in an Elite 486 computer controlled by specialised Stellate Systems "Eclipse" Version 1.2 sleep monitoring software.

Sleep polygraph and noise trace were downloaded at the end of each session from the computer's hard disk to a Verbatim VBR5B2 5.25" optical disk cartridge using a 600 megabyte magneto-optical disc drive (Micro Design International: Laserbank 600MX Multifunction).

Beat-by-beat BP was recorded using continuous volume clamp plethysmography of the finger in a temperature-controlled environment using a "Digipress" prototype non-invasive, continuous finger BP monitoring (CNIBP) device.[20] This computer-driven device had previously been shown to accurately track arterial BP during physiological and pharmacological interventions designed to markedly alter autonomic discharge.[21,22] Beat-by-beat BP and electrocardiograph (ECG) were digitized online at 1000 Hz using a 486 IBM compatible PC and data acquisition package (CVMS Data Acquisition Package, McPherson Scientific, Australia) incorporating a 12-bit analog to digital converter (CIO-AD-Jr, Computer Boards Inc., USA). The results of continuous monitoring of these parameters were time-coded and stored on computer for later analysis using specialized software (CVMS Data Acquisition Package, MacPherson Scientific, Australia).

Subjects, Test Procedure, and Noise Schedules

Subjects

Volunteers were selected by sphygmomanometer BP testing, a health questionnaire, and three self-evaluation questionnaires.

The psychological tests used were the Spielberger State-Trait Anxiety Inventory Forms Y-1 and Y-2;[23] the Beck Depression Inventory;[24] and the Profile of Mood States or POMS questionniare.[25]

Nine healthy female nurses on permanent night duty at Royal North Shore Hospital, Sydney were selected. They worked a regular pattern of shifts which began between 21:30 and 23:00, and finished at 7:30, predominantly in four-day spells with days off in between. All were in good general health, without sleep disorders or medications deemed to affect sleep or the cardiovascular system. Use of alcohol was minimal or absent. They were normotensive and free of illnesses constituting risk factors for high BP or heart disease. All reported satisfactory sleep patterns, with typically six to eight hours sleep daily, ranging from five to ten hours. Their psychological test scores were within normal limits. All had normal hearing. Subjects' ages ranged from 20.1 years to 33.7 years (mean 27.5 years). They had carried out shift work for an average of seven years (range 1—14 years). Resting (office) BPs ranged from 100—117 mm Hg (systolic) (average 110) and 60—82 mm Hg (diastolic) (average 71.4).

All subjects gave informed consent to participate in the study. The research protocol was approved by the Royal North Shore Hospital Medical Research Ethics Committee in accordance with the Australian National Health and Medical Research Council's requirements for experimentation with human subjects.

Test Procedure

Each subject attended a total of six sessions. Sessions 1 and 2 were devoted to screening, assessment and training. In Session 1 each subject was given a sleep diary in which to record their sleep schedules in a week of night work. This diary was used to arrange for the subject to sleep in the laboratory at her preferred sleep times on consecutive days (Sessions 3—6). Session 3 was a familiarisation session. Sessions 4—6 were experimental sessions. Subjects' habitual caffeine consumption ranged from 0—4 cups of coffee or tea in any 24-hour period. They were asked to keep to this during the experimental period to avoid "withdrawal" symptoms, and not to nap outside the laboratory during the study.

In Sessions 4—6 a 20-minute period of silence (to allow the subject to go to sleep) was followed by an 80-minute period divided into 10-minute intervals with 10-minute alternating noise and quiet intervals. Where sleep was disturbed additional quiet intervals were inserted. In each noise interval recordings of three truck passbys, civilian aircraft landings, military aircraft flyovers, or tones were presented in a randomized order. Each noise type was presented three times within its 10-minute Noise interval, at levels 55, 65, and 75 dB (L_{Amax}), again in a randomized predetermined schedule. Subjects completed their sleep in the same laboratory. Mean lights out time was 10:10 (range 9:07-11:10); mean lights on (end of EEG sleep monitoring) was 16:14 (range 14:11-18:20).

Noise Events

Presentation of each noise event was initiated by the Experimenter, and "tagged" by the computer. Elapsed time from commencement of recording at which each noise peak occurred (recorded in the noise channel of the polygraph), as well as the corresponding sleep epoch number, were also noted by the sleep stager. The schedule of noise types and levels identified the type of each noise event. Verification of the sound level (L_{Amax}) of each noise event in the sleep room was obtained from the microphone in the sleep room and the calibrated paper trace produced by the graphic level recorder.

The four separate truck, and civilian aircraft noises were presented in random sequence. Onset and offset times were defined as the points at which the noise level crossed the 30 dBA level. The duration ("noise period") of each noise was the time between onset and offset points, and varied with the recording used and its L_{Amax} level. For trucks the average times from noise onset to noise peak, and from peak to noise offset were 10s and 9.75s for 55 L_{Amax}; 18.75s and 20.25s for 65 L_{Amax}; and 19.75s and 26.75s for 75 L_{Amax}. For civilian aircraft these values were 12.25s and 7.75s; 14.25s and 17.5s; 15.75s and 24.75s respectively. For military aircraft they were 1.0s and 1.0s; 1.0s and 2.0s; 1.0s and 6.0s. Onset and offset of the 5.0s, 1000 Hz tones were instantaneous, filtered to remove onset and offset transients.

Beat-By-Beat BP and HR Recording

Beat-by-beat HR and BP recording encompassed the pre-noise 20-minute interval, and the immediately following 80-minute period of four 10-minute noise, and four 10-minute quiet intervals.

Sleep Polygraph Data

Sleep polygraphs were sleep-staged in 20-second epochs by an experienced sleep technologist, using standard criteria.[26]

The sleep technologist also noted the following:

* Noise type and level;
* Sleep stage of the epoch prior to that containing noise onset;
* Presence/absence of an awakening response to the noise event in the epoch containing the noise or the two subsequent epochs;
* Sleep stage change and the direction of the change ("up"/"down") in the "noise" epoch or the two subsequent epochs;
* Presence/absence of an alpha response (in 'noise' epoch or the two subsequent epochs).

Alpha responses to noise were episodes of alpha frequency in the vertex EEG of three seconds duration or longer, commencing at or after noise onset, without associated movement artifact, and with total alpha less than 50% of the epoch.

The "depth" of sleep was rated as increasing according to the following stages: Wake (W), Rapid Eye Movement (REM), stage 1, stage 2, 3 and 4. It is acknowledged that REM occupies an ambiguous position on this "scale" but is placed next to Wake because of the relatively high autonomic activity previously recorded during REM.[18]

RESULTS

A. Blood Pressure and Heart Rate Responses to Single Noise Events

Means and standard deviations of beat-by-beat SBP, DBP (mm Hg), and interbeat interval (IBI, msec) were calculated for the following beats:

* Ten beats immediately preceding onset of each noise event (designated "Pre-noise" beats);

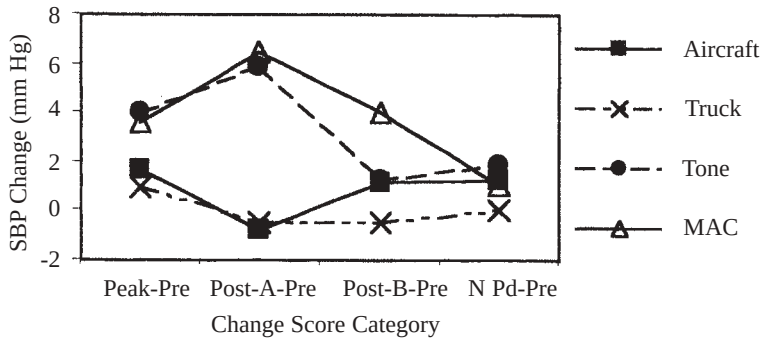

Figure 1—Mean systolic blood pressure changes (SBP, mm Hg) in response to four noise types, by change score category.

* Ten beats including and immediately after the peak level of the noise event ("Peak-noise" beats);
* Ten beats immediately following noise offset (designated "Post Noise A");
* Ten beats following Post Noise A ("Post Noise B").

Four SBP "change" scores were calculated by subtracting mean Pre-noise SBP from the corresponding Peak-noise SBP (Pk-Pre), Post A SBP (PostA-Pre) and Post B SBP means (PostB-Pre), and SBP means for the "Noise Period" (NoisePd-Pre) (onset to offset of the noise) for each noise presentation. Similar calculations were carried out to track DBP and IBI responses to the noise events.

The statistical analyses employed SPSS Version 10. Repeated measures three-way anovas were used to fit a full factorial model, involving the design variables (noise type, noise level, and sessions) to the data of each of the outcome variables. Where three-way interactions were non-significant the analysis was repeated for main effects and two-way interactions, and where these interactions were also non-significant models including only main effects were applied. In addition, general linear models were used to assess sleep stage at noise onset, sleep stage change, alpha response, and awakening as statistically significant independent predictors of the outcome variables.

The least significant difference method was used for post-hoc multiple pairwise comparisons between levels within statistically significant factors.

Systolic Blood Pressure (SBP)

Peak minus pre-noise Change (Pk-Pre). No significant three-way or two-way interaction was found between the main effects noise type, level and session. Analysis for main effects revealed a significant effect of noise type (df=3, F=2.807, $p<0.05$). Post hoc pairwise comparisons yielded significant differences between tone, and aircraft and truck noise ($p<0.05$ and $p<0.01$); and MAC and truck noise ($p<0.05$).

Effects of wakening (df=1, F=9.783, p=0.002) and alpha response (df=1, F=10.493, p=0.001) were significant and independent of each other and noise type. Mean SBP increase associated with wakening and alpha response were 2.6 and 2.4 mm Hg respectively.

PostA-Pre. The PostA-Pre data showed no significant three-way or two-way interactions. Of the main effects only noise type was significant (df=3, F=14.175, $p<0.001$). In post hoc pairwise comparisons, tone and MAC were significantly different from truck and civilian aircraft noise ($p<0.001$). Aircraft and truck were not significantly different.

Awakening and alpha response approached but did not reach significance.

PostB-Pre. These data showed no significant three-way or two-way interactions. Noise type was significant (df=3, F=4.041, p=0.008). Pairwise comparisons, showing responses to MAC noise, were significantly greater than to truck (p=0.001) and aircraft (p=0.017).

None of the sleep variables were significant.

NoisePd-Pre. There were no significant three-way or two-way interactions or main effects. Wakening and alpha responses to noise were significantly associated with greater NoisePd-Pre SBP change (df=1, f=9.649, p=0.002; and df=1, F=4.921, p=0.027 respectively). The mean values of these effects were 1.0 (wakening) and 2.0 (alpha) mm Hg.

Mean SBP change scores, plotted for each change category and noise type in Figure 1, indicate that sounds of sudden onset (tones and Tornado aircraft overflights) induced greater change than truck noise and noise of Boeing 747 landings. The effect persisted into the PostB period but was not apparent in data averaged over the time from onset to offset of the noise.

Diastolic Blood Pressure (DBP)

Peak minus pre-noise Change (Pk-Pre). The interaction between noise type and session was significant (df=6, F=2.229, p=0.041). A plot of estimated marginal means by noise type and session suggested that Sessions 1 and 2 results were very similar, and different from Session 3. The type by session interaction for Sessions 1 and 2 data only was non-significant. A main effects anova on noise type and session showed a significant effect of noise type (df=3, F=11.813, $p<0.001$). The trend of the results over Sessions 1—3 was for the response to increase, the opposite to that expected from habituation.

Pairwise comparisons on the noise type factor for Sessions 1 and 2 confirmed that responses to MAC and tone were significantly greater than aircraft and truck noise (all $p<0.001$). MAC and tone were not significantly different.

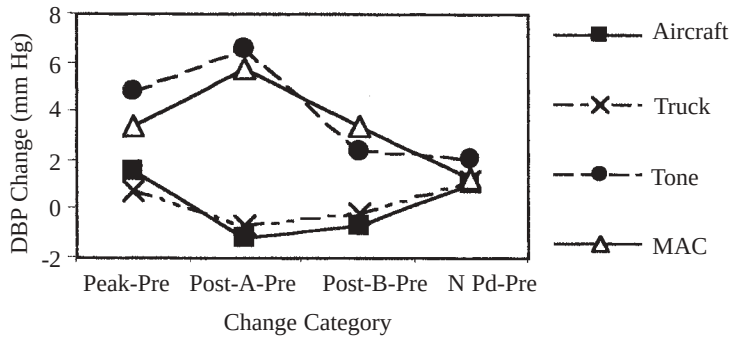

Figure 2—Mean diastolic blood pressure changes (DBP, mm Hg) in response to four noise types, by change score category.

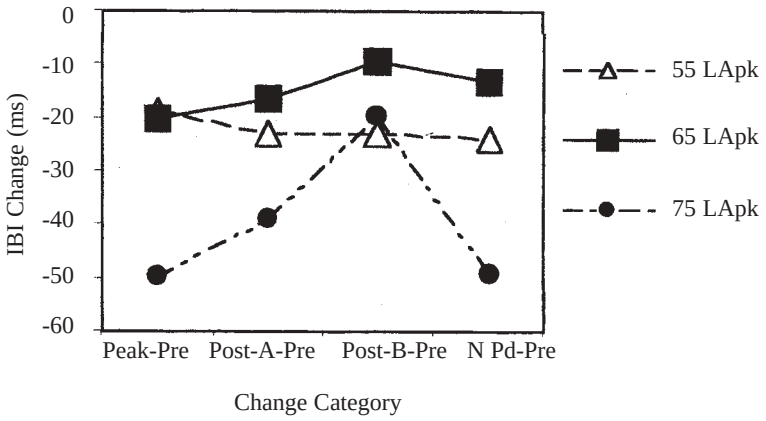

Figure 3—Mean interbeat interval changes (IBI, ms) in response to four noise types, by change score category.

Alpha response in Sessions 1 and 2 data was related to DBP change (p=0.002). On average DBP increased 2.7 mm Hg greater with concurrent alpha response.

There were no significant differences in Session 3 data considered alone.

PostA-Pre. The three-way and all two-way interactions were non-significant. Noise type (df=3, F=12.876, p<0.001) and noise level (df=2 F=3.049, p=0.049) were significant.

Pairwise comparisons on noise type showed that DBP responses to MAC were significantly different (greater) than to aircraft and truck noise (p<0.001); as were responses to tone (p<0.001). Truck and aircraft were not significantly different. On the level factor, DBP increases to 75 dBA noise were significantly different (greater) than those to 55 dBA (p=0.020).

Alpha response was related to PostA-Pre DBP change (df=1, F=7.14, p=0.008). The effects of alpha were greatest in response to tone and MAC (6.3 and 2.4 mm Hg) and were similar at all noise levels.

PostB-Pre. The three-way interaction between main effects was non-significant. Of the two-way interactions type by session was significant (df=6, F=3.830, p=0.001). From consideration of the estimated marginal means Sessions 1 and 2 were identified as comparable and different from Session 3. This was confirmed by anova of Sessions 1 and 2 data, in which noise type, session, and the type by session interaction were all non-significant. Post hoc multiple pairwise comparisons on the noise type factor were also non-significant.

Sleep variables were not significant.

In the Session 3 data noise type was significant (df=3, F=3.758, p=0.015). Pairwise comparisons indicated significantly greater response to MAC than aircraft and truck noises (p=0.012 and p=0.034 respectively). DBP change was greater in Session 3 than in Sessions 1 and 2, opposite to the trend which might be expected from habituation.

NoisePd-Pre. The noise level by session interaction was significant (df=4, F=2.753, p=0.029). Plots of the estimated marginal means (session by level) failed to clarify any trends. Anovas of Sessions 1 and 2 resulted in no significant main effects nor interaction. Multiple pairwise comparisons on the level variable did not produce any significant pairs. No sleep variable was significantly related to NoisePd-Pre DBP change.

Analysis of the Session 3 data showed no significant noise type or level effects. None of the pairwise comparisons on the noise level variable nor the sleep variables were significant.

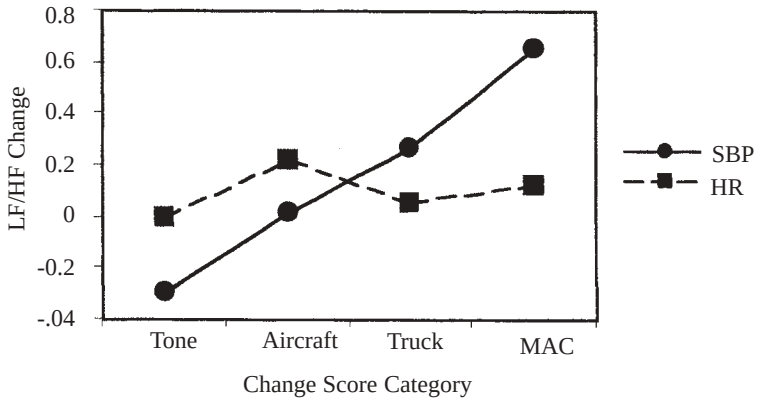

Figure 4—Mean changes in ratios of low frequency (LF) to high frequency (HF) spectral densities in response to four noise types. Systolic blood pressure (SBP) and heart rate (HR) variabilities were derived from 10-minute intervals (see text).

Although there was an effect of noise level in the PostA-Pre DBP data the significant differences were predominantly associated with noise type. The means across noise levels for noise type are plotted for each change score category in Figure 2 and suggest that the effect of noise on DBP was confined to the sudden-onset sounds. Maximum effect occurs in the Peak-Pre and PostA-Pre categories, which in the case of these relatively brief sounds is close in time to the noise onset and peak. The magnitudes and time courses of the SBP (Figure 1) and DBP responses (Figure 2) were similar.

Heart Rate (Interbeat Interval, IBI)

Peak minus pre-noise change (Pk-Pre). Three-way and two-way interactions were not significant. Noise level was the only significant main effect (df=2, F=3.503, p=0.032). Pairwise comparisons indicated that IBI decrease associated with 75 dBA noise was significantly greater than response to noise at 65 dBA (p=0.045) and 55 dBA (p=0.014). Sleep variables were not significant.

PostA-Pre. Interactions and main effects were not significant. Sleep stage at noise onset (df=2, F=9.439, p<0.001) and wakening (df=1, F=10.440, p=0.001) were significant. The effect of noise on IBI appeared greatest when subjects were awake at noise onset (-66 msec). On average wakening decreased mean IBI by 33 msec.

PostB-Pre. Interactions and main effects were non-significant. Sleep stage at noise onset (df=2, F=6.363, p=0.002) and sleep stage change (df=2, F=5.593, p=0.004) were related to PostB-Pre. IBI responses when in NREM and REM at noise onset were significantly different from Wake (p=0.001; p=0.004), IBI being most changed when subjects were awake. Change in IBI associated with stage change "up" (-78 msec) was significantly different (p=0.002) from stage change "down" (+32 msec).

NoisePd-Pre. There were no significant interactions or main effects in this change category. The sleep variables were also unrelated to IBI change.

In the above IBI analyses noise level was the only significant main effect, evident in the Peak-Pre data. Figure 3 plots the means for each noise level in each change category.

Inspection of Figure 3 confirms that the level effect is due to responses (decreased IBI) to the highest noise level (75 LA_{max}), suggesting that for HR there is a threshold noise level below which no effect occurs, in contrast with SBP and (less clearly) DBP. The time course of HR means for response categories also suggests a short latency HR increase which is dissipated soon after the noise peak is reached.

RESULTS B.

Assessment of Autonomic Activity in 10-minute Noise and Quiet Intervals by Fourier Analysis of Beat-by-Beat Systolic BP and HR Variabilities

At least five minutes of stationary data are required to ensure reliability and repeatability of results when Fourier analysis is used to compute power spectral densities in the 0.02-1.0 Hz band.[15,27] In the present study 10 minute data segments were used, following Zwiener.[27] These signals were digitized online at 1000 Hz and the direct Fast Fourier Transform spectral analyses carried out using a 486 IBM compatible PC and CVMS (McPherson Scientific, Australia) data acquisition package.

Power spectral densities (PSDs) were calculated in the bands 0.07-0.14 Hz (LF) and 0.15-0.35 Hz (HF) for each 10-minute noise and quiet interval in each session. Outcome variables were changes in the LF/HF ratios (LF/HF ratios for noise intervals minus LF/HF ratios for paired baseline, or quiet intervals) for each noise type and session.

Statistical methods were similar to those used for Results A.

Systolic Blood Pressure

The mean SBP LF/HF change values for each noise type are plotted in Figure 4.

A two-factor (noise type, session) repeated measures anova was carried out. The two-way interaction was non-significant. Analysis for main effects found that noise type was significant (df=3, F=5.378, p=0.002). Multiple pairwise comparisons on

noise type indicated that LF/HF increase was significantly greater in response to MAC than to civilian aircraft (p=0.035) and tone (p=0.002). Truck noise was significantly different from tone (p=0.008).

Heart Rate (HR)

The mean HR LF/HF change values for each noise type are also plotted in Figure 4.

Similar analyses were carried out for differences between HR LF/HF spectral ratios for noise and paired quiet intervals as for SBP. No significant differences were found for main effects (session and noise type). Pairwise comparisons on noise type were also non-significant.

DISCUSSION

The present study has shown that beat-by-beat BP and HR increase (IBI decreases) in response to certain noise events during sleep (Figs. 1-3). Autonomic change (a relative increase in sympathetic nervous tone as measured by LF/HF ratios derived from fast Fourier spectral analyses of beat-by-beat SBP variabilities) occurred in response to presentation of three recordings of a military aircraft flyover (at 55, 65 and 75 L_{Amax}) during 10-minute intervals. The fact that the LF/HF ratio responded to this rather mild noise schedule indicates that spectral analysis of SBP variabilities is a sensitive indicator of autonomic response to noise during sleep. However, LF/HF ratio change was not apparent in similar analyses of HR variabilities (Fig. 4). Beat-by-beat and spectral responses showed no evidence of habituation over the three sessions.

Beat-by-beat responses were examined in 10-beat intervals commencing at noise noise peak, noise offset, and 10 beats after noise offset; and over the period from noise onset to offset. Brief sounds of rapid onset (tones, military aircraft flyovers) resulted in greater SBP and DBP increase than truck or civilian aircraft noise. There was no association between noise level and SBP response. This was true also for DBP response except at one time point—for 10 beats immediately following offset of the noise where DBP change was significantly greater at the highest noise level. In contrast, HR increased at the highest noise level used (75 L_{Amax}), but was not related to noise type, suggesting a threshold effect not present for BP. Figures 1-3 also suggest that HR increased and declined more rapidly than BP.

Mean BP and IBI changes are generally smaller than those shown by previous investigators.[8,9,10] This could be because our testing was done in the first 90 minutes after lights out, before REM and SWS are fully developed, and/or to reduced cardiovascular reactivity to noise in stage NREM in daytime sleep compared with night time sleep.[28] The range of noise levels used could also be a factor. Further studies would be required to clarify the roles of these factors. However, the absence of habituation across the three experimental days suggests that changes in circadian rhythm may not be a major factor in reduced cardiovascular response.

Sleep variables were not strongly related to beat-by-beat cardiovascular responses in our data. Nevertheless there were differences between BP and IBI responses. Wakening and alpha were associated with greater SBP response (in the Pk-Pre and NoisePd-Pre response categories). Alpha response was associated with increased Pk-Pre and PostA-Pre DBP. Sleep stage at noise onset and sleep stage change were not related to BP responses. On the other hand, decrease in IBI (in the PostA-Pre and PostB-Pre data) was related to sleep stage at noise onset, the IBI response being greatest when the subject was awake. Wakening and sleep stage change "up" were also related to IBI decrease in PostA-Pre and PostB-Pre categories respectively.

Our data have shown that beat-by-beat BP and HR respond to different aspects of the noise. BP related to rate of onset of the noise while HR appeared to have a minimum threshold in terms of noise level. Morgan et al.[10] observed a small increase in HR but a decrease in stroke volume and cardiac output following auditory stimulation during sleep. They also found that BP increased in the absence of EEG or (neurographic) sympathetic response and suggested that the increase in BP was due to peripheral vasoconstriction.

Also, while spectral analyses of SBP variabilities showed noise effects, HR variabilities did not. These results and the beat-by-beat analyses are consistent with regional differences in sympathetic response to noise.[13] In contrast, Pagani et al.[17] found strong similarities between spectra of arterial (BP) and surface HR (R-R) responses under conditions of tilt and B-blockade. These apparently conflicting findings may indicate that the vasculature and the heart respond in similar, or different, ways depending on background conditions and environmental stimuli.

In our data HR increase was related to waking and noise level, indicating a cardiac sympathetic component. Alpha response was related to BP but not to HR increase. Also, alpha responses are not recalled the next day.[29] These results suggest that noise of sudden onset, vasoconstriction, alpha response, and failure to recall the events the next day go together; while high level noise, heart rate increase, wakening, and recall the next day are associated.

Conclusion

The above results are consistent with differential responses to noise during sleep in different "regions" of the cardiovascular system, depending on the nature of the acoustic stimulus. Absence of habituation suggests that long-term exposure to transportation noise during sleep may be harmful, but research employing representative samples of populations sleeping in familiar conditions (at home) would be necessary to verify this. Such research would have implications for standards and regulations for noise control in respect of sleep, currently based solely on sleep disturbance.[30]

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