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Cardiovascular effects of nocturnal aircraft noise on healthy volunteers
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Background: Nighttime aircraft noise can impair sleep quality and may also increase blood pressure and the risk for myocardial infarction for persons in highly noisy exposed residential areas. Pathophysiology and possible susceptibility factors for cardiovascular effects of aircraft noise remain unclear.

Methods and results: 75 healthy volunteers (mean age 26 y) were studied for three nights in their homes. One control pattern without noise (Control) and two noise patterns with 30 (Noise 30) or 60 (Noise 60) aircraft noise events were played back in random and blinded order. After each study night flow-mediated dilatation (FMD) of the brachial artery was measured the following the morning. Blood pressure (BP), heart rate and aciometric data were recorded continuously during noise exposure. Peak sound pressure levels were 60 dB(A) for both patterns, with Leq(30)=43.1±4.9 dB(A) for Noise 30 and Leq(30)=46.3±3.9 dB(A) for Noise 60. FMD (%) dose dependently declined from 10.4±3.8% (Control) to 9.7±4.2% (Noise 30) and 9.5±3.3% (p=0.025) after the night with 60 noise events (Noise 60). Effects on FMD were particularly evident if Noise60 followed after a night without noise, suggesting a possible priming effect of noise. BP rose from 109.8±15.4 mmHg to 114.9±13.9 mmHg in Noise 30 and to 115.2±14.4 mmHg in Noise 60. Average heart rate, heart rate variability and aciometric data did not differ between patterns. Adrenaline concentration increased from 38.6±11.4 ng/l to 33.0±16.9 ng/l (Noise 30) and 34.0±19.0 ng/l (Noise 60; p = 0.009). Serum cortisol remained unaffected by noise exposure.

Conclusion: A low risk population a short exposure to nocturnal aircraft noise may impair endothelial function, increase adrenaline concentrations and blood flow. Exposure to aircraft noise in prior nights seems to augment this effect.

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Low-dose dopamine infusion increases baroreflex sensitivity by inhibition of peripheral chemoreflex
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Background: Inverse relation between reflex responses from arterial baroreceptors and peripheral chemoreceptors is well described phenomenon. It is unknown whether dopamine infusion by inhibiting afferent signalizing from peripheral chemoreceptors would influence baroreflex sensitivity (BRS).

Methods: We performed double-blinded, crossover, randomized and placebo-controlled study on 111 healthy male volunteers (median age 26 years). During low-dose dopamine (2 mcg/kg/min) and saline infusions all subjects underwent a BRS assessment with a non-invasive, sequential method and an assessment of peripheral chemosensitivity with transient hypoxic method. Hemodynamic response to hypoxia was measured non-invasively using Nexfin monitor and expressed as slopes relating changes in systolic blood pressure (SBP) and heart rate (HR) to changes in oxygen saturation.

Results: Administration of low dose dopamine was associated with a significant reduction in peripheral chemosensitivity (median: 0.16 (In/SiP02/ In/SiP02) vs 0.40 mcg/ml/min (In/SiP02/In/SiP02); p < 0.001) with concomitant increase in BRS (1.42 bpm/mmHg; [1.17-1.99] vs 1.20 bpm/mmHg; [0.85-1.37], p < 0.05) compared to saline. Dopamine infusion decreased SBP and HR compared to saline (SBP: 5.7±3.8 vs 9.4±3.3 mmHg/SiP02, p=0.07; 14.2±6.4 vs 5.6±3.0 bpm/mmHgSiP02, 0.40±0.12 bpm/mmHgSiP02, 0.29±0.54 vs 0.56 bpm/mmHgSiP02, 0.46±0.64 vs 0.37±0.07 respectively, Low-dose dopamine infusion did not change SBP comparing to saline infusion.

Conclusion: Low-dose dopamine infusion has a beneficial effect on baroreflex sensitivity. We hypothesize that an increase in BRS is caused by concomitant attenuation of reflex response from peripheral chemoreceptors.

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Renal denervation attenuates impairment of renal function and kidney injury in obese spontaneously hypertensive rats
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Background: Hypertension and obesity are associated with increased systemic sympathetic drive possibly contributing to the development of renal dysfunction. Renal sympathetic denervation (RDN) is a new strategy to decrease sympathetic renalafferent nerve activity, leading to decreased central sympathetic drive.

Methods: Blood pressure was measured by telemetry. Kidney function was determined and renal perfusion was measured by Magnetic Resonance Imaging (MRI). Glomerulosclerosis was scored by semiquantitative morphometric evaluation and desmin staining was performed to quantify glomerular podocyte damage. Obese spontaneously hypertensive rats with RDN at the age of 34 weeks (SHR-ob RDN, n=15) were compared to sham operated SHR-ob (SHR-ob) and their normotensive lean controls (Ctr.). Animals were sacrificed at the age of 48 weeks.

Results: In SHR-ob, RDN significantly reduced blood pressure for 100 days (203±18 vs 166±7 mmHg, p < 0.001). Heart rate was not modulated. Renal cathecolamine levels, tyrosin hydroxylase staining and tyrosin hydroxylase protein levels were significantly reduced after RDN suggesting effective renal denervation. Development of renal dysfunction as characterized by increased urinary albumin/creatinine ratio and reduced glomerular filtration rate were attenuated by RDN in SHR-ob. Attenuation of renal injury by RDN was confirmed by renal desmin staining, a marker for glomerular podocyte damage. RDN prevented glomerulosclerosis, as scored by semiquantitative morphometric evaluation. Percentage of apoptotic TUNEL-positive cells and renal gene expression of p53 and BAX were significantly increased in SHR-ob but not modified by RDN. In SHR-ob, renal perfusion was significantly reduced compared to Ctr. and normalized by RDN.

Conclusion: In SHR-ob rats, RDN resulted in a stable and sustained blood pressure lowering effect for 100 days and attenuated impairment of renal function and glomerular damage. RDN may therefore provide protection in obese and hypertensive patients with renal dysfunction.