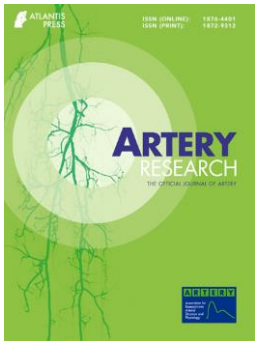

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13.6: SWITCHING TO BISOPROLOL/AMLODIPINE FDC ELIMINATES ADVERSE EFFECT OF A BETA-BLOCKER ON AORTIC PULSE PRESSURE AUGMENTATION

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13.5
VENTRICULAR-ARTERIAL COUPLING DURING TREATMENT WITH BISOPROLOL AND BISIPROLOL/AMLODIPIN IN HYPERTENSIVE PATIENTS

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Objective: To evaluate ventricular-arterial coupling in hypertensive patients after therapy with a beta-blocker and its fixed dose combination (FDC) with amlodipine.
Design and method: 28 patients (age 53,95±7,2, 20 males, BP 148,7±13,4/96,6±14,1 mmHg, HR 83,2±10,1 bpm) with untreated uncomplicated hypertension underwent simultaneous EchoCG and blood pressure (BP) acquisition at baseline, after 4 weeks of bisoprolol 5-10 mg monotherapy and after 8 weeks after switching to FDC bisoprolol 5-10/amlodipine 5-10 mg. Doses were titrated to reach BP <140/90 mmHg. Arterial elastance (Ea) and LV elastance (Ees) at rest were calculated as end-systolic pressure (ESP)/stroke volume (SV) and ESP/end-systolic volume (ESV). Ventricular-arterial coupling (VAC) was assessed as Ea/Ees. Mechanical efficiency of left ventricle (ELV) and peripheral arterial resistance (PAR) were evaluated also. p<0,05 was considered significant.
Results: After monotherapy with bisoprolol BP was 146,1±15,3/85,3±11,3 mmHg (p>0,05 vs baseline), HR 59,8±7,7 (p<0,05 vs baseline), after FDC 132,1±11,3/76,23±11,1 mmHg and 64,54±7,0 bpm, respectively (all p<0,05 vs baseline). Bisoprolol decreased Ees from 4,45±1,9 to 3,67±0,98 (p<0,05) whereas Ea, PAR did not change significantly. Ea/Ees increased significantly from 0,47±0,16 to 0,55±0,14 (p<0,05). Switching to bisoprolol/amlodipine FDC resulted in decrease of Ea from 1,88±0,39 at baseline and from 1,92±0,38 after bisoprolol monotherapy, PAR from 137,1±35,3 at baseline and from 128,9±36, respectively to 105,6±28. Ees did not change from that on bisoprolol, Ea/Ees (0,45±0,1) returned to baseline values. ELV did not change significantly throughout a study.
Conclusions: In hypertensive patients monotherapy with bisoprolol reduces initially increased Ees without negative effect on Ea and PAR and switching to bisoprolol/amlodipine FDC results in additional Ea reduction. Thus the study confirms potential benefits of bisoprolol/amlodipine in arterial hypertension in terms of cardiovascular functioning.

13.6
SWITCHING TO BISOPROLOL/AMLODIPINE FDC ELIMINATES ADVERSE EFFECT OF A BETA-BLOCKER ON AORTIC PULSE PRESSURE AUGMENTATION

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Objective: The aim of the study was to evaluate if combination with amlodipine eliminates the adverse effect of beta-blockers on aortic pulse pressure (PP) augmentation.
Methods: 28 previously untreated non-diabetic hypertensive subjects (age 53,6±5,7 years, 19 males) where treated bisoprolol 5-10 mg, if in 4 weeks BP >140/90 mmHg amlodipine 5 mg-10 mg was added to therapy to reach BP <140/90 mmHg. Before treatment, after monotherapy and after bisoprolol+amlodipin, applanation tonometry was done. The changes were considered significant if p<0,05.
Results: At the end of the study 23 patients were treated with bisoprolol 5/amlodipine 10 mg fixed dose combination, 5 — 10/10 mg. After 4 weeks of monotherapy brachial BP decreased from 153,9±9,1/83,4±7,5 to 146,7±8,3/85,1±3,4 mmHg, HR from 79,2±4,7 to 63,5±4,7 bpm (p<0,05). At the end of the study BP was 129,1±5,6/74,3±4,9 mmHg (p<0,05 vs baseline and monotherapy period), HR 62,8±4,9 bpm (p<0,05 vs baseline). Baseline central SBP was 143,2±8,2, PP 46,810,4 mmHg, augmentation index (AI) @HR 75 bpm 20±14%, PWV 10,5±2,1 m/s. After bisoprolol monotherapy the values were, respectively, 134±7,6, PP 44,2±7,3 mmHg, 27,1±16,1%, PWV 10,0±1,6 m/s. After further 4 weeks treatment with bisoprolo+amlodipine central SBP was 119,5±5,7 (p<0,05 vs baseline), PP 41,4±6,3 mmHg (p<0,05 vs baseline), AI@HR 75 bpm 21,9±6,5 % (p<0,05 vs baseline), PWV 9,6±1,0 m/s.
Conclusion: Monotherapy with bisoprolol increases central PP augmentation. Combining with amlodipine in a single pill eliminates the adverse effect of a beta-blocker on aortic PP augmentation and results in effective reduction of central SBP.

13.7
RENAL DENERVATION IN TREATMENT RESISTANT HYPERTENSION: EFFECTS ON CORONARY FLOW RESERVE AND FOREARM DILATION CAPACITY. A RANDOMIZED, DOUBLE-BLINDED, SHAM-CONTROLLED CLINICAL TRIAL

Morten Engholm [1], Jannik B. Bertelsen [1], Ole N. Mathiassen [1], Henrik Vase [1], Jesper N. Bech [3], Anne P. Schroeder [4], Ole Lederballe [4], Hans Rickers [5], Christian D. Peters [2], Ulla Kampmannf [6], Per L. Poulsen [6], Sten Langfeldt [7], Gratien Andersen [7], Klavs W. Hansen [8], Erling B. Pedersen [3], Jens F. Lassen [1], Hans E. Boetker [1], Niels H. Buus [9], Anne Kaltoft [1], Kent L. Christensen [1]
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Background: Microvascular impairment is well documented in hypertension. In this ReSET[1] sub-study we investigated the effects of renal sympathetic denervation (RDN) on coronary flow reserve (CFR) and coronary- and forearm minimum vascular resistance (C-Rmin and F-Rmin) in patients with resistant hypertension.
Methods: A randomised, single centre, double-blind, sham-controlled clinical trial in 58 patients with resistant hypertension randomised to RDN or SHAM. Inclusion criteria: ASBP-day > 145 mmHg following stable antihypertensive treatment and 2 weeks of compliance registration. RDN was performed with the unipolar Medtronic Flex catheter (Medtronic, California, USA). CFR and C-Rmin were determined with transthoracic Doppler echocardiography and F-Rmin with venous occlusion plethysmography at baseline and six-months follow-up.
Results: Baseline mean 24-h ambulatory BP was 111±1 mmHg (RDN, n=29) and 111±2 mmHg (SHAM, n=29). Similar reductions in MAP were seen at six-months follow up (-3.5±2.0 vs -3.2±1.8, p=0.92). Baseline CFR was 2.9±0.1 (RDN) and 2.4±0.1 (SHAM) with no significant change at follow-up (0.2±0.2 vs. -0.1±0.2, P=0.57). C-Rmin was 1.9±0.3 (RDN) and 2.7±0.6 (SHAM) (mmHg min/ml pr. 100 g LVM) and unchanged (0.3±0.5 vs. -0.4±0.8, P=0.48). F-Rmin was 3.6±0.2 (RDN) and 3.6±0.3 (SHAM) (mmHg min/ml pr. 100 ml tissue) and unchanged at follow-up (0.6±0.3 vs. 0.1±0.2, P=0.17). There was a tendency toward increased baseline LVMI in the SHAM-group (121±7 (SHAM) vs. 108±3 (RDN) g/m^2, P=0.08), but with proportional change at follow-up (-4±7 vs. 3±5, P=0.38).
Conclusion: RDN had no significant effect on CFR, C-Rmin and F-Rmin. Thus, data does not support microvascular improvement following RDN in resistant hypertension.
1. Mathiassen ON, Vase H, Bech JN, Christensen KL, Buus NH, Schroeder AP *et al*. Renal denervation in treatment-resistant essential hypertension. A randomized, SHAM-controlled, double-blinded 24-h blood pressure-based trial. *Journal of Hypertension* 2016.

13.8
VENTRICULO-VASCULAR INTERACTIONS AND THE ARTERIAL WINDKESSEL: NEW INSIGHTS FROM CARDIOVASCULAR MAGNETIC RESONANCE IMAGING BEFORE AND AFTER RENAL DENERVATION

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Background: Cardiovascular magnetic resonance (CMR) imaging is considered the gold standard for the evaluation of ventricular morphology and