P.076: VENTRICULAR-ARTERIAL COUPLING IN A RAT MODEL OF REDUCED ARTERIAL COMPLIANCE


To link to this article: https://doi.org/10.1016/S1872-9312(07)70099-6

Published online: 21 December 2019
**Methods:** Forearm flow-mediated dilatation (FMD) was used as a measure of endothelial nitric oxide (NO)-dependent vasodilatation, and brachial artery dilatation in response to sublingual glyceryl trinitrate (GTN 25 μg) was used to assess endothelial-independent dilatation. Carotid intima media thickness (IMT) and pulse wave velocity (PWV), an index of arterial stiffness, was assessed using the Sphygmocor® system. All data were expressed as mean±SEM. P < 0.05 (two tailed) was taken as indicating statistical significance.

**Results:** Twenty patients with HF (IHD-14 & DCM-6) and 24 controls of similar age and sex. Patients were studied on usual medication. FMD was impaired in patients with HF compared to controls (4.4±0.6 vs. 6.6±0.6, p = 0.025), whereas GTN-induced dilatation was similar to controls (10.9±1.9% vs. 11.1±0.1%, p = 0.01). IMT was higher in HF patients (1.19±0.09 mm vs. 0.83±0.04 mm, p = 0.008), and PWV was greater in patients with HF (10.7±1.1 m/s vs. 8.5±0.4 m/s, p = 0.048). In subgroup analysis of the HF subjects, IMT was elevated specifically in the patients with IHD (1.16±0.03 mm vs. 0.80±0.04 mm in controls, p < 0.01) but not in those with DCM (0.96±0.11 mm vs. 0.81±0.05 mm in controls, p = 0.05); furthermore, the patients with IHD had higher PWV (11.5±1.3 m/s vs. 8.9±0.3 m/s in controls, p < 0.01), whereas those with DCM did not (8.1±0.6 m/s vs. 8.5±0.3 m/s in controls, p = 0.05). FMD impairment was similar in IHD and DCM subjects.

**Conclusions:** Patients with HF have endothelial dysfunction as well as elevated arterial stiffness and increased IMT, and that the latter two changes are seen specifically in patients whose HF is secondary to IHD.

**P.075 PREDICTORS OF LARGE ARTERIAL STIFFENING AND WAVE REFLECTIONS IN DIABETES: THE EFFECTS OF AUTONOMIC NEUROPATHY AND ERECTILE DYSFUNCTION**

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**Background and Aim:** Premature arterial stiffening (AS) may contribute to macrovascular complications in diabetes. Cardiovascular autonomic neuropathy (CAN) and erectile dysfunction (ED) are also associated with adverse cardiovascular outcomes. The aim of this observational study was to investigate whether and to what extent CAN and ED co-exist and influence AS in patients with diabetes.

**Methods:** Thirty male subjects with diabetes (type 1 and 2) (age range 39–74 yrs) but without overt cardiovascular disease were studied. AS and wave reflections were assessed by measuring pulse wave velocity (PWV) (carotid-femoral [cf] and carotid-radial [cr]) and augmentation index (AIx) (Sphygmocor®). Cardiovascular autonomic function was assessed by measures of blood pressure and heart rate variability during continuous ECG recording (Sphygmocor). Carotid sinus baroreflexes were assessed by measuring the effects of blood pressure and heart rate variability on the haemodynamic response to sublingual glyceryl trinitrate (GTN 25 μg).

**Results:** (Mean±SD): Comparing subjects with CAN (n = 16) versus subjects without CAN (n = 14), cfPWV was higher (10.8±2.8 m/s vs. 8.9±1.5 m/s, p = 0.05) despite no differences in age, brachial blood pressure, erectile function, cfPWV or AIx. Comparing subjects with severe ED (n = 17) versus normal erectile function (n = 13) there were no differences in arterial function despite higher systolic and diastolic blood pressure in subjects with severe ED. Multiple regression analysis (R² = 0.38, p < 0.01) identified CAN (autonomic score) (β = 0.66, p < 0.01) and ED (IIEF score) (β = 0.62, p < 0.014) as independent predictors of cfPWV but not of crPWV or AIx.

**Conclusion:** Both CAN and ED are independently associated with increased arterial stiffness in diabetes. CAN appears the stronger predictor and may exert a pathophysiological role in the process of aortic stiffening.

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Rodent models of isolated systolic hypertension (ISH) are rare. One exception is the vitamin D and nicotine (VDN) model, in which arterial calcification raises arterial stiffness and vascular impedance. Complete analysis of the effect of VDN on ventricular-arterial interaction is lacking. Wistar rats were treated with VDN (VDN group, n = 9) and a control untreated group (CTRL, n = 10) was included. At sacrifice, invasive indexes of cardiac function were obtained using a conductance catheter. Aortic pressure and flow were measured to derive impedance and ventricular-arterial interaction. VDN caused significant increases in systolic (138±6 mmHg vs. 116±13 mmHg) and pulse pressure (42±10 mmHg vs. 26±4 mmHg). Aortic compliance decreased (0.12±0.08 ml/mmHg vs. 0.21±0.04 ml/mmHg) and pulse wave velocity increased significantly (8.8±2.5 m/s vs. 5.1±2.0 m/s).

**Conclusion:** Ventricular-arterial coupling in the VDN group. Preload recruitable stroke work and end-systolic elastance were both elevated in the VDN group thus decreasing the ratio of arterial elastance over end-systolic elastance (0.94±0.30 vs. 1.57±0.60 CTRL). Wave reflection was augmented in the VDN group, expressed by the increase in the wave reflection coefficient (0.63±0.06 vs. 0.52±0.05 CTRL), as well as the amplitude of the reflected pressure wave (13.3±1.1 mmHg vs. 8.4±1.0 mmHg CTRL). VDN lead to development of ISH and provoked alterations in cardiac function, arterial impedance, arterial function, and ventricular-arterial interaction, which in many aspects are similar to effects of an aged and stiffened arterial tree. The VDN model may be a useful model to study the patho-physiological effects of increased arterial stiffnes.