7B.11 SYSTOLIC BLOOD PRESSURE REDUCTION DURING ANTIHYPERTENSIVE THERAPY AND THE PREDICTION OF NEW-ONSET ATRIAL FIBRILLATION IN PATIENTS WITH ISOLATED SYSTOLIC HYPERTENSION. THE LIFE STUDY

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Objective: Atrial fibrillation (AF) is associated with increased cardiovascular risk and the incidence increases with age, hypertension and left ventricular hypertrophy (LVH). The predictive value of reduced in-treatment systolic blood pressure (SBP) for new-onset AF has not been examined in patients with isolated systolic hypertension (ISH).

Design and Method: Double-blind, randomized, parallel-group study included 1,320 patients with ISH and electrocardiographic LVH, enrolled in the Losartan Intervention For Endpoint reduction in hypertension (LIFE) study. Annual ECGs were Minnesota coded centrally, and 1,248 patients without AF by history or on baseline ECG, who were thus at risk of developing new-onset AF, were followed for 4.8±0.9 years.

Results: New-onset AF occurred in 61 patients (4.9%). Cox regression analyses were used to assess the predictive value of reduced in-treatment SBP for new-onset AF (Table).

Predictor	Variable	Hazard Ratio	95% CI	P Value
Univariate	SBP (per 10 mmHg decrease)	0.87	0.75–1.01	0.07
Bivariate†	SBP (per 10 mmHg decrease)	0.87	0.76–1.00	0.066
Multivariate‡	SBP (per 10 mmHg decrease)	0.83	0.73–0.95	0.008

† Adjusted for possible treatment effects with losartan vs atenolol.
‡ Adjusted for treatment effect, baseline SBP, Framingham risk score, history of coronary heart disease, and in-treatment heart rate and in-treatment ECG-LVH determined by Cornell voltage–duration product.

Conclusion: Lower in-treatment SBP was associated with a 17% risk reduction for new-onset AF per 10 mmHg decrease in SBP, independent of treatment modality, baseline risk factors, baseline SBP and in-treatment heart rate and ECG-LVH in patients with ISH. In multivariate analysis the association was highly significant, reflecting an enhanced confounding effect of treatment on time-varying SBP when also adjusting for the above mentioned factors.

7B.12 LACK OF REGRESSION OF LEFT VENTRICULAR HYPERTROPHY IS ASSOCIATED WITH HIGHER INCIDENCE OF REVASCULARIZATION IN HYPERTENSION. THE LIFE STUDY

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Objective: Regression of left ventricular hypertrophy (LVH) and albuminuria is associated with decreased cardiovascular (CV) risk. We aimed to investigate the importance of regression of LVH and albuminuria for the incidence of revascularization.

Design and Method: In 9124 hypertensive patients included in the LIFE study, we measured urine albumin/creatinine ratio (UACR), LVH by electrocardiography, serum high density lipoprotein (HDL) cholesterol and blood pressure after two weeks of placebo treatment and yearly during five years of anti-hypertensive treatment with either an atenolol- or a losartan-based regimen.

Results: In Cox regression analyses *coronary* revascularization was predicted by high time-varying Sokolow-Lyon voltage and low time-varying UACR, together with low time-varying HDL cholesterol, high Framingham risk score, history of angina pectoris, and prior myocardial infarction (Table). *Peripheral* revascularization was predicted by high time-varying Sokolow-Lyon voltage, but not time-varying UACR or Cornell product, together with low time-varying HDL cholesterol, high time-varying pulse pressure, high Framingham risk score, history of peripheral vascular disease and prior myocardial infarction.

Conclusions: Continuing high Sokolow-Lyon voltage was in contrast to UACR and Cornell product associated with higher incidence of coronary as well as peripheral revascularization independently of prior CV disease and traditional CV risk factors.

Variable	Coronary revascularization		Peripheral revascularization	
	HR (95% CI)	P value	HR (95% CI)	P-value
Time-varying Sokolow-Lyon	1.02 (1.00–1.03)	0.005	1.02 (1.00–1.03)	0.01
Time-varying HDL	0.47 (0.32–0.71)	<0.001	0.63 (0.40–1.00)	0.05
Time-varying UACR	0.93 (0.88–0.98)	0.003	1.01 (0.93–1.10)	0.80
Time-varying Cornell	1.01 (1.00–1.02)	0.07	1.00 (0.98–1.01)	0.39
Time-varying pulse pressure	0.94 (0.87–1.01)	0.08	1.16 (1.08–1.25)	<0.001
Treatment allocation	1.04 (0.84–1.29)	0.74	0.87 (0.67–1.13)	0.29
Framingham Risk Score	1.04 (1.03–1.05)	<0.001	1.04 (1.03–1.06)	<0.001
Myocardial infarction	1.46 (1.05–2.03)	0.02	1.74 (1.20–2.52)	0.004
Angina pectoris	2.71 (2.08–3.53)	<0.001		
Peripheral vascular disease			5.43 (4.02–7.35)	<0.001

Oral Session 7C

HEART

7C.1 LEFT VENTRICULAR STRUCTURE IN RELATION TO ALDOSTERONE AND RENAL SEGMENTAL SODIUM HANDLING

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Objective: Previous studies reported on the association of left ventricular mass index (LVMI) with urinary sodium or with circulating or urinary aldosterone. We investigated the independent associations of LVMI with the urinary excretion of both sodium and aldosterone.

Design and Method: We randomly recruited 317 untreated subjects from a White population (45.1% women; mean age 48.2 years). Measurements included echocardiographic left ventricular properties, the 24 h urinary excretion of sodium and aldosterone, plasma renin activity (PRA), and the proximal (RNaprox) and distal (RNadist) renal sodium reabsorption, as assessed from the endogenous lithium clearance. In multivariable-adjusted models, we expressed changes in LVMI per 1 SD increase in the explanatory variables, while accounting for sex, age, systolic blood pressure and the waist-to-hip ratio.

Results: LVMI increased independently with the urinary excretion of both sodium (+2.48 g/m^2; P = 0.005) and aldosterone (+2.63 g/m^2; P = 0.004). Higher urinary sodium excretion was associated with increased MWT (+0.126 mm; P = 0.054), but with no change in LVID (+0.12 mm; P = 0.64). In contrast, higher urinary aldosterone excretion was associated with higher LVID (+0.54 mm; P = 0.017), but with no change in MWT (+0.070 mm; P = 0.28). Higher RNadist was associated with lower RWT (−0.81×10^{-2}, P = 0.017), because of opposite trends in LVID (+0.33 mm; P = 0.13) and MWT (−0.130 mm; P = 0.040). LVMI was not associated with PRA or RNaprox.

Conclusions: LVMI independently increased with both urinary sodium and aldosterone excretion in a population-based sample. Increased MWT explained the association of LVMI with urinary sodium and increased LVID the association of LVMI with urinary aldosterone.

7C.2 EVOLUTION OF LEFT VENTRICULAR MASS IN WHITE-COAT HYPERTENSION: A PROSPECTIVE COHORT STUDY

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Objective: White-coat hypertension (WCH) has been considered a benign clinical condition for long, but recent longitudinal studies performed in large samples indicate that patients with WCH may be at increased risk of events. No study reported on the evolution of left ventricular mass in this hypertension category.

Design and Method: Participants were 470 never-treated young adults (330 men) with a mean baseline age of 33.8±8.5 years (range, 18 to 45 years), who were screened for stage 1 hypertension on at least two

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