Introduction: Chronic use of hormonal contraceptives (HCs) is associated with increased resting blood pressure without alterations in muscle sympathetic nerve activity (MSNA). Whether this is secondary to increased sympathetic transduction to blood pressure (BP) has not been examined.
Purpose: We tested the hypothesis that young women using HCs would display augmented increases in BP following spontaneous bursts of muscle sympathetic nerve activity (MSNA) at rest compared to naturally cycling women (NAT).
Methods: Women were tested during the placebo phase (HC; n = 7, 22 ± 3 year, 21 ± 2 kg/m^2, 109 ± 9/68 ± 7 mmHg) or the early follicular phase (NAT; n = 5, 21 ± 2 yr, 23 ± 4 kg/m^2, 106 ± 11/66 ± 12 mmHg) to minimize the potential impact of changes in sex hormones on sympathetic regulation of BP. R-R interval (electrocardiography), beat-by-beat BP (finger photoplethysmography) and MSNA (peroneal microneurography) were measured during 10 min of supine rest. Signal averaging was used to characterize changes in BP for the 10 cardiac cycles following spontaneous MSNA bursts. Data are presented as mean ± SD.
Results: Neither resting mean arterial pressure (MAP; HC: 82 ± 7 vs. NAT: 80 ± 11 mmHg, $P = 0.71$), heart rate (HC: 69 ± 7 vs. NAT: 77 ± 13 bpm, $P = 0.24$), nor MSNA at rest (HC: 5 ± 2 vs. NAT: 7 ± 4 bursts/min, $P = 0.48$; HC: 8 ± 3 vs. NAT: 9 ± 5 bursts/100 heart beats, $P = 0.67$) were different between groups. Following spontaneous MSNA bursts, MAP increased over the subsequent 10 cardiac cycles in both groups (Time: $P < 0.001$, Group: $P = 0.10$, Interaction: $P = 0.99$). However, the peak increase in MAP was not different between groups (HC: Δ 3.0 ± 0.5 vs. NAT: Δ 3.5 ± 1.6 mmHg, p = 0.44).
Conclusions: These preliminary data indicate that spontaneous bursts of MSNA do not elicit greater increases in BP in young women using hormonal contraceptives, suggesting that heightened sympathetic transduction to BP may not contribute to the association between hormonal contraceptive use and future hypertension.
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Poster #70

Assessing vasomotor reactivity in patients with terminal heart failure after heart transplantation by contactless optical occlusive plethysmography

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Background and objective: After heart transplantation, the neurogenic regulation of the heart rhythm disappears, whereas the vasomotor regulation in the postoperative period has been insufficiently studied. Occlusal plethysmography (OP) allows assessing vascular neurogenic response but its technical implementation is rather complicated. Contactless approach to the reactivity evaluation using imaging plethysmography (IPPG) may facilitate wider adoption of this technique in clinical and scientific practice. The aim of our study was assessing musculocutaneous blood-flow reactivity in response to cold stress and deactivation of cardiopulmonary baroreceptors (CPBR) by OP using two techniques: classical air-plethysmograph and IPPG.
Materials and methods: We examined 13 patients with terminal heart failure (THF) aged 45.5 ± 15.2 years at the time of inclusion and 12 healthy volunteers of comparable age of the control group. Before and during 12 months after the surgery, the blood-flow response to cold stress and deactivation of cardiopulmonary baroreceptors (CPBR) was assessed in a forearm using a low body negative pressure chamber (LBNP − 20 mmHg). Registration of blood flow dynamics was performed by occlusion plethysmography (OP) using air-plethysmograph by Dohn and contactless IPPG.
Results: Significant decrease in blood flow response to cold vasoconstriction was observed in patients with THF compared to the control group: 0.02 ± 0.33 vs. 0.35 ± 0.12 rel.un., $P < 0.01$. Similarly, a decrease in CPBR deactivation was also found: 0.09 ± 0.08 vs. 0.31 ± 0.08 rel.un., $P < 0.001$. After heart transplantation, blood-flow reactivity was increased in response to cold stress up to 0.22 ± 0.12, $P < 0.01$, whereas there was no increase in response to LBNP: 0.16 ± 0.12; $P = 0.54$. It is worth noting that the blood flow reactivity measured by air plethysmography and IPPG correlated with each other: r = 0.68, p = 0.02 in the cold test and r = 0.57, p = 0.05 with CPBR deactivation.
Conclusion: Patients with THF show a significant decrease in vasomotor reactivity in response to cold stress and deactivation of CPBR, which is partially restored after heart transplantation. Vascular reactivity can also be assessed using the simple, contactless, and easy-to-use IPPG method.
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Poster #71

Computational modeling reveals triphasic progression of Lewy body diseases

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Background: Lewy body diseases (LBDs) are aging-related neurodegenerative disorders characterized by intra-neuronal deposition of the protein alpha-synuclein and by deficiencies of the catecholamines dopamine and norepinephrine in the brain and heart. Previously, computational analysis of a model consisting of a system of 9 coupled first-order parametrized linear ordinary differential equations (ODEs), accounting for 17 reactions, revealed abnormalities of catecholamine synthesis, vesicular storage, and neuronal recycling in LBDs.
Methods: To investigate the progression of catecholamine depletion in LBDs we constructed a substantially updated model with a system of 11 coupled first-order parameterized nonlinear ODEs and 25 reactions. The model incorporates autotoxicity from the dopamine metabolite 3,4-dihydroxyphenylacetaldehyde (DOPAL). DOPAL augments vesicular leakage and inhibits tyrosine hydroxylase (TH), L-aromatic-amino-acid decarboxylase, and vesicular uptake of cytosolic catecholamines. We investigated the model using the numerical continuation and bifurcation methods. Model predictions were compared with longitudinal trends in myocardial ^{18}F-dopamine- and putamen ^{18}F-DOPA-derived radioactivity (respective biomarkers of cardiac norepinephrine and striatal dopamine stores), post-mortem myocardial catecholamine contents, and effects of hypofunctional genetic mutations in preclinical studies.
Results: The model generated a triphasic curve for the loss of vesicular intra-neuronal norepinephrine stores. Longitudinal neuroimaging data in the heart and putamen of LBD patients fit this pattern. Post-mortem myocardial norepinephrine was drastically decreased (by 91% from control), as predicted. The model also predicted correctly that genetically decreased ALDH or VMAT2 activity would hasten the decline in catecholamine stores, while decreased MAO activity would retard the decline, in line with empirical data from animal studies.
Conclusion: We present a computational model that predicts a triphasic progression pattern of catecholamine deficiency in LBDs