cardiovascular safety of febuxostat and allopurinol in individuals with symptomatic hyperuricemia previously receiving allopurinol. At the screening visit, patients were invited to participate in the Blood Pressure (BP) sub-study. A validated BP monitor and an instruction booklet were provided. Each set of home BP measurements was taken over four consecutive days, three times each in the morning and evening. BP was recorded at baseline (on allopurinol therapy), after 7-days allopurinol washout, and 8 weeks after randomisation to febuxostat or allopurinol. Differences in BP in the allopurinol and febuxostat group were evaluated. The relation in ship between serum uric acid level reductions and BP changes was also assessed.

**Results:** Between December 2012 and January 2014, 298 FAST participants consented to the BP sub-study. 223 participants who submitted a complete set of BP data were included in the analysis; 104 were randomised to Allopurinol and 119 to Febuxostat. Baseline characteristics were balanced between groups. Overall mean change in BP from washout to 8 weeks of randomised treatment was not significant in either group [Mean BP change in Allopurinol arm: SBP -0.10 mmHg (95% CI -1.76,1.56), p = 0.9; DBP 0.50 mmHg (95% CI -1.82,2.81), p = 0.6. Mean BP change in Febuxostat arm: SBP -0.77 mmHg (95% CI -4.20,2.67), p = 0.7; DBP -0.41 mmHg (95% CI -2.56,1.73), p = 0.7]. No significant BP differences were observed between groups at each time point. Reduction in serum uric acid by > 60 μmol/L at 8 weeks on either therapy was not associated with statistically significant changes in BP.

**Conclusions:** There was no significant difference in the effect of allopurinol or febuxostat on blood pressure after 8 weeks of randomised therapy in the FAST study. Larger reductions in serum uric acid at 8 weeks had no effect on BP change.

**IMPACT OF ROUTINE BRAIN IMAGING ON THE PROGNOSIS OF PATIENTS WITH LEFT-SIDED VALVE INFECTIVE ENDOCARDITIS WITHOUT NEUROLOGICAL MANIFESTATIONS**

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**Objective:** There are limited data on the impact of routine use of brain magnetic resonance imaging (MRI) on the prognosis of neurologically asymptomatic patients with left-sided infective endocarditis (IE).

**Impact of routine brain imaging on the prognosis of patients with left-sided valve infective endocarditis (IE) without neurological manifestations**

Design and method: Among patients diagnosed with possible or definite IE in two tertiary referral centers, we identified 527 left-sided IE patients without neurological symptoms or signs at the time of diagnosis. Patients who underwent brain MRI within 1 week after the IE diagnosis were classified as the routine brain imaging group (n = 216), and the rest were categorized as the control group (n = 311). All endpoints were compared after adjustment using inverse probability of treatment weighting (IPTW).

**Results:** During a median follow-up of 57 months, the routine brain imaging group had a similar risk of 3-month all-cause mortality to the control group in the multivariate analysis [hazard ratio (HR) = 1.13 (95% CI 0.64–1.98)] and IPTW-adjusted cohort (HR, 0.59; 95% CI, 0.30–1.10). The risks of attributable mortality (defined as death directly related to IE) and fatal neurological events were also similar between the two groups in the multivariable analysis and IPTW-adjusted cohort. In the subgroup analysis, the routine brain imaging group showed more favorable outcomes in cases of large vegetation (> 10 mm) or acute-onset microorganisms.

**Conclusions:** Routine use of brain MRI in left-sided IE patients without neurological manifestations is not associated with improved clinical outcomes. However, routine brain imaging in appropriate clinical settings, such as in cases of large vegetation or acute-onset microorganisms, could improve clinical outcomes.

**VASODILATION IS THE PREDOMINANT FACTOR OF BLOOD PRESSURE LOWERING DURING DEVICE-GUIDED SLOW BREATHING– ANALYSIS BY WAVE SHAPE PARAMETERS OF PULSE WAVE ANALYSIS**

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**Objective:** There is evidence that device-guided slow breathing with direct bio-feedback of pulse arrival time (PAT) leads to favorable changes (increase) in PAT and blood pressure (decrease). Slow breathing modulates peripheral blood flow and may decrease blood pressure by vasodilation.

The objectives of the study presented here was to identify hemodynamic mechanisms of the blood pressure lowering effect of device-guided breathing observed in a previously published study (Mengden Blood Pressure Monitoring 2023).

**Design and method:** Patients with a systolic BP 130-160 mmHg or treated essential hypertension (21 females / 23 males) were trained to perform repeated and unattended device guided slow breathing exercises for 10 minutes and 5 minutes cool down over 5 days.

Furthermore, they were skilled to perform a self-measurement of blood pressure before and after the breathing exercise using a validated upper-arm device.

A simple device was used to measure pulse arrival time by electrocardiography and photoplethysmography connected with a smartphone or tablet to provide biofeedback. Using pulse wave analysis, time to systolic peak (tys) and time to diastolic notch (tnotch) were measured and normalized to RR interval i.e., as tsys / RR and tnotch / RR.

**Results:** Oscillometric self measured blood pressure showed a reproducible decrease in systolic blood pressure of 5 mmHg (p<0.01, SD 8 mmHg).

 Absolute tys increased by 13ms (p<0.001, SD 36 ms) and relative tys / RR increased by 0.013 (p<0.001, SD 0.033) by the end of the exercise compared to baseline after cooling down (15 min). Similarly absolute tnotch increased by 11ms (p<0.001, SD 31 ms) as well as relative tnotch / RR in total by 0.011 (p<0.001, SD 0.036) compared to baseline.

**Conclusions:** The delayed arrival of the first pulse wave maximum and the prolonged ejection time after adjustment for heart rate can be assumed here as a surrogate of a decreasing afterload. It is thus evident from this perspective that vasodilation is an important factor of blood pressure lowering during device-guided breathing.

**ACUPUNCTURE IN ARTERIAL HYPERTENSION: EVALUATION OF ITS EFFICACY WITH BOTH OFFICE AND AMBULATORY BLOOD PRESSURE MEASUREMENTS**

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**Objective:** A possible alternative to pharmacological antihypertensive therapies in grade 1 low risk hypertensive patients or in those experienced drugs adverse effects could be acupuncture.

**Design and method:** We focused on its possible effects on BP both as Office BP (OBP) and as Ambulatory BP Monitoring (ABPM) evaluating it before starting and after its completion.

**Results:** Oscillometric self measured blood pressure showed a reproducible decrease in systolic blood pressure of 5 mmHg (p<0.01, SD 8 mmHg).

Within session SBP decrease was -5.8 mmHg (-3.75%) during the first session (from 85.3±9.1 to 82.1±7.5, p = 0.03; and from 88.5±9.3 to 85.7±7.8, p = 0.02).

Among patients with possible or definite left-sided IE diagnosed with or without neurological symptoms or signs at the time of diagnosis, patients with left-sided infective endocarditis (IE).
while it falls to -2.1 mmHg (-1.25%) and stands firmly under 2 mmHg for all the next session. At the last session SBP reduction was -1.9 mmHg (-1.6%).

Conclusions: We found a significant reduction in office, 24h and day-time ABPM SBP determined by a 6-weeks twice weekly acupuncture cycle that lasts at least for the first two months after its completion.

EFFECTS OF PCSK-9 INHIBITORS ON ARTERIAL STRUCTURE AND FUNCTION: PRELIMINARY RESULTS

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Objective: One of the latest chapters in the research for new and stronger lipid lowering therapies has led to the development of PCSK9 inhibitors. In addition to the benefit on lipid profile, a limited number of studies have been published showing a possible improvement in vascular function and structure following their administration. To evaluate the effects of PCSK9 inhibitors on structural (via carotid Intima Media Thickness – IMT) and functional (carotid-femoral Pulse Wave Velocity – cf-PWV, and brachial artery Flow Mediated Dilatation - FMD) arterial properties.

Design and method: Twenty-two dyslipidemic patients with a clinical indication to PCSK9 inhibitors were prospectively recruited at the Cardiology Rehabilitation of ASST Grande Ospedale Metropolitano Niguarda Hospital (Milan, Italy). Anthropometric, clinical and therapeutic data were recorded. Each patient underwent instrumental examinations to assess cf-PWV, IMT and FMD at T0 (before the start of therapy), T1 (after 6 months) and T2 (after 12 months).

Results: In the 22 patients who reached 6 months of follow up there was a significant reduction in LDL cholesterol (from 127.8±31.7 to 47.4±29.1 mg/dl, p=0.001) while there was no significant difference in arterial parameters (cf-PWV: from 10.5±3.5 to 9.4±2.3 m/s, p = 0.092; FMD: from 11.4±1.8 to 9.8±3.8%, p = 0.407; IMT: from 775.6±192.8 to 768.7±144.7, microm, p = 0.854). Similar results were also obtained in the 12 patients who reached one year of follow-up: for LDL from 129.4±29.1 to 48.8±21.9 mg/dl, p<0.0001; cf-PWV from 10.4±3.3 to 7.7±2.9, p = 0.087; FMD from 10.2±0.9 to 11.0±5.6%, p = 0.930; IMT from 745.1±203.9 to 770.0±127.3 microm, p = 0.710.

Conclusions: Our study confirm the strong LDL reduction with PCSK9 inhibitors that are needed in order to reach the targets set by the ESC 2019 guidelines. In contrast, they do not show a significant effect on arterial function and structure in terms of cf-PWV, FMD and carotid IMT.

ISTO STUDY - INTENSIVE-INTERMEDIATE STATIN THERAPY OBSERVATIONAL STUDY

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Objective: lifestyle changes are frequently insufficient to reduce cardiovascular risk (CVR) in patients with dislipemia. This study aims to characterize the long-term evolution of lipid profile and CVR of patients under primary prevention.

Design and method: A retrospective study was performed of outpatients at a Portuguese cardiovascular risk clinic with > 2 CVRF, followed >2 years between 1995 and 2015 (Fig. 1). Intermidiate-intensity statin therapy had initiated early, in accordance with the clinic’s practice. After written informed consent was obtained, sociodemographic and clinical characteristics were collected from medical records, at baseline and last visit. Changes in lipid profile and CV risk scores (FRS, ASCVD, SCORE) were estimated. Associations between HDL-C or LDL-C changes and gender, age, observation times and treatments were assessed through bivariate analysis and multiple linear regression models.

Results: Out of 516 participants with mean follow-up of 11.4 +/- 4.3 years, 56.6% were female and received intermediate-intensity statins. Lipid profile showed statistically significant improvement, including median changes in LDL-C and HDL-C of -77.0 mg/dl and +19 mg/dl, respectively. CVR also showed statistically significant improvements according to all scores. Statin therapy resulted in a mean HDL-C increase of 7.4 mg/dl (independently of gender and other treatments) and a mean LDL-C reduction of 51.8 mg/dl (irrespective of age and other treatments).

Conclusions: Results from this long-term real-life study indicate that primary prevention, specifically early and continuous therapy with intermediate-intensity statins as an add-on to lifestyle interventions, was important in obtaining consistent and adequate metabolic correction in patients with additional risk factors.

LIPOPROTEIN (A): TREATMENT IN THE METABOLIC CONTEXT

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Objective: Lipoprotein(a) [Lp(a)] is an independent cardiovascular risk factor but is closely associated with other similar risk factors that are manageable with appropriate treatment and guidance. We aimed to study the impact of using combined therapy for managing Lp(a) levels in patients at high cardiovascular risk but without major adverse cardiovascular events, in primary prevention.

Table 1: Associations between gender and pharmacological therapies and changes in Lp(a) levels between the initial and final measurement (excluding patients who died)

| Variables | n | Mean Difference | Median Difference | Range (min-max) |
|-----------|---|----------------|------------------|----------------|
| Gender    |   |                |                  |                |
| Male      | 279 | -20.7±64.7      | -29.0±46.0       | 0.000 |       |
| Female    | 202 | -33.5±66.7      | -32.0±45.0       | 0.000 |       |
| Statins   |   |                |                  |                |
| Yes       | 38  | -18.3±13.2      | -18.0±10.0       | -0.000 |       |
| No        | 443 | -33.0±15.0      | -31.0±10.0       | -0.000 |       |
| ACEIs     |   |                |                  |                |
| Yes       | 222 | -27.8±9.4       | -25.0±7.0        | -0.000 |       |
| No        | 125 | -35.3±15.5      | -34.0±9.0        | -0.000 |       |
| ARBs      |   |                |                  |                |
| Yes       | 274 | -29.3±14.3      | -27.0±9.0        | -0.000 |       |
| No        | 206 | -35.7±16.8      | -34.0±9.0        | -0.000 |       |
| OXMs      |   |                |                  |                |
| Yes       | 327 | -30.0±15.3      | -27.0±9.0        | -0.000 |       |
| No        | 143 | -36.2±14.6      | -36.0±9.0        | -0.000 |       |
| Antipillatins |   |                |                  |                |
| Yes       | 217 | -27.3±14.8      | -24.0±9.0        | -0.000 |       |
| No        | 263 | -25.7±13.4      | -25.0±9.0        | -0.000 |       |
| CCBs      |   |                |                  |                |
| Yes       | 240 | -29.0±14.5      | -27.0±9.0        | -0.000 |       |
| No        | 191 | -36.9±15.9      | -35.0±9.0        | -0.000 |       |
| Alligepidos |   |                |                  |                |
| Yes       | 242 | -27.8±14.1      | -25.0±9.0        | -0.000 |       |
| No        | 239 | -25.9±15.8      | -24.0±9.0        | -0.000 |       |
| Antipillatins |   |                |                  |                |
| Yes       | 102 | -20.3±14.9      | -20.0±10.0       | -0.000 |       |
| No        | 179 | -32.4±15.8      | -30.0±10.0       | -0.000 |       |

AESC: angiotensin-converting enzyme inhibitors; ARBs: angiotensin receptor blockers; CCBs: calcium channel blockers; MM: non-parametric Mann-Whitney test; OXMs: oral anticoagulants drugs; SD: standard deviation.

Design and method: We conducted a retrospective observational study in 516 patients randomly selected from a group of 1677 patients who attended cardiovascular risk clinic, following >2 years between 1995 and 2015. The disorders observed and therapies used were classified into nosological and pharmacological groups, respectively. Cardiovascular risk was calculated based on the Framingham risk score, the European Society of Cardiology’s SCORE and the American College of Cardiology’s ASCVD Risk Estimator, and changes in patients’ lifestyle were assessed.

Results: Significant differences (p<0.001) were found in almost all metabolic variables, except fasting insulin and C-peptide. Lp(a) levels were also significantly reduced (p<0.001). Carotid intima-media thickness improved, decreasing from 2.90 mm to 1.40 mm; however, there was no reduction in the number of cases of vascular stenosis. Of patients with hepatic steatosis (85.5%), 40.7% presented hepatomegaly, but liver function was only altered in a few patients (14.5%). Lipid-lowering therapy, especially statins, significantly decreased Lp(a), benefiting from synergy with other treatments aimed at other metabolic situations associated.

Conclusions: Lp(a) is a key overall indicator of vascular risk and should be considered a target in context of combined therapy. Besides a healthy lifestyle, primary prevention should include combined drug therapies to address all cardiovascular risk factors and to delay the atherothrombotic process.

DIETARY SODIUM RESTRICTION REDUCES NOCTURNAL BLOOD PRESSURE IN PATIENTS WITH TREATMENT RESISTANT HYPERTENSION

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