by volume overload in both rat strains. Inhibition of RAS components attenuates progression of VO and its adverse consequences. Supported by grants VEGA 2/0158/19, 2/0076/16; APVV 15-0119, 15-0376 and EU ITMS 26230120006.

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Heart Rate Variability Mechanisms Analysed by Multiscale Information Decomposition

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Heart rate variability (HRV) – a marker of the cardiac autonomic nervous system control – results from the activity of several mechanisms operating across multiple temporal scales. To better characterize the mechanisms beyond HRV, the aim of our study was to evaluate the strength of information transfer among systolic blood pressure (SBP), heart rate (its reciprocal value – RR interval from ECG) and respiration volume (RESP) oscillations across multiple temporal scales. Seventy-eight healthy young volunteers (32 male, age range: 16.0 – 25.8 yrs.) participated in this study. We applied the multiscale partial information decomposition to quantify the amount of information transferred towards RR from SBP and RESP signals during supine rest, orthostasis (head-up tilt, HUT) and cognitive load (mental arithmetics, MA). The analysis was performed separately for raw data and slower oscillations. The unique transfer entropy from SBP to RR (a baroreflex influence) was significantly higher for the slower oscillations compared to raw data at rest and increased for raw data during both challenges. The unique transfer entropy from RESP to RR (a component of respiratory sinus arrhythmia independent of baroreflex) decreased during stress. The redundancy and synergy between RESP and SBP interacting with RR further elucidated mutual interactions among analysed signals. We conclude that the contribution of baroreflex and respiration in the HRV origin varies with the time scale. To better understand HRV, the measures quantifying the influence of major source signals (SBP or RESP) on RR signal (unique transfer entropies) together with interactions between sources – redundancy and synergy – should be assessed. Grants: VEGA 1/0117/17 and VEGA 1/0200/19 and “BioMed Martin” no. 26220220187.

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The Effect of Hypnosis on Systolic Blood Pressure

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Hypnosis is frequently used in numerous fields of complementary medical treatments. Blood pressure is an important vital sign. The changes manifest itself with hypo/ hypertension. Hypertension is a common and important health problem in society. Our aim is to evaluate the effect of hypnosis on blood pressure in healthy volunteers and to make a preliminary study for treatment of hypertensive patients. Healthy twelve volunteers, six women and six men, aged between 18 - 65 years were included after getting ethical permits and consent. The room selected for hypnosis was quiet and room temperature was standart to minimize the effects on blood presure. We used rapid hypnosis technique. Measurements were made under hypnonic trans of 10-15 minutes. Volunteers were awakened by countdown method. The non-parametric Wilcoxon Signed Tanks test was used as statistics to chary comparison test. $P<0.05$ was accepted as significant. The statistical results of all changes made by measurement of blood presure was found to be $p>0.05$. Although $p> 0.05$ was not significant in pre-hypnosis, we observed average 4 mmHg decrease of systolic blood pressure during hypnosis. As a result of our data, it can be a preliminary study to show that longer hypnosis can be effective in treatment of people with systolic hypertension. It will be appropriate to repeat this study with more people.

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The effect of lower body negative pressure on phase 1 cardiovascular responses at exercise onset in healthy humans

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We tested the hypothesis that vagal withdrawal and increased venous return interact in determining the rapid cardiac output response (Phase I) at exercise onset. We used lower body negative pressure (LBNP)

to increase blood dislocation to the heart by muscle pump action and simultaneously reduce resting vagal activity. At exercise start, we expected larger response amplitude for stroke volume and smaller for heart rate at progressively stronger LBNP levels, so that the cardiac output response would remain unchanged. Ten subjects performed 50 W exercise supine in Control condition and during -45 mmHg LBNP exposure. On single beat basis, we measured heart rate (HR), stroke volume (SV), and we calculated cardiac output (CO). We computed Phase I response amplitudes (A1) using an exponential model. SV A1 was higher under LBNP than in Control ($p < 0.05$). Conversely, the A1 of HR, was 23 ± 56 % lower under LBNP than in Control (although NS). Since these changes tended to compensate each other, the A1 for CO was unaffected by LBNP. The rapid SV kinetics at exercise onset is compatible with an effect of increased venous return, whereas the vagal withdrawal conjecture cannot be dismissed for HR kinetics. The rapid CO response may indeed be the result of two independent yet parallel mechanisms, as hypothesized, one acting on SV, the other on HR.

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Effects of wild-type and mutant forms of atrial natriuretic peptide on cardiac fibrosis in type-2 diabetes

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Diabetic cardiomyopathy is a slow progressive disease; it begins with a mild systolic dysfunction, followed by fibrosis and ends with hypertrophy and heart failure. Atrial natriuretic peptide (ANP) exerts a cardioprotective effect in pathophysiological processes such as fibrosis, diabetes and heart failure. A mutant form of ANP (mANP) was found to possess enhanced physiological properties as compared to the wild-type one. mANP appears to be more resistant to degradation and clearance than ANP, but its involvement in cardiac protection remains largely unclear. In this study, ANP and mANP similarly decreased in vitro proliferation and collagen secretion of freshly isolated murine ventricular fibroblasts treated with high glucose. This was mediated through a modulation of cGMP/PKG signaling and subsequently SMAD2/3 pathway inhibition. In vivo, type-2 diabetic mice showed systolic dysfunction and hypertrophy that were significantly ameliorated with mANP as compared to ANP. In addition, TGF-β1-related SMAD2/3 signaling showed less pro-fibrotic pattern in diabetic mice treated with mANP. This was associated to an increase in cardiac cGMP/PKG pathway culminating in an improvement of fibrosis. Our study shows for the first time that mANP activates cGMP/PKG more than wild-type ANP, in heart, and inhibits the fibrotic signaling pathways of TGF-β1 in type-2 diabetes. mANP could constitute an interesting therapeutic tool in cardiovascular diseases management.

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Cardiovascular responses and baroreflex sensitivity during apnoea phase 1 in spinal cord injury

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The first phase (ϕ1) of the human cardiovascular responses to apnoea implies a rapid decrease in systolic blood pressure (SBP) until a SBP minimum (Pmin) is attained. This is accompanied by an increase in heart rate (HR), interpreted as baroreflex attempt at correcting SBP fall. After Pmin attainment, SBP, total peripheral resistance (TPR) and stroke volume (SV) increase, reflecting overall sympathetic stimulation. In patients with spinal cord injury (SCI), we hypothesized different cardiovascular responses to apnoea for impaired autonomic system regulation. In this preliminary report, we show data on 9 patients (age 58.1±21.7) with different severity (2 complete and 7 incomplete lesions) and neurological level of SCI (4 cervical, 1 thoracic and 4 lumbar lesion), who performed apnoeas in supine position. Beat-by-beat SBP, mean blood pressure (MBP), HR, SV and TPR were continuously determined before and during apnoeas by a Portapres device. Baroreflex sensitivity (BS) in ϕ1 was determined as the slope of the linear HR versus MBP relationship before Pmin. At apnoea start (control), SBP and HR were respectively 134.1±27.9 mmHg and 77.0±10.1 bpm; the SV and TPR were 86.0±17.5 ml and 12.4±2.7 mmHg*min/l (HRUs). In ϕ1 (30.3±12.7 s), at Pmin, SBP was 95.2±28.5 mmHg and SV was 58.9±16.0 ($p<0.05$ versus control), while HR and TPR did not change. At ϕ1 end, HR was 80.0±12.7 bpm and TPR was 14.4±3.4 HRUs (NS versus control); SBP returned to control value (133.3±39.5 mmHg). BS, detected in 5 out of 9 patients, was -0.37±0.16 beats/min*mmHg. We conclude that the tested hypothesis cannot be dismissed after these results.

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Search for the source of the retinal relaxing factor