same period. Even with the increase of the number of drugs over the evaluated period, we found that the defined diary dose of the medications in 2014 were similar when compared to 2009. Finally, LVH significantly decreased in 2014 compared to 2009 (93 \pm 27 g/m2 vs. 143 \pm 38 g/m2, p < 0.0001, respectively).

Conclusions: We conclude that an intensive treatment may improve BP control and target organ damage. A close follow-up is essential to assess and assist the evolution of the patients, and consequently, provide better cardiovascular outcomes.

PP.10.19 RENAL DENERVATION RAPIDLY RESTORES CIRCULATING PROGENITOR CELLS IN PATIENTS AFFECTED BY RESISTANT HYPERTENSION

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Objective: To investigate whether blood pressure (BP) lowering after renal sympathetic denervation (RSD) affects CD34+ cell number in drug-resistant hypertension (R-HTN).

Design and method: We enrolled 11 patients with R-HTN, already treated with at least 6 antihypertensive drugs, including a diuretic, at full dosages; patients with office BP of > 160 mmHg (>150 mmHg for type 2 diabetes) were considered eligible for the procedure. Adherence to drug treatment was accurately checked by patient's general practitioners. Mean age was 61 ± 7.9 years; M: F 8:5. We measured clinic (sphygmomanometer) and ambulatory (Tonoport V GE-Healthcare) BP, and heart rate (HR; electrocardiogram), at baseline and 30 days after RSD procedure (Symplicity; Medtronic). 24 h BP recordings and home BP protocols were consulted in addition to office BP measurements at the hospital before enrollment.

Results: At T0: SBP: 179.1 ± 9.3 mmHg; DBP: 101.2 ± 5.5 mmHg; HR 79.9 ± 9.4 ; CD34+ cells: 1.66 ± 0.51 . At T1 SBP values were reduced on the average of 40.2 mmHg (138.9 ± 7.3 ; -22.5%, p < 0.001) DBP of 18 mmHg (83.2 ± 3.2 ; -17.7%, p < 0.001), and HR of 10.4 bpm (67.3 ± 6.0 ; -17.7%, p < 0.005), and CD34+cell number increased on an average of 0.34 cells /microL (2.0 ± 0.51 ; +21.2%, p < 0.001).

Conclusions: RSD rapidly restores CD34+cell number in patients affected by true R-HTN; if these results will be confirmed on a larger scale, they could provide new insights about CD34+ cells and pathophysiological aspects of arterial hypertension.

PP.10.20 FLUORESCENCE ANALYSIS OF URINE SAMPLES FOR EVALUATION OF PHARMACOLOGICAL ADHERENCE IN RESISTANT HYPERTENSION

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Objective: Resistant hypertensive subjects (RH) present blood pressure (BP) above 140/90 mmHg, despite the use of three or more antihypertensive agents of different classes, including if possible a diuretic. The lack of BP control caused by non-adherence to medication can be falsely interpreted as resistance to drug treatment. Although it is well established that adherence to treatment is critical to achieve BP control, some practical difficulties, such as application of a direct and simple method with low cost and feasibility in the clinical setting still happen. Our goal was to test a simple method to evaluate the medication adherence of RH.

Design and method: Twenty-one patients referred to the Resistant Hypertension Clinic (Campinas, Brazil) had the triamterene included in their current prescription and were followed for 30 days period. We performed two random home visits to collect urine samples and test whether the patient took the medication in that day. Office BP, ambulatory BP monitoring (ABPM), home BP measurement (HBPM), biochemical data, and Morisky Adherence Scale (MMAS-8) were systematically acquired. Triamterene and its metabolite were measured by fluorimetric method. According to the fluorescence of urine, the subjects were divided between adherent and nonadherent.

Results: We found 9 compliant patients and 12 (57%) non-adherents. No differences were found between groups with respect to baseline characteristics or medications in use; the Kappa test showed concordance between MMAS-8 and fluorescence methods by 0.61 (95% CI 0.28–0.94; p < 0.01).Non-adherent patients had higher office (81 ± 11 vs 73 ± 6 mmHg, p = 0.03), baseline 24-h ABPM (75 ± 9 vs 66 ± 7 mmHg, p = 0.01), post protocol 24-h ABPM (74 ± 9 vs 67 ± 6 mmHg, p = 0.03) and HBPM (77 ± 9 vs 67 ± 8 mmHg, p = 0.01) than their counterparts.

Conclusions: Non-adherence to antihypertensive therapy is prevalent in resistant hypertension even if the patients are followed-up in specialized clinics. The fluorimetry method to detect triamterene intake in RH showed to be safe, feasible and easy to assess non-adherence and also associated with clinical parameter.

PP.10.21

THE EFFECT OF RENAL DENERVATION ON INFLAMMATORY MARKERS IN PATIENTS WITH RESISTANT HYPERTENSION AND DIABETES MELLITUS TYPE 2

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Objective: Chronic activation of the sympathetic nervous system (SNS) is causative in the pathophysiology of hypertension. Besides this, SNS activates immune system. Inflammation, and hypertension may interact and treatment of one of the two conditions could have some impact on the other. Renal denervation (RDN) is a promising treatment for hypertension. Beforehand, we hypothesized that a reduction of SNS following RDN may lead to a decreased inflammatory cytokines. The aim of this study was to evaluate the dynamics of TNF- α and interleukin-1 β after catheterbased RDN in patients with resistant hypertension and diabetes mellitus type 2.

Design and method: Thirty two patients with true resistant hypertension and type 2 diabetes mellitus were included in single-arm prospective interventional study (detailed protocol was published on ClinicalTrial.gov, number NCT01499810). Office blood pressure (BP) measurement, ambulatory 24-h BP, assessments levels of inflammatory cytokines (TNF- α , IL-1 β) were performed at baseline and 12 months after RDN. On average, patients were taking 4 (3–6) antihypertensive drugs. None of the patients changed the antihypertensive treatments during follow-up. A 12 months follow-up was completed by 26 patients (43–75 years old, mean aged 59.3 ± 7.9 years, 14 male).

Results: RDN reduced both systolic/diastolic office and 24-hour BP by -31.7/-12.8 mmHg, P > 0.01 for office BP and -13.4/-10.0 mmHg, P < 0.01, for 24-h BP. After RDN there was a decrease both TNF- α levels (from 2.21(1.54–3.65) to 1.4(1.11–1.47pg/mL), p = 0.007) and IL-1 b level (from 1.27 ± 0.60 to 1.00 ± 0.51, P = 0.02). There were no direct relationships between decrease in these cytokines and BP reduction.

Conclusions: Renal denervation may reduce activity of chronic inflammatory process and therefore may have potential benefit of inflammation inhibition in patients with resistant hypertension and diabetes mellitus type 2.

PP.10.23

EVALUATION OF SODIUM INTAKE IN PATIENTS WITH RESISTANT HYPERTENSION: CLINICAL PRACTICE IN GREECE

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Objective: Sodium and water retention is common in patients with resistant hypertension and sodium restriction has been found very effective in such patients. Measurement of sodium excretion in 24-hour (24 h) urine is a reliable marker of sodium intake. The present study evaluated the implementation of the measurement of 24hour urine sodium in patients with resistant hypertension in the daily practice.

Design and method: 186 patients with resistant hypertension were evaluated in our department's hypertension outpatient clinic. Patients were questioned if they: (a) have received advice for sodium intake restriction from their physicians, (b) have restricted sodium intake, (c) consumed small, moderate or large amounts of sodium and (d) have performed a 24 h urine sodium excretion measurement.

Results: 169 out of the 186 patients (91%) were instructed to restrict daily sodium intake but the duration of the given instruction was less than one minute in the majority of the cases (86%). 141 of them (76%) stated that they restricted sodium consumption. As for the amount of the intake, 69 (37%) stated that were receiving small amounts, 58 (31%) moderate amounts, 43 (23%) large amounts and the rest 16 (9%) very large amounts of sodium. 24 h urinary collection measurements were performed in 13 patients alone (7%), however only for urine albumin excretion calculations, while only in 2 (1%) of them, sodium excretion was calculated.

Conclusions: 24 h urine sodium excretion assessment is performed in a very small portion of patients with resistant hypertension in the daily clinical practice. Physicians should be efficiently informed for this easy, low cost and reliable method of sodium intake specification.