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Acute Effects of Environmental Noise Exposure on the Control of Cardiac Output in Healthy Adults

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Background and Objective: Epidemiologic studies have demonstrated that environmental noise exposure is associated with hypertension at night, but the related mechanism is unclear. This study aimed to investigate acute effects of environmental noise exposure on the control of cardiac output.

Method: We recruited 60 volunteers and divided them into a high-noise-exposure group of 23 and a low-noise-exposure group of 37 based on 24-hour personal noise measurement from a university. We determined individual noise exposure and measured personal cardiac parameters simultaneously at night. Linear mixed-effects regression models were used to estimate transient effects of noise exposure on cardiac parameters by adjusting some confounders.

Results: We found that high-noise-exposed subjects (70.6 ± 7.9 dBA) had significantly higher mean values of heart rate (HR ; 65.59 ± 10.45 bpm), stroke volume (SV ; 69.10 ± 11.24 ml) and cardiac output (CO ; 4.46 ± 0.80 L/min) than the low-noise-exposed ones (42.3 ± 10.7 dBA). An increase of one dBA increase in noise exposure at night was significantly associated with an increment of 0.03 L/min CO in the high-noise-exposed group, as well as 0.007 L/min CO in the low-noise-exposed group, after 60-minute time lag noise exposure. Additionally, the high-noise-exposure group had the significantly higher CO (0.19 ± 0.07 L/min) and HR (4.96 ± 1.14 bpm) compared with low-noise-exposed group over a night-time period.

Conclusions: Our findings suggest that exposure to environmental noise may be associated with a transient elevation of cardiac output at night by stimulating sympathetic nerves. One possible mechanism of hypertension caused by environment noise exposure may be through sympathetic stimulation to increase HR and CO. Future studies are encouraged to investigate the association between cardiac output and vascular changes for the development of hypertension.