

The effects of recorded traffic noise on hemodynamic parameters in healthy women – a pilot study

K. Paunović¹, G. Belojević¹, V. Stojanov², B. Jakovljević¹

¹ Institute of Hygiene and Medical Ecology, School of Medicine, University of Belgrade, Dr Subotića 8, 11000 Belgrade, Serbia, paunkaya@gmail.com

² Center for Arterial Hypertension, Clinical Center of Serbia; School of Medicine, University of Belgrade Pasterova 2, 11000 Belgrade, Serbia, stojanovves@eunet.rs

INTRODUCTION

Road traffic noise is known to increase blood pressure or cause arterial hypertension in adults (Babisch 2006). However, the effects of noise on hemodynamic parameters have rarely been studied. During an experimental noise exposure in young adults, Belojevic et al. (2003) showed strong vasoconstrictive and hypodynamic cardiovascular effect of noise, but no statistically significant effects on blood pressure and heart rate. At the time, the effects of noise exposure on the autonomic nervous system (sympathetic and parasympathetic activity or sympathovagal balance) were not investigated. In another experimental study in an occupational setting, Kristiansen et al. (2009) showed changes in systolic blood pressure and an increase in sympathetic activity among noise-exposed individuals, who were engaged in cognitively demanding work tasks. Finally, Graham et al. (2009) reported a significant effect of the indoor traffic noise exposure on cardiac parasympathetic withdrawal during sleep, but no effect of traffic noise on cardiac sympathetic tone.

Thoracic electrical bioimpedance (TEB) is a comprehensive method of non-invasive follow-up of major cardiovascular hemodynamic parameters, such as blood pressure, heart rate, cardiac index (a minute volume of heart per square meter of body area), systolic index (the amount of pumped blood during each systole normalized for body surface area), enddiastolic index (the volume of blood in a ventricle at the end of diastole normalized for body surface area), contractility (rate of shortening of myocardial muscle fibers), vascular resistance index (the resistance to the flow of blood in the vasculature normalized for body surface area), ejection fraction (the percentage of blood pushed in each heart beat, equal to systolic index divided by enddiastolic index), left cardiac work index (an indicator of the amount of work; the left ventricle must perform to pump blood each minute, normalized for body surface area), and thoracic fluid conductivity (Stojanov et al. 2005). Beside non-invasiveness, the advantages of TEB include continuous ('beat to beat') and simultaneous monitoring of all hemodynamic parameters, monitoring of heart rate variability and blood pressure, and the overview of sympathetic and parasympathetic activity (sympathovagal balance). Beside its use in the follow-up of patients with cardiovascular diseases in the clinical setting, thoracic electrical bioimpedance can be applied in many areas of research, including the assessment of physiological effects of noise.

The aim of this experimental study was to assess hemodynamic changes using thoracic electrical bioimpedance in healthy women during a 10-minute exposure to recorded traffic noise in comparison to quiet conditions before and after noise exposure.

METHODS

This is a pilot phase of a large experimental study in the collaboration of the School of Medicine and the Multidisciplinary Center for Arterial Hypertension, Clinical Center of Serbia. Around 100 medical students were invited in 2009 to participate in the investigation. A total of 31 students (10 men and 21 women) agreed to take part in the study and signed an informed consent form. The study was approved by the Ethics Committee of the Clinical Center of Serbia.

All participants underwent a medical examination and blood pressure measurement by sphygmomanometer to exclude the presence of arterial hypertension. Participants with underlying diabetes ($n=1$), obesity ($n=2$), hypertension ($n=1$), arrhythmia ($n=1$), and participants with errors during the testing procedure ($n=5$) were excluded from the analysis. The final sample included 16 women and 5 men. We included only women in the study due to a small number of men for comparison. Mean age of the participants was 25.1 ± 1.6 years.

All participants were asked to avoid smoking, drinking coffee or engaging in intensive physical activity for at least two hours before the testing procedure.

The testing procedure consisted of three phases: a 10-minute rest in quiet conditions ($L_{Aeq}=40$ dB) at start (before noise exposure), a 10-minute exposure to recorded traffic noise ($L_{Aeq}=89$ dB), and a 10-minute rest in quiet conditions ($L_{Aeq}=40$ dB) after noise exposure. Two loudspeakers were placed at both sides of a subject's head at the distance of 30 cm. Equivalent noise levels were measured with Hand Held Noise Level Analyzer Type 2250 'Brüel & Kjær' at the level of participants' ear. The participants were lying on their back during the experiment, connected to impedance cardiogram and electrocardiogram, with an oscillometric blood pressure device on left arm, and a two-finger cuff for plethysmography or 'beat to beat' blood pressure monitoring. The following hemodynamic parameters were monitored with thoracic electrical bioimpedance device (Task Force® Monitor, CNS System Medizintechnik AG, Graz, Austria): blood pressure, heart rate, cardiac index, systolic index, total vascular resistance index, and sympathovagal balance (a ratio between a low frequency power – sympathetic influences on heart rate, and a high frequency power – vagal or parasympathetic modulation of heart rate).

The differences between noisy and quiet conditions were tested with Friedman's test for several related samples and with Wilcoxon signed ranks tests for two related samples.

RESULTS

Significant differences between the three experimental conditions were found for cardiac index (Friedman's test chi-square=14.926; $p=0.001$), systolic index (chi-square=8.222; $p=0.016$), and total peripheral resistance index (chi-square=8.375; $p=0.015$), but not for systolic pressure (chi-square=3.375; $p=0.185$), diastolic pressure (chi-square=2.317; $p=0.314$), heart rate (chi-square=5.375; $p=0.068$) and sympathovagal balance (chi-square=2.462; $p=0.292$).

Post hoc testing showed that cardiac index and systolic index were significantly decreased during noise exposure in comparison to quiet conditions before noise exposure, indicating a hypodynamic effect. On the other hand, peripheral vascular re-

sistance was significantly increased during noise exposure, indicating vasoconstriction (Table 1).

Both systolic and diastolic blood pressures were significantly higher during noise exposure in comparison to quiet condition before noise exposure. Heart rate was similar during noise exposure compared to quiet condition before noise exposure, but was significantly higher during noise exposure in comparison to the quiet condition after noise exposure. The effect of noise exposure on sympathovagal balance was not statistically significant (Table 1).

Table 1: Hemodynamic parameters of the investigated women before, during and after noise exposure

| Hemodynamic parameters | Quiet condition at start | Noise exposure | Quiet condition after noise | Between group comparison* (p value) | |
|---|--------------------------|----------------|-----------------------------|-------------------------------------|--------------------------------------|
| | | | | Quiet at start vs. noise exposure | Noise exposure vs. quiet after noise |
| Cardiac index (l/(min*m ²)) | 3.99±1.07 | 3.85±1.02 | 3.71±0.93 | 0.013 | 0.394 |
| Systolic index (ml/m ²) | 53.35±15.90 | 52.02±14.47 | 52.38±13.69 | 0.027 | 0.049 |
| Total peripheral resistance index (dyne*s*m ² /cm ⁵) | 1698.19±781.33 | 1823.88±836.07 | 1863.44±770.68 | 0.012 | 0.326 |
| Systolic pressure (mmHg) | 105.52±12.78 | 109.03±11.84 | 108.76±9.04 | 0.008 | 0.469 |
| Diastolic pressure (mmHg) | 67.05±11.09 | 69.92±12.27 | 70.92±9.47 | 0.023 | 0.733 |
| Heart rate (beat/min) | 76.62±15.64 | 75.32±12.77 | 71.63±11.16 | 0.339 | 0.026 |
| Sympathovagal balance | 1.02±0.88 | 1.00±0.58 | 0.88±0.49 | 0.998 | 0.102 |

Table legend: * Wilcoxon Signed Ranks Test

CONCLUSIONS

Exposure to recorded noise level of 89 dBA for 10 minutes had strong vasoconstrictive and hypodynamic effects in healthy women, with a significant increase of blood pressure. Further studies with thoracic electrical bioimpedance on a larger sample including both sexes will be valuable in explaining the effects of acoustic stress on cardiovascular regulatory mechanisms of blood pressure.

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