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Cor et Vasa

S PŘÍLOHOU Kardio

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Cor et Vasa

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RISK STRATIFICATION, INCIDENCE, AND LONG-TERM CLINICAL OUTCOMES IN TAKOTSUBO CARDIOMYOPATHY: INSIGHTS FROM A 7-YEAR FOLLOW-UP STUDY

Abumayyaleh M[1], Hamdani N[2], Schupp T[3], Behnes M[3], El-Battrawy I[4], Akin I[3]

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Form: oral presentation, **theme:** cardiomyopathy and cardiac fibrosis
Background: Takotsubo cardiomyopathy (TTC) has been increasingly recognized over recent years. However, data regarding long-term clinical outcomes, temporal trends, and risk stratification remain limited.
Methods: Patients diagnosed with TTC between 2009 and 2018 were included in this retrospective study. Clinical endpoints included all-cause mortality at 7-year follow-up, in-hospital mortality, stroke, life-threatening arrhythmias, heart failure, thromboembolic events, and recurrence of TTC.
Results: A total of 153 patients with TTC were included. Of these, 85% were female, with a mean age of 66.4 ± 12.6 years. In-hospital mortality was 7.2%, and the incidence of in-hospital life-threatening arrhythmias was 9.3%. During the 7-year follow-up period, overall all-cause mortality was 39.9%. The incidence of stroke was 9.2%, life-threatening arrhythmias 9.9%, incident heart failure 24.3%, thromboembolic events 20.5%, and recurrence of TTC 7.2%. Newly diagnosed malignant tumors occurred in 11.1% of patients during follow-up. In multivariable analysis, female sex (HR 0.26, 95% CI 0.12–0.58, $p < 0.001$), body weight (HR 0.97, 95% CI 0.95–0.99, $p = 0.002$), apical ballooning pattern (HR 3.91, 95% CI 1.58–9.65, $p = 0.003$), chest pain at presentation (HR 0.35, 95% CI 0.19–0.66, $p = 0.001$), and creatinine levels (HR 1.91, 95% CI 1.30–2.80, p = 0.001) were identified as independent predictors of long-term mortality.
Conclusions: In this monocentric study, TTC was associated with high all-cause mortality and a substantial incidence of heart failure during 7-year follow-up, whereas long-term life-threatening arrhythmias were uncommon. These findings highlight the long-term clinical burden of TTC and emphasize the need for improved risk stratification and follow-up strategies in this patient population.

DIURNAL VARIATION IN SYMPTOM ONSET IN SPONTANEOUS CORONARY ARTERY DISSECTION: EVIDENCE FOR A BIMODAL PATTERN

Al-Gully J[1], Montero-Cabezas JM[1], Eikenboom J[2], den Exter PL[2], ter Haar SNM[2], Forouzanfar J[1], Campbell C[1], den Haan MC[1], van der Kley F[1], Jukema JW[1], Bingen BO[1], Al Amri I[1]

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Form: poster or oral presentation, **theme:** circadian rhythms and cardiometabolic syndrome
Background: Atherosclerotic acute coronary syndromes (ACS) show time-of-day variation in symptom onset, with a well-described morning predominance and, in several series, a secondary evening peak. Whether spontaneous coronary artery dissection (SCAD), a mechanistically distinct, non-atherosclerotic cause of ACS, shows time-of-day variation in onset is unknown.
Objectives: To characterize the diurnal distribution of symptom onset in SCAD and to explore, as secondary and exploratory analyses, associations between time of onset and clinical, trigger-related, and angiographic characteristics.
Methods: We retrospectively analysed 195 consecutive SCAD patients from an institutional registry. Symptom onset time, or time of first medical contact when onset was unavailable, was classified into four 6-hour intervals: night (00:00–05:59), morning (06:00–11:59), afternoon (12:00–17:59), and evening (18:00–23:59). The pre-specified primary analysis assessed variation in hourly onset frequency using Poisson harmonic regression with likelihood-ratio testing. Baseline characteristics, trigger type, and angiographic findings were compared across time intervals as secondary exploratory analyses.

Results: The distribution of onset times departed significantly from uniformity (p <0.001). A bimodal fit was superior to a unimodal model (p = 0.002), with estimated peaks at 10:36 (95% CI 9:36–11:36) and 20:15 (95% CI 18:19–21:43). The bimodal pattern was preserved in sensitivity analyses restricted to patients with an exact onset time, to patients aged ≥55 years, to non-smokers, and to female patients. Among 79 exploratory across-time-block comparisons, four reached nominal significance (5.1%, consistent with chance alone): active smoking (night 17%, morning 9%, afternoon 30%, evening 14%; p = 0.020), SCAD type 1 (night 17%, morning 3%, afternoon 4%, evening 12%; p = 0.039), fibromuscular dysplasia (FMD) diagnosis among screened patients (p = 0.029), and elevated C-reactive protein at presentation (p = 0.026).
Conclusions: Symptom onset in SCAD shows significant diurnal variation, best described by a bimodal pattern with peaks in the late morning and evening. Associations between onset time and clinical or angiographic characteristics were exploratory and require confirmation.

PREVALENCE AND GENETIC ARCHITECTURE OF PERIPARTUM CARDIOMYOPATHY, SINGLE CENTER STUDY

Andrik M[1], Tománková S[1], Peldová P[2], Kubánek M[1], Adamová M[1], Piherová L[3], Melenovský V[1], Macek Jr. M[2], Krebsová A[1]

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Form: oral presentation, **theme:** cardiomyopathy and cardiac fibrosis
Background/Objectives: Peripartum cardiomyopathy (PPCM) is a heart failure with LV EF <45% occurring during the peripartum period or in the months following delivery, termination or miscarriage. The goal of this study was to evaluate the frequency and genetic architecture of PPCM in our cohort of females with non-ischemic dilated cardiomyopathy (NICM).
Patients and methods: In the last two decades we identified 846 female probands with NICM, out of them 36 met the criteria of PPCM (4.2%). All probands received clinical genetic consultation and molecular genetic analysis using next generation sequencing (targeted/exome sequencing; SOPHiA GENETICS, Switzerland).
Results: ACMG.net class 4 and 5 variants in associated genes were disclosed in 257 out 846 (30%) of female NICM cohort and in 14 out of 36 PPCM individuals (39%). Identified genes in PPCM harbouring the pathogenic mutation encode for TTN (8/14, 57%), TNNT2, RBM20, SCN5A and TRIM63 (1/52, 2% each).
Conclusions: PPCM represents about 4% of the NICM cases in females. The genetic yield in PPCM is significantly higher than in NICM generally. TTN is the most prevalent genetic cause in PPCM as well as in NICM. Our results show the prevalence of PPCM and underscore the necessity of genetic analysis in this disease helping to assess the prognosis and enabling the primary prevention of sudden cardiac death in relatives.

HEALTHCARE COSTS, CARBON EMISSIONS, AND WORKFORCE IMPLICATIONS OF DEVICE-BASED MULTISENSORY MONITORING IN HEART FAILURE

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Form: oral presentation, **theme:** heart failure and cardiovascular imaging
Purpose: Multisensory monitoring using cardiac implantable electronic devices (CIEDs) may improve clinical outcomes in heart failure (HF), but its broader health system impact is uncertain. We evaluated HF-related healthcare costs, carbon emissions and outpatient workload of algorithm-guided monitoring compared with standard care.
Methods: System-level analysis of a propensity score-matched cohort of 191 patient pairs with HF and a CIED, followed for 632 patient-years. Patients managed with HeartLogic or TriageHF within a structured care pathway were compared with standard care. Observed HF-related healthcare utilisation was translated into costs, carbon emissions and activity-based workload estimates for cardiologists, HF nurses, and device technicians, using Dutch reference prices and published emission factors.
Results: CIED-algorithm-guided HF monitoring reduced costs by €496 per patient-year (€860 vs €1,356; 95% CI, –€959 to –€107; p = 0.009) and carbon emissions by 21.81 kg CO_2-equivalent (32.96 vs 54.76 kg; 95% CI, –45.82 to –2.14; p = 0.029), mainly due to fewer hospitalisations and emergency department visits. Cardiologist was lower (rate ratio 0.869; p = 0.043), whereas HF nurse and device technician were higher (rate ratios 2.534 and 1.190; both p ≤0.005). Per 1,000 patients annually, this corresponded to 0.03 fewer cardiologist and 0.20 and 0.08 additional HF nurse, and device technician full-time equivalents.
Conclusion: Algorithm-guided CIED monitoring was associated with lower HF-related healthcare costs and carbon emissions while shifting outpatient care towards nurse-led remote management. These results warrant prospective multicentre confirmation to guide workforce planning and reimbursement.

EARLY HAZARD PHASE IN SPONTANEOUS CORONARY ARTERY DISSECTION: DE NOVO RECURRENCE PATTERNS FROM A PROSPECTIVE REGISTRY

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Form: oral presentation, **theme:** coronary artery disease
Background: Spontaneous coronary artery dissection (SCAD) is a non-atherosclerotic cause of acute coronary syndromes (ACS), predominantly affecting young and middle-aged women. While cumulative recurrence rates of 10–30% at 5 years have been described, the temporal distribution of *de novo* recurrent events remains poorly defined. Whether an early vulnerable phase exists after the index SCAD event is unknown.
Purpose: To investigate the timing and characteristics of *de novo* recurrent SCAD in a prospective registry, with specific focus on early recurrence patterns.
Methods: We established a prospective single-centre registry including patients with angiographically or intravascular imaging-confirmed SCAD. *De novo* recurrence was defined as a new angiographically confirmed dissection in a different coronary territory from the index event. Temporal distribution of events was assessed using Kaplan–Meier analysis.
Results: Twenty-one patients were included (81% women; mean age 47.5 ± 4.9 years). STEMI was the index presentation in 53.8%, and single-vessel involvement occurred in 76.9%, most frequently affecting the LAD (53.8%). PCI was performed in 46.2%, while the remainder were managed conservatively.
During one-year follow-up, *de novo* recurrent SCAD occurred in 5 patients (23.8%). Notably, 4 patients (19%) experienced recurrence within 72 hours of the index event, each involving a different coronary territory and presenting as STEMI. Kaplan–Meier analysis demonstrated an early clustering of events, consistent with a potential early hazard phase following initial SCAD.
Conclusions: In this prospective cohort, we observed a substantial burden of very early *de novo* SCAD recurrence. Although limited by sample size and single-centre design, the temporal clustering of events raises the hypothesis of an early post-index hazard phase.
This concept warrants validation in larger multicentre studies and may have implications for early surveillance intensity and post-index management strategies. Potential mechanisms may include transient systemic arteriopathy, inflammatory activation, or heightened vascular fragility following the initial event.

ROLE OF COMPLETE REVASCULARIZATION IN PATIENTS WITH ACUTE CORONARY SYNDROME AND HISTORY OF CABG: A SINGLE-CENTER REGISTRY ANALYSIS

Bauer D[1], Berka V[1], Fojtík A[1], Mihalovič M[1], Lerner M[1], Trinh MD[1], Rudasevschi D[1], Neuberg M[2], Kočka V[1], Prachtová R[1], Kroupa J[1], Šolc AJ[1], Jelínková L[1], Toušek P[1]

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Form: oral presentation, **theme:** cardiovascular interventions and cardiac arrest
Background: Patients with complex coronary disease treated by coronary artery bypass graft (CABG) are at high risk of recurrent cardiovascular events. Our aim was to investigate contemporary treatment and prognosis of acute coronary syndrome (ACS) in post-CABG patients.
Methodology: We analyzed patients with a history of prior CABG hospitalized for ACS in a single center during a 5-year period (from October 2018 to October 2023). Complete revascularization (CR) was defined as all major epicardial vessels with a diameter ≥2.5 mm without significant lesions (<70%) or revascularized by a functional graft. We correlated the completeness of revascularization with mid-term mortality (mean follow-up of 3.7 [± 2.0] years).
Results: Among 2890 patients with ACS, 205 (7.1%) had a history of prior CABG; 77.6% (n = 159) of men with a mean age of 71.8 (± 9.7) years, mostly with NSTEMI (59.0%, n = 121). The average time from CABG to ACS was 11.3 (± 7.7) years and the mean number of grafts was 2.7 (± 0.9). PCI was performed in 90% (n = 185), re-CABG in 2 cases. In 23.9% (n = 49) CR was achieved, mostly by PCI of the native vessel (81.6%, n = 40). Patients with CR had better mid-term survival compared to incompletely revascularized patients, $p = 0.047$. However, after multivariable adjustment and propensity score matching, CR was not associated with improved survival. There was no survival benefit of either revascularization strategy, PCI of native vessel or bypass graft, respectively.
Conclusion: Complete revascularization in ACS patients with prior CABG was not associated with improved mid-term survival after multivariable adjustment and propensity score matching.

REAL-TIME DETECTION OF RESPIRATORY MOTION ARTIFACTS IN CINE CARDIAC MAGNETIC RESONANCE USING A FRAME-LEVEL CLASSIFIER

Belcak D[1,2], Youngman E[1], Demirel OB[1,3], Perazzolo A[1], Kaya K[1], Ouyang R[1], Chao TC[1], Leiner T[1]

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Form: oral presentation, **theme:** heart failure and cardiovascular imaging

Background: Respiratory motion artifacts can render cine cardiovascular magnetic resonance (CMR) non-diagnostic, yet image quality is often assessed only after acquisition. Automated real-time image-quality assessment (IQA) could trigger immediate review and reacquisition. With clinically labelled respiratory artifact datasets scarce, we trained a binary 2D IQA model using expert-calibrated, physics-based synthetic degradation.
Methods: Artifact-free short-axis cine CMR from ACDC (n = 150), M&Ms (n = 345, multivendor), and an institutional multicenter, multivendor cohort (n = 180) were assembled as 4D volumes (x, y, z, t). Respiratory motion was simulated in pseudo-k-space with a shared trajectory across cardiac phases and body-size-normalized amplitude along the phase-encoding direction. Expert-calibrated artifacts generated ~632,000 labelled 2D frames balanced across non-degraded, moderate, and severe quality. An ImageNet-pretrained ResNet-18 was fine-tuned with patient-level separation; scratch-trained ResNet-18 served as comparator. Frozen weights were evaluated in 170 independent patients (61,108 images; 1,260 series), including 7 manually labelled artifact-positive series (0.56%). Frame probabilities were averaged per series. AUROC, AUPRC, Brier score, and patient-level bootstrap 95% CIs were calculated.
Results: Frame-level AUROC/AUPRC were 0.715/0.021. Series-level AUROC was 0.974 (95% CI 0.940–0.997) and AUPRC 0.153 (95% CI 0.024–0.659; prevalence baseline, 0.006). Scratch-trained ResNet-18 achieved lower discrimination (AUROC 0.943, 95% CI 0.860–0.994; AUPRC 0.147). In exploratory analysis, a threshold optimized for a 10 : 1 false-negative : false-positive cost ratio identified 5/7 artifact-positive series with 97.4% specificity, referring 2.9% of series for review; capturing 7/7 required reviewing 7.0%. Prior correction improved calibration, reducing the Brier score from 0.0073 to 0.0056. Mean end-to-end IQA time was 1.32 s per series (median, 0.72 s), with inference alone requiring 68 ms.
Conclusion: A 2D convolutional network trained on expert-calibrated synthetic respiratory artifacts generalized to clinical cine CMR, achieving strong series-level discrimination while flagging few series for review. These findings support synthetic artifact generation for real-time CMR quality control and reacquisition. Larger prospective studies are needed to confirm performance and extend the approach to other artifact types.

THE ROLE OF FAMILY HISTORY IN CLINICAL PROFILE OF ACUTE CORONARY SYNDROME IN YOUNG PATIENTS: A SINGLE-CENTRE ANALYSIS

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Form: poster
Introduction: The incidence of acute coronary syndrome (ACS) in patients under 50 years of age is increasing. The knowledge of influence of family history on clinical course, extent of coronary disease and procedural characteristics in young patients is, however, limited.
Methods: Analysis of single-center data of all ACS patients over a five-year period (from October 2018 to October 2023) was performed. Young patients ≤50 years of age were selected and divided into two groups – with positive (Group A) and negative (Group B) family history of CAD (CAD in a male/female relative before 55/65 years, respectively). Data were summarized using descriptive statistics. Mann–Whitney U test and chi-square test were used for quantitative and qualitative analyses, respectively. Mortality data was visualized using a Kaplan–Meier curve.
Results: Of the 3127 patients admitted with ACS, 283 (9.1%) were ≤50 years old, out of which 82.7% were men, with mean age of 45.1 years (± 4.2). There were 91 patients in Group A and 192 in Group B. History of hypertension was more often in Group A (53.8%, n = 49) vs. Group B (39.6%, n = 76), $p = 0.02$. Patients in Group A presented more often with three-vessel disease (31.9%, n = 29) compared to Group B (18.8%, n = 36), $p = 0.05$, and more often as NSTEMI (36.3%, n = 33 vs. 22.9%, n = 44, respectively), $p = 0.05$. There were no significant differences in the metabolic profile: weight (94.3 kg [± 18.25]) vs. 92.6 kg [± 16.8], $p = 0.40$), fasting glycaemia (6.4 mmol/l [± 2.2] vs. 6.3 mmol/l [± 1.8], $p = 0.90$), total cholesterol (4.8 mmol/l [± 1.3] vs. 5.3mmol/l [± 4.0], $p = 0.82$), and LDL cholesterol (3.0 mmol/l [± 1.2] vs. 3.2 mmol/ [± 1.8], $p = 0.68$) were similar between Group A and Group B, respectively. Most patients were treated by PCI (82.4% in Group A and 85.9% in Group B, $p = 0.41$) with similar total stent length (32.2 mm [± 22.2] vs. 28.8 mm [± 17.9], $p = 0.29$). There were no significant differences in procedural time: 43.9 min (± 28.0) vs. 39.6 min (± 19.3), $p = 0.67$. The overall survival rate over a mean follow-up period of 4.5 years (± 1.4) was 96.7% (Group A) vs. 93.7% (Group B), $p = 0.30$.
Conclusions: Young patients with ACS (≤50 years) with a positive family history of CAD present more often with NSTEMI with the finding of 3 vessel disease. However, clinical profile and procedural characteristics are similar compared to patients without family history of CAD. Young patients with ACS have excellent mid-term outcomes regardless of family history of CAD.

CELL THERAPY FOR HEART FAILURE

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Form: oral presentation, **theme:** cell therapy, myocarditis, amyloidosis
Two decades and >200 trials have shown that adult stem/progenitor cells are safe, and several Phase I/II studies suggest that they are efficacious in heart failure and refractory angina, although large Phase III trials are needed to establish efficacy conclusively.
At the preclinical level, there is overwhelming evidence that various stem/progenitor cells can improve cardiac function after myocardial infarction. The mechanism re-

mains unclear but it does not involve regeneration of new myocytes because all stem/progenitor cells fail to engraft in the heart and disappear quickly after transplantation. Therefore, transplanted cells must work via secretion of factors that act in a paracrine fashion. The use of repeated dosing is particularly important because mounting evidence shows that the efficacy of cell therapy is underestimated if only one dose is used. In the clinical arena, it seems unlikely that cell therapy will prove beneficial in patients with STEMI; instead, heart failure and refractory angina appear to be the main target populations. The use of the intravenous route for cell delivery may fundamentally transform the field of cell therapy.
We are at a pivotal stage in the field in which information from many relatively small clinical trials must guide carefully executed efficacy trials. This lecture critically examines the current state of clinical research on cell-based therapies for cardiovascular disease, highlighting the controversies in the field, improvements in clinical trial design, and the application of exciting new cell products.

TRANSCATHETER AORTIC VALVE IMPLANTATION VS. SURGERY FOR FAILED BIOPROSTHESIS: A META-ANALYSIS OF OVER 20 000 PATIENTS

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Form: oral presentation, **theme:** valvular heart disease
Introduction: Valve-in-valve transcatheter aortic valve implantation (ViV-TAVI) has gained popularity as a less invasive alternative to a redo surgical aortic valve replacement (redo-SAVR); which one is the preferred technique in these cases, however, remains a topic of debate, as the available data refer to retrospective studies with few patients or limited follow-up. The present meta-analysis aimed to compare the short-term and long-term outcomes of the two techniques in the setting of a failed surgical bioprosthesis.
Methodology: PubMed, MEDLINE, and Embase were searched on 10 November 2023 yielding 355 results (PROSPERO ID: CRD42023490612), of which 27 were suitable for meta-analysis. The primary outcomes were short-term and long-term all-causes and cardiovascular mortality. Logarithmic risk ratio (Log RR) and mean difference were used for categorical and continuous data, respectively.
Results: Both redo-SAVR and ViV-TAVI exhibited similar procedural and short-term mortality. However, ViV-TAVI demonstrated lower 1-year mortality (RR: 0.74, 95% confidence interval [CI] 0.57–0.96, p = 0.02), acute kidney injury (RR: 0.47, p <0.001), bleeding (RR: 0.44, p <0.001), stroke (RR: 0.70, p <0.05), and new pacemaker implantation (RR: 0.69, p <0.05). Conversely, redo-SAVR demonstrated more favorable mean postoperative aortic valve gradients (mean difference 2.59, 95% CI 0.86–4.31, p <0.01).
Conclusion: Short-term mortality was similar between the groups, but ViV-TAVI showed better survival at 1 year as well as reduced rates of acute kidney injury, bleeding, stroke, and pacemaker implantation. However, redo-SAVR leads to a better hemodynamic profile. Even if collected data come from retrospective studies, the present results could help to guide the choice of the best approach case-by-case according to the patient's clinical profile.

NOVEL THERAPEUTIC OPTIONS FOR CARDIAC FIBROSIS

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Form: oral presentation**, theme:** cardiomyopathy and cardiac fibrosis
In response to stress or damage, the heart undergoes extensive remodeling, including hypertrophy and fibrosis. Cardiac fibrosis significantly increases the risk of morbidity and mortality, yet lacks effective treatment options despite significant effort to address this unmet clinical need. New therapies are urgently needed, and we have identified three promising approaches to fibrosis mitigation.
We have shown that scleraxis is a critical driver of cardiac fibroblast activation to myofibroblasts, and our recent data has suggested that scleraxis also enables myofibroblasts to escape apoptosis. We developed a dominant negative form of scleraxis, showing that it attenuates fibroblast activation, and found that scleraxis knockdown by shRNA was similarly effective. Scleraxis also appears to be constitutively phosphorylated by casein kinase II, and pharmacologic inhibition of this phosphorylation with the drug TBB similarly impairs scleraxis function.
We reported that glutaminolysis in an important source of energy for cardiac fibroblast activation. GLS1 is the rate-limiting enzyme of glutaminolysis, and we have shown that a GLS1 inhibitor called CB-839, currently under investigation as an anti-cancer agent, attenuates fibroblast activation. We further showed that *GLS1* gene expression is directly regulated by scleraxis, which binds to the GLS1 promoter and which may also activate the expression of other glutaminolysis pathway genes. Scleraxis was also required for pro-fibrotic TGFα-mediated induction of GLS1 expression.
Finally, our recent data indicates that angiotensin II (AngII) induces scleraxis expression via a mechanism dependent on Serum Response Factor and Myocardin-Related Transcription Factor A. AngII-induced collagen gene expression was dependent on scleraxis. *In vivo*, the AngII receptor blocker losartan attenuated cardiac fibrosis, whether caused by AngII infusion or by pressure overload via thoracic aortic constriction. These results suggest that losartan may be an effective anti-fibrotic even in situations where systemic AngII is not elevated.

Together, these pre-clinical findings provide new directions for anti-fibrotic intervention, including redirecting existing drugs (TBB, CB-839, losartan) or targeting scleraxis directly. Given the urgency of the need for anti-fibrotic therapies, this work should be quickly pursued.

MITRAL VALVE REPAIR IN ATRIAL FUNCTIONAL MITRAL REGURGITATION VERSUS DEGENERATIVE ETIOLOGY: A 10-YEAR TIME-TO-EVENT DATA RECONSTRUCTION OF OVER 18,000 PATIENTS

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Form: oral presentation, **theme:** valvular heart disease
Atrial functional mitral regurgitation (AFMR) is a distinct entity driven by left atrial enlargement and mitral annular dilation, typically associated with atrial fibrillation, rather than intrinsic leaflet disease. The long-term safety and durability of mitral valve repair in AFMR remain inadequately defined compared with degenerative MR. We aimed to assess long-term survival, MR recurrence, heart failure, and stroke after repair.
Methodology: An advanced search of PubMed, Embase, and MEDLINE was performed on 1 June 2025, identifying 554 records, of which 43 studies met the inclusion criteria. Studies evaluating mitral valve repair in AFMR or degenerative MR and reporting relevant clinical outcomes were included. Two reviewers independently performed two rounds of blinded screening, with disagreements resolved by a third reviewer. Time-to-event data were reconstructed using RStudio and SPSS.
Results: A total of 18,056 patients were included: 939 with AFMR and 17,117 with degenerative MR. AFMR patients were older, more frequently female, and had higher rates of diabetes and atrial fibrillation; Maze ablation was also more frequent. At 10 years, AFMR was associated with lower post-repair survival than degenerative MR (HR 1.381, 95% CI 1.15–1.66; p <0.001) and significantly higher MR recurrence (HR 2.32, 95% CI 1.65–3.26; p <0.001). Freedom from heart failure and stroke also differed significantly between groups (p = 0.03 and p <0.001, respectively). In the AF-only subgroup, survival was comparable between AFMR and degenerative MR (p = 0.35).
Conclusions: Mitral valve repair for AFMR appears relatively safe compared with degenerative MR in patients with comparable risk profiles. The worse long-term outcomes observed in the overall AFMR cohort may be partly explained by the higher prevalence of atrial fibrillation and differences in its management. Importantly, survival was comparable in the AF-only subgroup, while the excess heart failure observed in the overall cohort was no longer evident. These findings support mitral valve repair as a reasonable surgical strategy for AFMR, while highlighting the importance of atrial fibrillation management and long-term surveillance for recurrent MR.

BASELINE SDNN IS ASSOCIATED WITH CARDIORENAL DYSFUNCTION IN PATIENTS WITH STAGE III–IV CHRONIC KIDNEY DISEASE

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Form: oral presentation, **theme:** heart failure and cardiovascular imaging
Introduction: Heart rate variability (HRV), particularly the Standard Deviation of Normal-to-Normal R–R intervals (SDNN), may reflect cardiorenal dysfunction. We assessed SDNN, its cardiorenal associations, and changes during 6-months guideline-based therapy in patients with stage III–IV chronic kidney disease (CKD).
Methodology: We studied 117 patients (77 women; mean age 52.27 ± 10.66 years) with grade 1–2 essential hypertension and stage III–IV CKD, with or without diabetes mellitus (DM), and 20 healthy controls. Four gr were defined: gr1: CKD III without DM (n = 31); gr2: CKD III with DM (n = 33); gr3: CKD IV without DM (n = 28); gr4: CKD IV with DM (n = 25). Mean age and sex distribution were comparable across gr (p >0.05). Baseline and 6-month assessments included creatinine, cystatin C, estimated glomerular filtration rate (eGFR), glycated hemoglobin (HbA_{1c}), N-terminal pro-B-type natriuretic peptide (NT-proBNP), echocardiography, and 24-h Holter ECG monitoring, with SDNN as the primary HRV parameter. Statistical analysis included Mann–Whitney U test and Bonferroni adjustment for multiple comparisons; categorical variables were analyzed using Fisher's exact or χ^2-test. Continuous data are presented as mean ± SD; p <0.05 was considered significant.
Results: Baseline SDNN were 86.2 ± 7.2, 68.2 ± 10.2, 74.6 ± 8.1, and 54.7 ± 9.1ms in gr 1–4, respect actively lower (p <0.05), than in controls (138.2 ± 6.2 ms) with lower values in DM vs non-DM patients at both CKD stages (p <0.05). SDNN <70 ms was associated with lower eGFR (32.8 ± 2.8 vs 41.6 ± 3.9 ml/min/l,73 m²), higher cystatin C (2.11 ± 0.3 vs 1.59 ± 0.2 mg/L) and NT-proBNP (1649 ± 127 vs 1249 ± 108 pg/mL), and lower LVEF (37.5 ± 4.2 vs. 42.7 ± 5.4%; all p <0.05). After 6 months, SDNN increased significantly in CKD stage III (+24 ms in gr1; +9 ms in gr2) and stage IV without DM (+23 ms), but nonsignificant in stage IV with DM (+7 ms).
Conclusion: SDNN declined with CKD severity and was lower with DM. Baseline SDNN <70 ms was associated with worse renal and cardiac function. SDNN increased after 6 months therapy, with significant changes in CKD stage III and in stage IV without DM, while no significant change was observed in stage IV with DM. Prospective studies are required to validate the SDNN threshold and its clinical utility.

ADDITIVE VALUE OF LEFT ATRIAL RESERVOIR STRAIN AND LEFT VENTRICULAR GLOBAL LONGITUDINAL STRAIN BEYOND THE HCM RISK-SCD SCORE IN PATIENTS WITH LOW-TO-INTERMEDIATE RISK HYPERTROPHIC CARDIOMYOPATHY

Dinc Asarcikli L[1], Leanens D[2], Elmasry N[1], Santi PL[1], Caunite L[1], Myagmardorj R[1], Zegkos T[3], Kamperidis V[3], Ziakas A[3], Parcharidou D[3], Li T[4], Fai WKK[4], Sia CH[4], van Rosendael PJ[1], Bootsma M[1], Bax JJ[1,5], Marsan NA[1]

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Form: oral presentation, **theme:** cardiomyopathy and cardiac fibrosis
Background, aims: Sudden cardiac death (SCD) risk stratification remains challenging in hypertrophic cardiomyopathy (HCM) patients classified as low-to-intermediate risk. Current guideline-recommended the HCM Risk-SCD score incorporates left atrial (LA) diameter and do not capture LA myocardial dysfunction, which can precede chamber enlargement. Additionally, it doesn't include LA or left ventricular (LV) functional measures which are increasingly recognized as sensitive markers of subclinical myocardial impairment in HCM. We assessed whether LA reservoir strain (LARS) and LV global longitudinal strain (LVGLS) provide incremental prognostic value for arrhythmic events beyond this score.
Methods: In this multicenter cohort of HCM patients with low-to-intermediate SCD risk (HCM Risk-SCD <6%), we excluded those with prior aborted SCD or sustained ventricular tachycardia/fibrillation. The primary endpoint was a composite of SCD, aborted SCD, and appropriate implantable cardioverter-defibrillator (ICD) therapy. Three nested Cox models sequentially added LARS (≤23%) and LVGLS (≤14%) to the HCM Risk-SCD score. Discrimination and model fit were assessed with C-index, time-dependent AUC, and the likelihood ratio test; net reclassification improvement was evaluated as a complementary measure.
Results: Among 989 patients (mean age 55 ± 15 years; 55% male), 71 (7.2%) experienced SCD-related events: 15 (1.5%) SCD, 9 (0.9%) aborted SCD, and 47 (4.8%) appropriate ICD therapies. Impaired LARS (HR 1.81, 95% CI 1.06–3.09, p = 0.031) and impaired LVGLS (HR 1.89, 95% CI 1.12–3.19, p = 0.017) were each independently associated with SCD-related events after adjustment for the HCM Risk-SCD score. Sequential addition of both strain parameters significantly improved model performance. Discrimination improved progressively across the models: Model 1 (HCM Risk-SCD score alone) C-index 0.723, Model 2 (+LARS) 0.734, and Model 3 (+LARS and LVGLS) 0.749. Adding LARS to the SCD risk score significantly improved model fit (LRT $\Delta\chi^2$ = 11.0 vs. Model 1 [SCD risk score], p <0.001), and the full model incorporating both LA & LV strain parameters showed further improvement ($\Delta\chi^2$ = 16.3 vs. Model 1, p <0.001) with a net reclassification improvement of 57 % versus baseline model providing supportive evidence of benefit.
Conclusion: Impaired LARS and LVGLS incrementally predict SCD-related events beyond the HCM Risk-SCD score in patients with a low-to-intermediate risk.

CIRCULATING PLASMA MIRNA DIFFERENCES BETWEEN PATIENTS WITH AND WITHOUT FAMILIAL HYPERCHOLESTEROLEMIA DURING STATIN THERAPY: A PILOT STUDY

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Form: poster
Background: Statins have been shown to modulate the expression of several circulating microRNAs (miRNAs) involved in cholesterol homeostasis, inflammation, endothelial function, and vascular remodeling. We investigated whether statin therapy differentially affects circulating miRNA profiles in patients with heterozygous familial hypercholesterolemia (HeFH) and those without FH (non-FH).
Methods: A total of 28 patients (mean age, 55.0 ± 12.9 years; 5 with HeFH) were included in the study. Blood samples were collected at baseline, before initiating statin treatment following a two-month washout period (n = 28), and after 1 month (n = 28) and 12 months (n = 11) of statin therapy. Twenty-three candidate miRNAs were quantified using the QuantStudio 6 Flex Real-Time PCR System.
Results: At baseline, six miRNAs differed between HeFH and non-FH patients. Throughout the follow-up period, let-7d-5p and miR-484 levels were higher (p = 0.01), whereas miR-505-3p levels remained lower in HeFH (p = 0.05). The fold change in let-7d-5p was positively associated with changes in LDL-C after one month of statin therapy (ρ = 0.535, p = 0.03), whereas the fold change

in miR-505-3p was negatively associated with changes in HDL-C ($\rho = -0.503$, $p = 0.04$) in non-FH patients. However, none of these findings remained significant after multiple-testing correction.
Conclusions: FH status may influence circulating miRNA profiles during statin therapy, particularly the levels of let-7d-5p, miR-484 and miR-505-3p. Larger studies are needed to confirm these findings and clarify their potential relationship with lipid metabolism.

SECRETONEURIN DYNAMICS AND THREE-YEAR PROGNOSIS IN PATIENTS WITH HEART FAILURE: A PROSPECTIVE OBSERVATIONAL STUDY

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Form: poster
Background: Despite the use of established biomarkers such as NT-proBNP, prognostic stratification in patients with heart failure (HF) remains suboptimal. Secretoneurin (SCN) is an emerging biomarker reflecting neuroendocrine activation and disturbances in intracellular calcium handling. While elevated absolute SCN levels have been associated with adverse outcomes, the prognostic significance of longitudinal changes in SCN and their relationship to long-term survival have not yet been sufficiently explored.
Methods: We analysed a prospective cohort of 304 patients with chronic HF followed at a specialised tertiary centre. Patients underwent up to four scheduled visits (baseline, 3, 6 and 9 months) with repeated measurements of SCN, NT-proBNP and high-sensitivity troponin. For analyses of biomarker dynamics, patients with at least two consecutive measurements were included. SCN dynamics were defined as an increase, stability or decrease between baseline and follow-up assessment. The primary endpoint was all-cause mortality at 3 years. Surviving patients were treated as right-censored. Prognostic associations were assessed using Kaplan–Meier survival analysis and Cox proportional hazards models.
Results: The analysed cohort comprised 304 patients (median age 69 years, 83% men), with a median left ventricular ejection fraction of 30% and a median baseline NT-proBNP concentration of 1,240 ng/L, reflecting a population with advanced chronic HF.
Patients exhibiting an increase in SCN levels during follow-up had a markedly worse three-year prognosis compared with those with stable or decreasing SCN values. This association persisted despite the observation that a proportion of patients with elevated absolute SCN levels remained long-term survivors. Changes in SCN over time more accurately reflected time-dependent mortality risk than single baseline measurements. NT-proBNP and high-sensitivity troponin demonstrated similar, albeit less pronounced, relationships with long-term survival.
Conclusions: Longitudinal changes in SCN represent a significant prognostic marker of three-year survival in patients with HF. While individual SCN measurements are not deterministic predictors of death, an increase in SCN over time identifies patients at increased long-term mortality risk. These findings support the clinical value of repeated SCN assessment to refine risk stratification beyond single biomarker measurements.

A DYNAMIC MULTIMARKER WORSENING SCORE BASED ON SECRETONEURIN, NT-PROBNP AND HIGH-SENSITIVITY TROPONIN IMPROVES LONG-TERM RISK STRATIFICATION IN CHRONIC HEART FAILURE

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Form: poster
Background: Single-biomarker risk stratification in chronic heart failure (HF) may fail to capture the multidimensional pathophysiology of disease progression. A dynamic multimarker approach integrating neuroendocrine stress (secretoneurin, SCN), haemodynamic load (NT-proBNP) and ongoing myocardial injury (high-sensitivity troponin, hs-Tn) may provide incremental prognostic information.
Methods: We analysed a prospective cohort of 304 patients with chronic HF followed at a specialised tertiary centre. Patients were predominantly male (≈83%), with a median age of 69 years (IQR ~60–75), median body mass index ~29 kg/m^2 (overweight range), and median left ventricular ejection fraction ~30%, consistent with a predominantly HFrEF population. Median baseline NT-proBNP was approximately 1,200 ng/L, indicating advanced disease severity.
Biomarkers (SCN, NT-proBNP and hs-Tn) were measured at baseline and during scheduled follow-up visits (3, 6 and 9 months, depending on availability). For each biomarker, dynamic change was defined as a ≥20% increase from baseline to the last available follow-up measurement. A dynamic multimarker worsening score (0–3) was calculated as the number of biomarkers showing worsening. The primary endpoint was all-cause mortality; surviv-

ing patients were treated as right-censored. Survival was assessed using Kaplan–Meier analysis.
Results: Among patients with available follow-up time and at least two biomarkers evaluable for dynamic assessment, the distribution of the dynamic multimarker worsening score was: 0 (n = 73), 1 (n = 92), 2 (n = 32) and 3 (n = 6). Observed mortality increased stepwise with higher worsening scores, with the highest risk observed in patients with a score of 3. Survival curves demonstrated a clear gradient of risk across score categories, supporting the additive prognostic value of concurrent worsening across multiple biomarker pathways.
Conclusions: A dynamic multimarker worsening score integrating SCN, NT-proBNP, and hs-Tn identifies patients with progressively higher mortality risk in chronic HF. This approach highlights the value of repeated biomarker assessment and multimarker integration for refined long-term risk stratification beyond single-marker evaluation.

ENHANCING CARDIAC PHOSPHODIESTERASE ACTIVITY: A THERAPEUTIC STRATEGY IN HEART FAILURE?

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Form: oral presentation, **theme:** heart failure and cardio-oncology
The β-adrenergic stimulation of cardiac function involves the second messenger cAMP which activates the cAMP-dependent protein kinase (PKA) to modulate key proteins of the excitation-contraction coupling (ECC) process. Intracellular cAMP levels produced upon β-stimulation are counterbalanced by the degradation of the cyclic nucleotide by enzymes called phosphodiesterases (PDEs). We have shown in the past that PDEs not only terminate the activation of cAMP effectors but also compartmentalize this second messenger in discrete subcellular domains. Clinically, PDE inhibition has been considered a promising approach to compensate for the catecholamine desensitization that accompanies heart failure (HF). In that respect, PDE3 inhibitors, such as milrinone or enoximone, have been used clinically to improve systolic function and alleviate the symptoms of acute HF. However, their chronic use has proven to be detrimental, increasing adverse remodeling and ventricular arrhythmias.
Recently, we proposed to test the opposite strategy, i.e. increasing rather than inhibiting PDE activity. We believe that this strategy, which is reminiscent of the counter-intuitive beneficial effect of beta-blockers in HF, could be therapeutically relevant in HF because it would prevent a deleterious accumulation of cAMP during catecholamines spill over. In that line, we found that constitutive overexpression of PDE4B, one of the main PDE4 isoform expressed in the cardiomyocyte to control the β-adrenergic regulation of the ECC, is cardioprotective. We also found that gene therapy with AAV9-PDE4B exerts cardioprotective effects limiting adverse remodeling evoked by catecholamines or increased postcharge. Similarly, we found that PDE2A constitutive overexpression exerts anti-hypertrophic effects and transgenic mice overexpressing PDE2A in the heart have preserved ejection fraction after myocardial infarction and are protected against catecholamine induced ventricular arrhythmia. Also, *PDE2* gene therapy limits cardiac adverse left ventricle remodeling and dysfunction induced by sympathomimetic amines as well as ventricular arrhythmias.
Altogether, our findings suggest that increasing PDE activity, either with gene therapy or pharmacologically with PDE-specific activators yet to be discovered, could represent an interesting novel therapeutic strategy to treat heart failure.

IMPORTANCE OF FAMILY CASCADE SCREENING AND GENETICS IN AORTIC OR CORONARY DISSECTIONS, SINGLE-CENTER STUDY

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Form: oral presentation, **theme:** preventive cardiology and vascular disease
Background: Aneurysm and dissection of great arteries may be a sign of inherited aortic diseases in individuals < 60 years of age. Our aim was to assess the yield of genetic and clinical family cascade screening in this condition.
Patients and methods: We studied 136 (33 post mortem, 103 survivors) cases (83 males, 53 females, average age 52,5 years), 117 suffering from type A–, 7 type B and 12 coronary artery dissections. All probands/relatives received clinical genetic consultation and molecular genetic analysis. Family cascade screening was carried out in 229 relatives (1.7 per case).
Results: ACMG.net class 4 and 5 variants in definitely associated genes were disclosed in 18 (13%) in genes *FBN1*(6/136) and *COL3A1* (5/136), *TGFBR1* (4/136), *MYH11* (2/136) and in TGFBR2 (1/136). Further 9 LP/P DNA variants were identified in potentially associated genes (*COL4A3*, *COL4A5*, *GATA4*, *KRIT1*, *MYH7*, *AEBP1*, *FKBP14*, *TNXB*). Family cascade screening identified 14 G+ individuals, while clinical screening showed up to 67 relatives of G- individuals with signs of inherited aortic syndrome.
Conclusion: The genetic yield in dissections is low (13%). While the cardiological family cascade screening identi-

fies a high number of relatives at risk (37%). This may lead to primary prevention of acute dissection by control of blood pressure, lifestyle changes or preventive surgical treatment.

ENDOTYPE-GUIDED CORONARY FUNCTIONAL TESTING IMPROVES ANGINA IN ANOCA: FIRST REAL-WORLD CZECH EXPERIENCE

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Form: oral presentation, **theme:** coronary artery disease
Background: Angina with non-obstructive coronary arteries (ANOCA) affects up to 70% of patients undergoing invasive coronary angiography and significantly impairs quality of life. Despite evidence from randomized trials supporting endotype-guided therapy, coronary functional testing (CFT) remains underutilized in routine practice in central Europe. We report the first routine implementation of comprehensive CFT in the Czech Republic.
Purpose: To evaluate the feasibility, diagnostic yield, and symptomatic impact of routine comprehensive CFT followed by endotype-guided therapy in patients with ANOCA.
Methods: In this prospective single-centre observational study at Motol University Hospital (Czech Republic), consecutive ANOCA patients underwent invasive CFT, including thermodilution-based assessment of coronary flow reserve and microvascular resistance, followed by intracoronary acetylcholine testing. Patients were classified as coronary microvascular dysfunction (CMD), microvascular spasm, vasospastic angina (VSA), or combined endotypes and received targeted pharmacotherapy. Angina burden was assessed using the Seattle Angina Questionnaire (SAQ) at baseline and 2-month follow-up. The study was approved by the institutional Ethics Committee (EK-151.23/25); all patients provided written informed consent. The study was prospectively registered at ClinicalTrials.gov (NCT07103317).
Results: Between April 2023 and January 2026, 55 consecutive ANOCA patients were enrolled (n = 37, 67% women; median age 63 years [22–81]). CFT was positive in 41 patients (75%). CMD was detected in 13 (23.6%), and acetylcholine-provoked spasm in 39 (70.9%). The predominant endotype was VSA (n = 21, 38.2%), followed by microvascular spasm (n = 7, 12.7%) and isolated CMD (n = 2, 3.6%); 11 patients (20%) had combined endotypes.
At 2 months, the mean SAQ score increased from 71.2 to 81.3 (+14.2%, $p = 0.0019$).
Conclusion: Routine implementation of comprehensive CFT in ANOCA demonstrates a high diagnostic yield and is associated with clinically meaningful symptomatic improvement following endotype-guided therapy, consistent with previously published randomized controlled trials. These real-world data support the systematic incorporation of coronary functional assessment into routine clinical pathways.

MYSTERIES AND OPPORTUNITIES TO CHALLENGE THE ENDEMIC OF HEART FAILURE WITH PRESERVED EJECTION FRACTION (HFPEF) IN SLAVIC COUNTRIES

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Form: oral presentation, **theme:** heart failure and cardio-oncology
There has been a significant increase in the number of cases of heart failure with preserved ejection fraction (HFpEF), especially in the South and West Slavic Nations after the outbreak of COVID-19. During COVID-19 and after COVID-19 (2020–2024), the mortality per million population was reported to be relatively higher in most Slavic Nations in comparison to other nations.
Recently, genetic polymorphisms with respect to the renin-angiotensin system have also been reported in these countries, in addition to specific genomic mutations related to cardiomyopathy, stroke, and vascular risk factors. The classical treatment, such as the use of angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) failed in patients with HFpEF. ACE-2 has emerged as a regulatory target in RAS. There is a shift in understanding of the RAS to check the maladaptive excesses of angiotensin II. ACE2 is considered as a target to explore in the therapeutic landscape for HFpEF and other complications.
In the context of HFpEF, ACE2 expression, which is localised to cardiomyocytes, cardiac fibroblasts, epicardial adipose tissue and the coronary vascular endothelium, may get deregulated, causing fibrosis. Further, ACE2 activity declines, leaving the heart vulnerable to unchecked Ang-II toxicity. With this evidence, therapeutic strategies have evolved in three categories: recombinant proteins, gene therapy and small molecule modulators. Recombinant ACE2 (GSK2586881 and NCT 008 8635) has undergone critical trials to boost ACE2/Ang (1–7) axis for HFpEF. Similarly, AB-1002, RT-100 and RT-200 are in different stages of development as HF therapy. Further, small drug molecules are being developed to activate/modulate ACE2, as Angiotensin (1–7) analogues and MAS receptor agonists. We have developed a herbal formulation, Coroquil Zn, which has shown promising results as an ACE2 modulator and is expected to provide a lead for various post COVID-19 or long COVID-19 complications.

INOTROPIC EFFECTS OF 3-OH-HYDROXYBUTYRATE IN A RODENT HEART: PRESSURE-VOLUME AND ISOLATED RAT VENTRICULAR TRABECULAE ANALYSIS

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Form: oral presentation, **theme:** heart failure and cardiovascular imaging
Introduction: Ketone body 3-hydroxybutyrate (3-OHB) is produced in the liver from fatty acids during periods of reduced carbohydrate availability such as fasting, prolonged exercise, or caloric restriction. An administration of 3-OHB in experimental animals or humans is associated with an increase of cardiac output, but whether this hemodynamic benefit reflects direct myocardial inotropy or afterload reduction mediated by peripheral vasodilation remains unclear. It is also unknown whether these myocardial effects are driven by receptor-mediated inotropy or the modulation of myocardial bioenergetics.
Methodology: To address these questions, we studied concerted cardiovascular response (pressure-volume analysis of the left ventricle) and isolated left ventricular trabeculae from normotensive male HanSD rats (age 20–30 weeks) after administration of 3-OHB (1 mmol/l) and drugs targeting adrenergic and cAMP-dependent signaling – metoprolol, adenylyl cyclase inhibitor 2,5-dideoxyadenosine and isoprenaline, during Tyrode solution perfusion and 1 Hz pacing. We also studied group of animals (20–30 weeks old) with heart failure (HF) induced by permanent coronary ligation of LAD (n = 8, HF). The ligation was performed at the age of 13 weeks, and echocardiography was performed to confirm reduced ejection fraction in the HF group 7 weeks after the procedure. Due to limited number of post myocardial infarction animals, only responses to 3-OHB were studied.
Results: Pressure-volume analysis demonstrated increased left ventricular end-systolic elastance (+34%), improved ventriculo-vascular coupling (+17%) and increased LV efficiency (+28%), indicating direct inotropic effect of 3-OHB. In isolated LV trabeculae, baseline and 3-OHB-stimulated twitch force were significantly greater in healthy hearts (+55.8%) than in the HF group (+35.6%); $p = 0.014$. The inotropic effect of 3-OHB was not further augmented by coadministration of isoprenaline and was not attenuated to B1 adrenergic blockade or cAMP inhibition.
Conclusion: Results suggest that 3-OHB directly improves LV inotropy and by a mechanism that is independent of receptor-mediated signaling, most consistent with a direct bioenergetic pathway. These findings support 3-OHB as a candidate inotropic agent with a mechanistic profile distinct from conventional cAMP-dependent inotropes.

ELEVATED LP(A) IS ASSOCIATED WITH SMALLER AORTIC ANNULUS SIZE AND GREATER AORTIC REGURGITATION IN PATIENTS WITH SEVERE AORTIC STENOSIS

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Form: oral presentation, **theme:** valvular heart disease
Introduction: Elevated lipoprotein(a) [Lp(a)] levels have been associated with the development and more rapid hemodynamic progression of calcific aortic stenosis. We aimed to further characterize the phenotype of patients with severe aortic stenosis according to Lp(a) levels.
Methods: A total of 169 patients (56% men; mean age 78.8 ± 5.8 years) with severe aortic stenosis scheduled for transcatheter aortic valve implantation (TAVI) were included. Patients were divided into two groups according to Lp(a) concentration: Lp(a) <300 mg/L (n = 131) and Lp(a) ≥300 mg/L (n = 38). Clinical characteristics, echocardiographic findings, and CT parameters, including aortic valve calcium score, were compared between the groups.
Results: The groups were comparable with respect to age, sex, comorbidities, and aortic valve calcium score. Coronary artery disease was numerically more frequent in patients with Lp(a) ≥300 mg/L (48.8% vs. 36.8%, $p = 0.20$). Patients with elevated Lp(a) had a smaller aortic annulus diameter (24.41 ± 2.35 vs. 25.37 ± 2.55 mm, $p = 0.031$) and greater severity of aortic regurgitation (1.7 ± 1.2 vs. 1.1 ± 1.1, $p = 0.004$). The associations of elevated Lp(a) with aortic annulus diameter and aortic regurgitation remained significant after adjustment for age, sex, body surface area, and aortic valve morphology.
Conclusion: In patients with severe aortic stenosis undergoing TAVI, elevated Lp(a) was associated with a smaller aortic annulus and greater severity of aortic regurgitation, independently of age, sex, body surface area, and aortic valve morphology. In contrast, Lp(a) levels were not associated with the extent of aortic valve calcification. These findings suggest that elevated Lp(a) may be associated with a distinct anatomical and hemodynamic phenotype of severe aortic stenosis.

SELF-REPORTED STATIN-ASSOCIATED MUSCLE SYMPTOMS ARE LINKED TO LOWER MAXIMAL AEROBIC CAPACITY

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Form: oral presentation, **theme:** preventive cardiology and vascular disease
Introduction: Statin-associated muscle symptoms (SAMS) affect approximately 10% of patients receiving statin

therapy. SAMS can significantly impact the quality of life and one's ability to care for oneself. We have studied potential differences in physical activity parameters in subjects treated with high statin doses.
Methodology: This study examined 19 adult patients (16 males and 3 females, aged 53.9 ± 11.7 years), who were non-diabetic and had experienced their first myocardial infarction (MI). None had received statins prior to the index event. All were subsequently initiated on guideline--recommended high-intensity statin therapy (atorvastatin 80 mg daily). Participants underwent a six-minute walk test (6MWT) and bicycle ergometry at baseline and at 3- and 6-month follow-up. Eight patients reported SAMS during the study period.
Results: As expected, intensive statin therapy resulted in significant improvements in lipid parameters across the cohort (all p <0.05). At baseline and after 3 months, no significant differences were observed between patients with and without SAMS in 6MWT performance (distance and steps), basal metabolic rate, metabolic turnover, or fat mass. However, after 6 months, patients without SAMS demonstrated significantly higher fat-free mass (p <0.05) and maximal oxygen uptake (VO_{2max}; p <0.05) compared with those reporting SAMS. The difference for VO_{2max} remained significant after adjusting for sex.
Conclusion: Our findings suggest that self-reported SAMS although this has not been proven clinically may be associated with reduced physical performance in patients receiving high-intensity statin therapy.

EFFECTS OF ANTIARRHYTHMICS ON CARDIAC PHENOTYPE AND REDOX BALANCE IN SPONTANEOUSLY HYPERTENSIVE RATS

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Form: oral presentation, **theme:** arrhythmia and electrophysiology I
Arterial hypertension is a major risk factor for the development of cardiac arrhythmias, and some antihypertensive therapies may also influence arrhythmia susceptibility. This study investigated the effects of three class III antiarrhythmic drugs—dronedarone, amiodarone, and dofetilide—on cardiac function, myocardial structure, and oxidative stress in spontaneously hypertensive rats. Rats received one of the drugs for four weeks, after which cardiac performance was evaluated *in vivo* by echocardiography and *ex vivo* using the Langendorff heart preparation. In addition, oxidative stress parameters, myocardial histology, and the expression of HSP70 and SERCA2 were assessed. All three drugs produced partial improvements in cardiac function *in vivo*, with dronedarone showing the most pronounced antihypertensive effect. Under *ex vivo* conditions, dofetilide enhanced myocardial contractility, whereas dronedarone reduced cardiac performance and amiodarone exhibited intermediate effects. Dofetilide and amiodarone also strengthened antioxidant defense system, while dronedarone had variable effects on oxidative stress. Histological analysis demonstrated that dofetilide most effectively preserved myocardial architecture, whereas amiodarone and dronedarone only partially reduced hypertension-induced remodelling. Furthermore, treatment with all three agents decreased HSP70 expression and increased SERCA2 expression, particularly in the dofetilide-treated group. These findings indicate that class III antiarrhythmic drugs exert distinct effects on cardiac function, redox homeostasis, calcium regulation, and myocardial remodelling, which may contribute to their different antiarrhythmic and cardioprotective profiles.

PACING, DEVICE AND ARRHYTHMIA--RELATED OUTCOMES OF PATIENTS WITH PERMANENT PACEMAKER FOLLOWING ALCOHOL SEPTAL ABLATION

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Form: oral presentation, **theme:** cardiomyopathy and cardiac fibrosis
Introduction: Management of left ventricular outflow tract obstruction (LVOTO) is one of the main pillars of care for patients with hypertrophic cardiomyopathy (HCM). Alcohol septal ablation (ASA) remains a viable option for LVOTO elimination in suitable patients, but carries a risk of AV conduction disturbances necessitating a permanent pacemaker (PPM) implantation. Data on pacing and arrhythmia outcomes in patients implanted with a PPM following ASA remain scarce.
Methods: We analyzed data of prospectively followed patients who underwent ASA and were subsequently implanted with a PPM in a single referral HCM center. We retrospectively retrieved data regarding pacing and arrhythmias from device interrogation reports, as well as the data regarding clinical outcomes and mortality using our regular follow-up records and the national electronic health registry.
Results: Overall, data of 44 patients with implanted PPM following ASA were analyzed. The patients had a mean age of 63 ± 11years and were followed for 6.2 ± 5.5 years. The mean percentage of ventricular pacing at the last available follow-up visit was 35.9% per patient, while 61.3% of patients had relevant amount of ventricular pacing. During follow-up, 56.8% of patients had at least one atrial high-rate episode (AHRE) and 50.0% of patients had at least one non-sustained ventricular tachycardia (nsVT) episode recorded in the device memory. Device upgrade was performed in 3 (6.8%) patients. Device infection occurred in 1 (2.2%) patient. Overall mortality was 29.6% with a mean time to death after pacemaker

implant of 8.8 ± 7.1 years (3.3%/year). HCM-related death occurred in 15.4% (1 fatal embolic stroke and 1 heart failure death), cardiac non-HCM related in 15.4%, non-cardiac in 53.9% and unknown in 15.4% patients.
Conclusion: The majority of HCM patients implanted with a PPM following ASA have relevant amount of ventricular pacing during long-term follow-up. Although arrhythmia episodes, both AHRE a nsVT are common, death due to embolic stroke was rare and no patient died due to sudden cardiac death. A small portion of patients required a device upgrade, and device infections were uncommon. Overall, HCM-related death affected less than 1 in 6 patients.

PROLONGED NATIVE T1 OF EPICARDIAL ADIPOSE TISSUE IS ASSOCIATED WITH EPICARDIAL CORONARY SPASM IN PATIENTS WITH ANGINA AND NON-OBSTRUCTIVE CORONARY ARTERIES

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Form: oral presentation, **theme:** coronary artery disease
Background: Epicardial adipose tissue (EAT) is a biologically active visceral fat depot with potential local effects on the coronary circulation. Prior work has linked EAT to coronary microvascular disease, whereas cardiac magnetic resonance (CMR) data suggest that EAT native T1 may capture tissue characteristics beyond fat burden.
Aims: To determine whether CMR-derived EAT volume and mapping characteristics are associated with acetylcholine-induced epicardial coronary spasm in patients with angina and non-obstructive coronary arteries (ANOCA).
Methods and results: In this prospective single-center observational cohort study, consecutive patients with ANOCA referred for comprehensive coronary functional testing, including acetylcholine provocation, underwent prospective CMR assessment of EAT. The primary comparison was epicardial spasm versus no epicardial spasm (microvascular spasm or negative acetylcholine testing). Right ventricular free-wall (RVFW) EAT native T1 was available in 40 patients (26 with and 14 without epicardial spasm). Native T1, the primary imaging endpoint, was longer with epicardial spasm (309.6 ± 79.6 vs 261.3 ± 50.4 ms; $p = 0.040$), whereas exploratory post-contrast T1 and native T2 did not differ; indexed EAT volume (EFVi) was also comparable. In HC3-robust regression, epicardial spasm remained associated with longer native T1 after adjustment for age, sex, and EFVi (adjusted $\beta = 49.6$ ms, 95% CI 5.7–93.5; $p = 0.027$).
Conclusion: Acetylcholine-induced epicardial coronary spasm was associated with selectively prolonged RVFW EAT native T1 despite comparable EAT burden. These hypothesis-generating findings support a potential qualitative EAT phenotype associated with epicardial coronary vasomotor dysfunction.

CT-DERIVED PERIVASCULAR ADIPOSE TISSUE ATTENUATION IN BICUSPID AORTIC VALVE: INSIGHTS INTO PERIVALVULAR AND ASCENDING AORTIC REMODELING

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Form: oral presentation, **theme:** valvular heart disease
Background: Bicuspid aortic valve (BAV) is associated with progressive valvular degeneration and ascending aortic dilatation. Recent studies suggest that inflammatory mechanisms may contribute to this process; however, noninvasive imaging markers that reflect these alterations remain limited. Computed tomography (CT)-derived perivascular adipose tissue attenuation (PVAT) has been associated with the degree of inflammation in other arterial territories. We hypothesized that CT-derived PVAT attenuation could provide novel insights into inflammatory changes around the aortic valve and ascending aorta in BAV.
Purpose: To evaluate CT-derived PVAT attenuation at the perivalvular level and along the ascending aorta in patients with BAV.
Methods: In this retrospective study, 142 patients undergoing CTA were included: 70 BAV with aortic stenosis (AS), 26 BAV with aortic regurgitation (AR) and dilated ascending aorta, and 46 controls. PVAT was assessed using a semiautomated software adapted from coronary PVAT analysis. PVAT was defined as adipose tissue extending 3 mm radially from the aortic valve and 5 mm from the ascending aorta. Attenuation was quantified by calculating CT attenuation of voxels within –190 to –30 Hounsfield units (HU), and mean and maximal PVAT values were recorded and compared across groups.
Results: At the perivalvular level, mean PVAT was higher in BAV than controls (–65.2 vs –71.4 HU, $p < 0.001$). Mean PVAT increased stepwise from controls (–71.4 HU) to BAV with AS (–67.7 HU) and was highest in BAV with AR and dilated aorta (–59.5 HU, $p < 0.05$). Maximal PVAT showed also similar significant differences with a more pronounced gradient (–58.6 HU, –53.4 HU, and –43.4 HU, respectively; $p < 0.001$, **Fig. 1**).

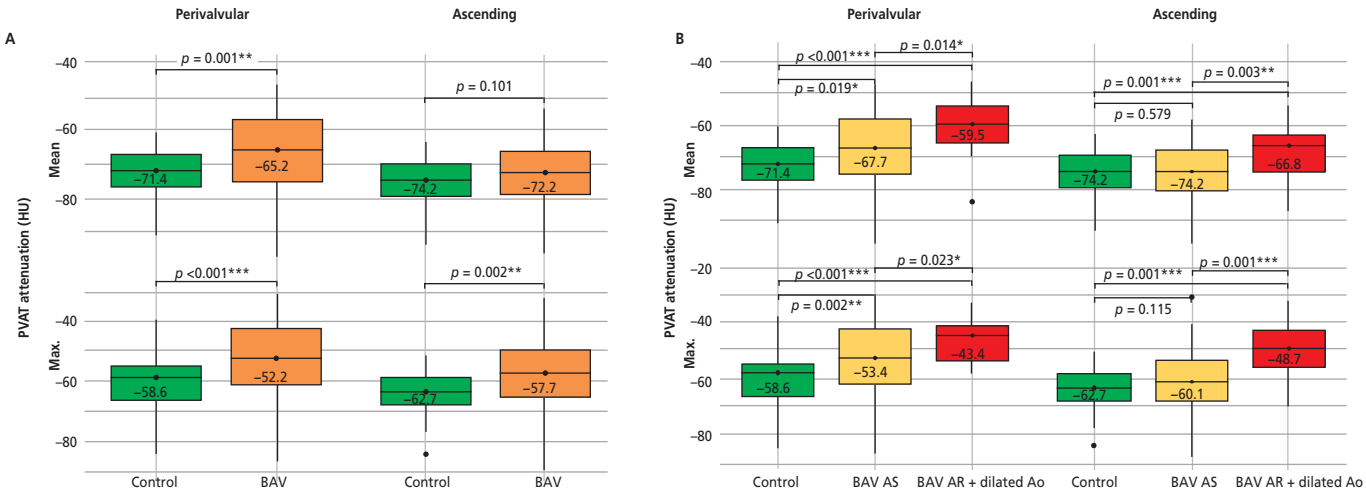


Fig. 1 – Comparison of perivalvular and ascending aortic PVAT attenuation. A) BAV versus controls. B) BAV with aortic stenosis (AS), BAV with aortic regurgitation (AR) and dilated ascending aorta, and controls.

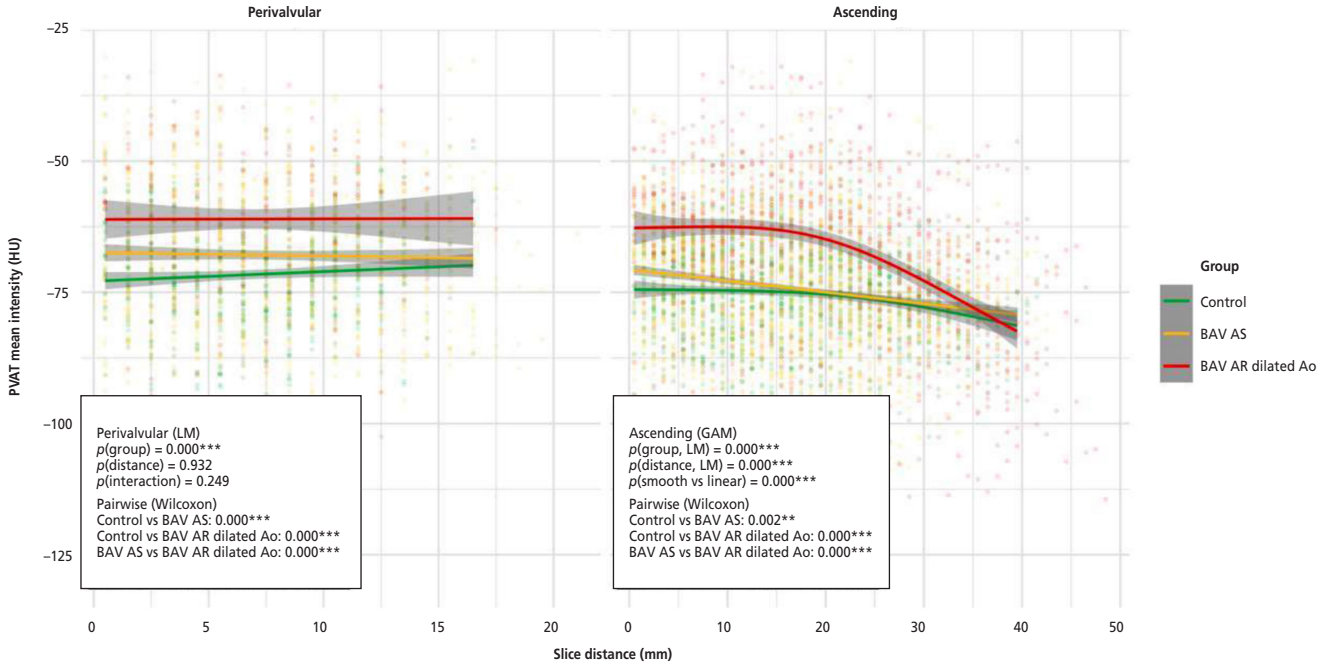


Fig. 2 – Distance-dependent PVAT attenuation in perivalvular and ascending aortic regions.

Along the ascending aorta, mean PVAT were comparable between controls (–74.2 HU) and BAV with AS (–74.2 HU; $p = 0.579$), whereas BAV with AR and dilated aorta demonstrated higher attenuation (–66.8 HU) compared with both controls ($p = 0.001$) and BAV with AS ($p = 0.003$). A similar pattern was observed for maximal PVAT (–62.7 HU in controls, –60.1 HU in BAV with AS, and –48.7 HU in BAV with AR and dilation; $p < 0.001$ for comparisons involving the dilated group). PVAT along the ascending aorta demonstrated a significant distance-dependent and nonlinear pattern ($p < 0.001$), with the highest values observed proximally and progressive decline distally (**Fig. 2**).

Conclusion: PVAT attenuation is elevated in patients with BAV, particularly at the perivalvular level and in those with AR and dilated ascending aorta. Along the ascending aorta, attenuation is highest proximally and decreases distally. These findings suggest that higher PVAT attenuation in BAV may reflect regional inflammatory changes associated with perivalvular and aortic remodeling and support further investigation of PVAT attenuation as a potential noninvasive imaging marker in BAV.

PREVALENCE AND GENETIC ARCHITECTURE OF ARRHYTHMOGENIC MITRAL VALVE PROLAPSE (AMVP), RETROSPECTIVE SINGLE CENTER STUDY

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Form: oral presentation, **theme:** valvular heart disease
Background/Objectives: AMVP is a newly recognized diagnostic subgroup of patients with mitral valve prolapse associated with the risk of malignant ventricular arrhythmia. The goal of the study was to assess the prevalence and possible genetic architecture of AMVP among MVP patients in our center.
Patients and methods: In the last two decades we collected a cohort of 177 patients (105 females – 59%, 72 males – 41%) with mitral valve prolapse (MVP), average time of observation (6 ± 4,6 years). According to diagnostic criteria we identified 58 patients (33%), who met the diagnostic criteria of AMVP (41 females – 70%, 17 males – 30%). AMVP individuals received clinical genetic consultation and molecular genetic analysis using next generation sequencing (targeted/exome sequencing; SOPHiA GENETICS, Switzerland) was performed.
Results: AMVP individuals had a positive family history of SCD, heart failure or aortic disease in 23/58 – 40%). Cardiac arrest (CA) survived or not survived was documented 18/58 individuals (11 females – 61%, 7 males – 39%, 1% from all MVP, average age at CA 34,9 ± 9,7). ACMG.net class 4 and 5 variants in associated genes were disclosed in 8 cases (13%), comprising mostly LP/P variants in titin (TTN) and lamin (LMNA/C). In individuals with positive family history, the genetic yield was 26%.
Conclusion: AMVP represents around 33% of all MVP cases, with risk of cardiac arrest in about 1% of all MVP. Females are prevalent in the whole cohort, but the ratio between sexes is identical for AMVP and sudden cardiac arrest events. Genetic yield in AMVP is low, the direct genotype/phenotype correlation unclear. The study of correlation of ECG, echocardiography, MR findings, exercise ECG and Holter ECG monitoring to the risk of cardiac arrest is ongoing.

THE RAFAEL TRIAL: REGRESSION OF ATHEROSCLEROSIS INDUCED BY LIFE CHANGING EXPERIENCE WITH PSILOCYBIN – ONGOING STUDY

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Form: oral presentation, **theme:** coronary atherosclerosis
Aim: The RAFAEL study aims to prove the efficacy of psychotherapy augmented by psilocybine (PAP) on plaque regression in patients with chronic coronary syndromes (CCS).
Methods: The study is designed as a double blind, single center, randomized trial using PAP on top of standard secondary prevention for patients with CCS. The primary endpoint is the change in percent atheroma volume, which is assessed by intravascular ultrasound (IVUS). The secondary endpoints are: reduction of the number and extent of thin cap fibroatheroma (TCFA) plaque phenotype; and the increase in fibrous cap thickness, which will be assessed by IVUS and optical coherence tomography (OCT). These examinations will be performed at Charles University Hospital in Prague, the Czech Republic, and analyzed in a core lab located in the Iowa Institute for Biomedical Imaging at the University of Iowa, USA. The explorative endpoints are: impact of PAP on global distress, quality of life, and well-being (using standard questionnaires); analysis of the relationship between changes in questionnaire responses, and changes in plaque volume and plaque composition; and the comparison of changes in plaque volume and plaque composition with the results from our previously published HEAVEN trial. All objective scales will be assessed by a psychiatrist/psychotherapist. Self-assessing scales will be provided by the study subjects.
Conclusion: The RAFAEL trial is the first trial to ever work with the objective of proving a link between the use of psychotherapy, augmented by psylocibin treatment, and a positive effect on somatic disorders—in this case, coronary artery disease. Our aim is to improve the efficacy of secondary prevention for patients with chronic coronary artery disease.

THE NEW ROLE AND FUTURE OF PREVENTIVE CARDIOLOGY

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Form: oral presentation, **theme:** preventive cardiology and vascular disease
Background: High-sensitivity cardiac troponin I (hs-TnI) is a promising biomarker for cardiovascular disease (CVD) risk stratification. This study assessed clinical outcomes and cost-effectiveness of an hs-TnI guided CVD screening strategy in a general asymptomatic adult population.
Methods: In a modeled observational program, 4000 adults aged 40–70 years without known CVD underwent hs-TnI testing. Participants were stratified into low (<4 ng/L), moderate (4–10 ng/L), and high (>10 ng/L) risk categories, and underwent further cardiac evaluation, and intervention, if indicated. A discrete-event microsimula-

tion estimated CVD events, mortality, quality-adjusted life years (QALYs), and costs over 10 years.
Results: Among 4000 participants (mean age 57.1 ± 8.6 years; 52% women), 3548 (88.7%) were low-risk, 390 (9.8%) moderate-risk, and 62 (1.5%) high-risk. Non-invasive cardiac workup was performed in 452 (11.3%), and coronary angiography in 112 (2.8%). Significant coronary artery disease (CAD) was diagnosed in 49 (1.2%), with revascularization in 45. Compared to standard care, hs-TnI screening reduced CVD events by 37% and cardiovascular deaths by 34%, gaining 16.3 QALYs per 1000 participants. Incremental cost per person was €528, with an ICER of €32,100/QALY, remaining cost-effective in 93% of simulations.
Conclusions: Hs-TnI guided cardiovascular risk assessment effectively stratifies asymptomatic adults, identifies subclinical CAD, and facilitates preventive intervention, appearing cost-effective in reducing CVD burden.

ASSESSMENT OF CORONARY ARTERY CALCIFICATION PROGRESSION ON STANDARD NON-GATED CT SCANS IN MELANOMA PATIENTS TREATED WITH IMMUNE CHECKPOINT INHIBITORS

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Form: oral presentation, **theme:** coronary artery disease
Background: Immune checkpoint inhibitors (ICIs) have improved the outcomes of patients with advanced melanoma. However, ICIs are linked to a range of immune related adverse events. Furthermore, growing evidence is suggesting that ICIs may also contribute to acceleration of atherosclerosis. Despite the potential clinical implications, the evolution of coronary artery calcification (CAC) in this population remains poorly defined, particularly when evaluated on routinely acquired imaging studies.
Purpose: This study aimed to investigate changes over time in CAC as an indicator for atherosclerotic progression in melanoma patients treated with ICIs.
Methods: A retrospective cohort study was conducted including patients with stage III and IV melanoma who received ICIs between 2012 and 2024 and underwent serial CT imaging during their follow-up. CAC was graded using the visual ordinal scoring system at baseline, 1 year and 2 years after the treatment was initiated. Changes in CAC were analyzed using the paired Wilcoxon signed-rank test. Additionally, the percentage of patients exhibiting CAC progression was determined for each follow-up time point. The effect of statin use was assessed by comparing the proportions of patients demonstrating progression between statin users and non-users using Fisher's exact test.
Results: Among 118 patients, 116 and 82 underwent CT imaging at 1- and 2-year follow-up, respectively. Mean age was 62.1 years and 53.4% were male. Most patients had stage IV melanoma (80.5%), and 57.6% received ipilimumab plus nivolumab. The proportion without CAC decreased from 46.7% at baseline to 40.5% at 1 year (p = 0.023) and 36.6% at 2 years (p = 0.023). Seven patients developed CAC at each follow-up point. Median CAC increased significantly at both 1 year and 2 years (both p <0.001). CAC progression occurred in 26.7% at 1 year and 42.7% at 2 years. At baseline, 27 patients (22.9%) used statins and had higher CAC scores than non-users (median 4 [IQR 1–7] vs. 0 [IQR 0–2], p <0.001). CAC progression occurred in 37.0% vs. 23.6% of statin users and non-users at 1 year (p = 0.215), and 59.1% vs. 36.7% at 2 years (p = 0.082).
Conclusion: Melanoma patients treated with ICIs showed a significant increase in coronary artery calcification during 1- and 2-year follow-up. Statin therapy was not associated with reduced CAC progression. Routine non-gated CT scans with simple CAC scoring may facilitate identification of calcification burden and progression.

IN-HOSPITAL INFLUENZA VACCINATION IN PATIENTS WITH ACUTE CORONARY SYNDROME: IMPROVING UPTAKE IN A HIGH-RISK POPULATION

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Form: oral presentation, **theme:** acute cardiovascular care
Introduction: Patients with acute coronary syndrome (ACS) represent a high-risk group for major cardiovascular complications. Influenza and its systemic inflammatory effects may contribute to atherosclerotic plaque destabilization, thereby increasing the risk of recurrent ischemic cardiovascular events. Seasonal influenza vaccination is an effective strategy to reduce morbidity and mortality, particularly in high-risk populations; however, it remains underutilized in routine clinical practice.
Methodology: Based on current ESC recommendations, an in-hospital influenza vaccination program was implemented for patients acutely hospitalized with ACS at our cardiology department. We conducted a prospective observational study of patients hospitalized for ACS from October 17, 2025, to February 28, 2026. We assessed whether influenza vaccination was offered by the treating physician and whether patients accepted or refused vaccination. Selected cardiovascular risk factors and comorbidities were compared between vaccinated patients and those who refused vaccination. Vaccination uptake was compared with the previous scheme, which required infectious disease consultation and resulted in 12 vaccinations.
Results: A total of 178 patients with ACS were hospitalized, with a mean age of 65.6 ± 12.9 years. Of these, 121 (68.0%) were eligible for in-hospital influenza vaccina-

tion. Among eligible patients, 27 (22.3%) were vaccinated, 24 (19.8%) refused vaccination, and vaccination was not offered to 70 (57.9%). Mean age was 63.8 ± 13.6 years in vaccinated patients and 65.3 ± 11.3 years in those who refused vaccination. Diabetes mellitus was significantly more prevalent among patients who refused vaccination than among vaccinated patients (50.0% vs. 18.5%, p = 0.036). No vaccination-related safety concerns were observed during hospitalization. Compared with the previous scheme, implementation of in-hospital vaccination and simplification of the vaccination process were associated with increased vaccination uptake.
Conclusion: Offering influenza vaccination directly during hospitalization for ACS increases vaccination uptake in this high-risk population and appears to be feasible and safe. Nevertheless, a substantial proportion of eligible patients were not offered vaccination by healthcare professionals, indicating significant room for further improvement. This approach represents a practical strategy to enhance secondary prevention in patients with ACS.

DIFFERENCE IN THE BASELINE CHARACTERISTICS OF DECEASED AND SURVIVING PATIENTS FROM THE MOST-HF REGISTRY

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Form: oral presentation, **theme:** heart failure I
Study objective: The aim of the present study was to compare the baseline characteristics of deceased and surviving patients from the MOST-HF registry at one year follow-up.
Patients and methods: 1050 consecutive patients from 16 centers were included to the study. At time of one year follow-up, 995 patients had information about their outcome.
Results: At one year follow-up, 60 patients (6%) died and 932 (94%) survived. Heart failure was a cause of death in 54% of patients. There were no gender differences. Patients who died were significantly older (mean 73.2 years vs 64.4 years, median 75 vs 66 ys). Mean BMI was significantly lower in whole group and in men but not in women. Duration of heart failure was not different between two groups. The proportion of patients with ischemic heart disease was higher in group of subjects who died. Patients who died at one year had more advanced heart failure according to NYHA (NYHA III 63% vs 20% a NYHA IV 5% vs 0.8%), quality of life (KCCQ total score 55.4 vs 65), heart rate, systolic blood pressure, cardiothoracic ratio (0,59 vs 0,55), median NT-proBNP (3161 pg/ml vs 721.5) and atrial fibrillation on ECG (30% vs 13%). Among echocardiographic parameters, the most significant differences were found for E/E´ratio, LAVi, TAPSE, and IVC collapsibility index. Difference in median LVEF (31% vs 35%) was not significant. Patients who died had more often heart failure symptoms and signs of congestion at baseline. Lower proportion of patients were treated with four fundamental drugs for HFrEF in the group of subjects who died (29.5% vs 59.1%) and lower proportion of patient were treated with individual drug group BB, ARNI and MRA, except of SGLT2 inhibitors. The patients who died were more often treated with digoxin (26.7% vs 10.5%). There were no differences in the device therapy including ICD (51.7% vs 51.1%) and CRT-D in patients with HFrEF and QRS > 120 ms (53.3 % vs 55.9 %) between deceased and surviving patients.
Conclusions: Outpatients with chronic heart failure, who died during one follow-up were older, had lower BMI and signs of more advanced heart failure according to clinical, physical, laboratory and echocardiographic parameters at baseline. Deceased patients had lower rate of optimal medical therapy, except of SGLT2 inhibitors, and they had more often signs of congestion. There was no difference in the use of device-based therapy between deceased and surviving patients.

PROGNOSTIC VALUE OF RIGHT VENTRICULAR MYOCARDIAL WORK IN PATIENTS WITH HEART FAILURE WITH REDUCED AND MILDLY REDUCED EJECTION FRACTION

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Form: oral presentation, **theme:** heart failure I
Introduction: Right ventricular (RV) dysfunction is a major determinant of adverse outcomes in heart failure (HF). However, conventional echocardiographic parameters of RV function fail to fully capture RV–pulmonary arterial (PA) coupling. RV myocardial work (RVMW), derived from non-invasive pressure-strain loops, integrates myocardial deformation and pulmonary pressures. Global work ef-

ficiency (GWE) has shown prognostic value in several cardiovascular diseases, but its prognostic value in HF has not yet been investigated.
The study aims to evaluate the association between GWE and all-cause mortality in patients with HF with reduced (HFrEF) and mildly reduced ejection fraction (HFmrEF).
Methodology: In this single-centre retrospective study, we included consecutive patients with ischemic HFrEF and HFmrEF, without significant tricuspid regurgitation (TR), in whom RVMW analysis was feasible based on image quality and TR signal availability. RVMW indices—including global work index (GWI), global constructive work (GCW), global wasted work (GWW), and GWE—were derived from RV pressure-strain loops using speckle tracking echocardiography.
Results: A total of 239 patients were stratified according to RV GWE tertiles: high (≥90%, n = 83), mid (85–89%, n = 75), and low (≤84%, n = 81). Low-GWE patients were older (67 ± 12 vs 64 ± 12 vs 62 ± 13 years, $p = 0.012$), had worse kidney function, larger LV and LA volumes, worse LV function, and lower conventional RV function parameters. GCW was lower and GWW higher in the Low-GWE group. During a median follow-up of 77 (37–138) months, 72 deaths (30.1%) occurred. After adjustment for age, kidney function, NYHA class, LVEF ≤40%, LA volume index, and significant mitral regurgitation, both Mid GWE (HR 2.58, 95% CI 1.215–5.479, $p = 0.014$) and Low GWE (HR 3.54, 95% CI 1.762–7.111, $p < 0.001$) remained independently associated with all-cause mortality versus High GWE. Five-year mortality increased across GWE tertiles: 10.5%, 20.0%, and 30.1%. Adding GWE to the baseline model significantly improved prognostic performance, with the highest $\Delta\chi^2$ (15.26, $p < 0.001$) and C-index improvement (+0.034) compared with other RV-PA coupling indices.
Conclusions: In patients with ischemic HFrEF and HFmrEF, RV GWE was independently associated with all-cause mortality and provided incremental prognostic value beyond conventional echocardiographic measures of RV-PA coupling, potentially improving risk stratification.

MAKING ADVANCED TRANSCRIPTOMICS ACCESSIBLE FOR CARDIOVASCULAR RESEARCH

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Form: poster
Introduction: Transcriptomic technologies provide powerful tools for investigating cellular identity, differentiation, disease-associated phenotypes, and responses to therapeutic interventions. However, generating reliable and biologically meaningful transcriptomic data across diverse experimental models requires workflows that can accommodate differences in sample type, cell number, sample quality, and experimental design.
Methods: The GeneCore Facility at the Institute of Biotechnology of the Czech Academy of Sciences is an academic core facility specializing in advanced transcriptomics. We provide integrated support for RNA sequencing projects, from experimental design and sample quality assessment to library preparation, quality control, sequencing, and bioinformatic analysis. Our portfolio includes bulk RNA sequencing, single-cell, and single-nucleus RNA sequencing, spatial transcriptomics, and small RNA profiling from plasma and other biofluids.
Results: To address the diverse needs of modern molecular biology, we implement flexible and cost-effective workflows for single-cell transcriptomics, combining established commercial technologies with approaches that do not require dedicated proprietary instrumentation. Depending on the research question, these workflows can support large-scale studies involving hundreds of samples or highly sensitive plate-based analyses of rare and limited cell populations. Such flexibility is particularly valuable in stem-cell research, where cellular heterogeneity, differentiation state, sample availability, and experimental throughput can strongly influence study design and data quality. Small RNA profiling further enables the investigation of circulating RNA species as potential biomarkers of disease states and treatment responses.

LEFT ATRIAL REMODELING AND FUNCTIONAL MALADAPTATIONS IN OBESITY: AN INVASIVE PRESSURE-VOLUME ANALYSIS

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Form: oral presentation, **theme:** heart failure and cardio-oncology
Background: Obesity is a major risk factor for atrial fibrillation (AF) and heart failure (HF). We aimed to characterize left atrial (LA) structural and functional changes across body mass index (BMI) strata in patients with AF referred for catheter ablation.
Methodology: We studied 1,040 consecutive patients (67% males, mean age 62 years, 71% in sinus rhythm). Participants were stratified by BMI (<25, 25–29.9, 30–34.9, ≥35 kg/m²). Comprehensive echocardiographic assessment of LA structure and function was integrated with invasive LA pressure measurements obtained via transseptal access during the ablation procedure. Asso-

ciations across BMI strata were evaluated using regression analyses.
Results: Patients with higher BMI showed larger estimated total blood and plasma volumes along with higher LA volume, pressure, and cyclic wall stress, and a rightward shift of the estimated LA pressure–volume relationship. Conversely, estimated operant LA stiffness did not differ across BMI strata. BMI-related differences in LA volume were attenuated by body surface area indexing but preserved with height-based indexing. Conventional volumetric indices of LA phasic function showed only weak associations with BMI, whereas LA strain and the LA function index were lower with higher BMI. Compared with patients without HF, those with HF exhibited more pronounced structural, functional, and hemodynamic atrial remodeling, consistent with a more advanced atrial myopathy phenotype.
Conclusions: In patients undergoing AF ablation, higher BMI is associated with a predominantly volume load-related pattern of LA remodeling, characterized by larger LA size, higher pressures and cyclic wall stress, and a rightward shift of the estimated pressure–volume relationship. BMI-associated early atrial remodeling may precede overt atrial dysfunction, potentially contributing to the link between excess adiposity, AF, and HFpEF.

EFFECT OF FUROSEMIDE ON SURVIVAL AND CARDIAC FUNCTION IN A RAT MODEL OF HEART FAILURE: A PILOT STUDY

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Form: oral presentation, **theme:** heart failure and cardio-oncology
Background: Chronic heart failure (CHF) is one of the most common causes of acute hospitalization. Diuretic therapy represents the cornerstone of symptomatic CHF treatment, as it alleviates congestion and improves quality of life. However, long-term prognostic data for loop diuretics in CHF are lacking.
Purpose: A placebo-controlled study in an animal model of CHF investigating the effect of diuretic therapy on mortality and selected hemodynamic, laboratory, and echocardiographic parameters. Here we present a pilot substudy of feasibility, optimal dosage of furosemide and necessity of potassium supplementation.
Methods: Aortocaval fistula (ACF) was created in Ren-2 transgenic rats (TGR), a model of angiotensin II-dependent hypertension, at the animal age of 8 weeks. Sham-operated animals served as controls. One week after surgery, animals were randomized into five groups: placebo, low-dose diuretic (5 mg · kg^{-1} · day^{-1}, n = 12), and high-dose diuretic (15 mg · kg^{-1} · day^{-1}, n = 12) administered in drinking water, both doses with and without potassium supplementation, also dissolved in drinking water. The follow-up period was 8 weeks. GraphPad Prism 10 was used for statistical analysis with a log-rank (Mantel-Cox) test to analyze survival data.
Results: All sham-operated animals survived until the end of the experiment. Survival in untreated animals was 42%. The treatment with furosemide improved the survival rate: 67% in the high-dose group (p = 0.18 vs. placebo) and 58% in the low-dose group (p = 0.39 vs. placebo). Although the results were not statistically significant due to small sample size, they demonstrated a clear trend toward improved survival. The diuretic-treated groups had also lower organ weights at the end of the study, reflecting reduced congestion, higher hemoglobin levels, and a smaller inferior vena cava diameter. Renal parameters remained unchanged across all groups. The mortality benefit was not observed in any group receiving potassium supplementation, suggesting a potential negative effect of excessive potassium substitution in CHF.
Conclusion: Furosemide treatment improved congestion in an animal model of CHF induced by aortocaval fistula. A bigger sample size in the following study will be necessary to statistically prove improvement in survival. There will be no need for routine potassium supplementation in this model.

DIFFERENCES IN PULSE PRESSURE AND STROKE VOLUME VARIATION DURING VARIOUS REGULAR PACED RHYTHMS IN CRITICALLY ILL PATIENTS

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Form: oral presentation, **theme:** acute cardiovascular care
Introduction: Pulse pressure variation (PPV) and stroke volume variation (SVV) are established tools for assessing fluid responsiveness. These dynamic parameters are reliable and validated only in patients on controlled mechanical ventilation with regular sinus rhythm. In regular paced rhythm, however, the situation may differ because of the various underlying mechanisms generating respiratory variations of these parameters. This may be particularly relevant when comparing atrial pacing and

sequential atrioventricular pacing, in which lower heart rate variability (HRV) is expected, with atrial-triggered ventricular pacing, in which respiratory HRV is preserved. Main objective of this exploratory study was to test the differences in PPV and SVV values in sinus rhythm and different pacing modes in critically ill patients.
Methodology: Intensive care unit patients with minimally invasive hemodynamic monitoring of cardiac output using arterial pressure pulse contour analysis, with dual-chamber cardiac implantable electronic devices, who were not pacing-dependent and had a regular sinus rhythm, were eligible for the study. Four pacing settings were tested in random order: atrial pacing, sequential atrioventricular pacing, atrial-triggered ventricular pacing, and no pacing (sinus rhythm). HRV and hemodynamic parameters were evaluated during 5-minute periods, each preceded by 5-minute period after changing the device setting to allow hemodynamic stabilization. A Hemosphere monitor with FloTrac or Acumen IQ sensors (Edwards Lifesciences, USA) was used to assess hemodynamic parameters.
Results: Fourteen measures in 7 patients (mean age 78 ± 6 years, 5 males) were performed. Five measures were obtained during controlled mechanical ventilation. HRV was significantly lower during atrial pacing or sequential atrioventricular pacing compared to atrial-triggered ventricular pacing and sinus rhythm (SDNN 12.4 ± 11.2 vs. 51.0 ± 19.6 ms, p <0.001; PNN50 3.0 ± 2.4 vs. 18.2 ± 17.8%, p <0.001. Accordingly, PPV and SVV were significantly lower in pacing modes with atrial pacing compared to atrial-triggered ventricular pacing and sinus rhythm (5.2 ± 3.1 vs. 6.6 ± 3.2%, p = 0.001 and 6.3 ± 3.0 vs. 7.6 ± 3.8%, p <0.001, respectively). Significant correlation between PPV or SVV and some parameters of HRV was observed.
Conclusion: Type of pacing mode should be taken into account when assessing fluid responsiveness using PPV and SVV in patients with a regular paced rhythm.

PREDICTORS OF ISCHEMIC STROKE IN CARDIAC AMYLOIDOSIS

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Form: oral presentation, **theme:** cell therapy, myocarditis, amyloidosis
Background: Cardiac amyloidosis (CA) is associated with atrial dysfunction, atrial fibrillation (AF), and thromboembolic events. However, the prognostic relevance of a documented stroke prior to CA diagnosis and echocardiographic (echo) predictors of future ischemic cerebrovascular events remains unclear.
Purpose: To identify clinical and echo parameters associated with increased risk of stroke during follow-up in patients with CA.
Methods: A total of 1394 patients (age 71 ± 15 years, 72% male) with confirmed CA were included (transthyretin amyloidosis 73%; immunoglobulin light-chain amyloidosis 23%). Clinical and echo characteristics were compared between patients with and without documented stroke (9% vs. 91%) prior to CA diagnosis. Baseline echo included conventional measures such as left ventricular global longitudinal strain (LV GLS) and left atrial volume index (LAVI), with severe LA dilation defined as LAVI >48 mL/m² according to ESC/EACVI guidelines. The study endpoint was ischemic stroke during follow-up. Cox proportional hazards models were used for univariable and multivariable analyses. Inverse probability of treatment weighting (IPTW) based on established stroke risk factors (age, sex, hypertension, diabetes, hyperlipidemia and AF) was applied to reduce confounding. A Fine–Gray model accounted for death as a competing risk.
Results: Patients with documented stroke prior to CA diagnosis were older and had more hypertension, diabetes, hyperlipidemia and AF, as well as more advanced heart failure related symptoms (New York Heart Association functional class ≥III) and higher NT-proBNP, compared to patients with no previously documented stroke. Echo of these patients also showed greater LV mass index and LAVI, more impaired LV GLS and a lower E/e' ratio. Over a median follow-up of 2 years (IQR 1–4), 38 (3%) patients experienced ischemic stroke and 375 (27%) patients died. In multivariable IPTW-adjusted Cox analyses, prior stroke and LAVI >48 mL/m² were independently associated with 3-year stroke risk (prior stroke: HR 5.05; LAVI >48 mL/m²: HR 1.52). After accounting for death as a competing risk, prior stroke remained independently associated with an increased stroke risk (sHR 4.29).
Conclusion: In CA, documented stroke prior to diagnosis and severe LA dilation (LAVI >48 mL/m²) identify a high-risk subgroup with increased risk of future ischemic cerebrovascular events, independent of established stroke risk factors.

PLASMA AND MYOCARDIAL PROTEOMIC SIGNATURES OF LEFT VENTRICULAR RECOVERY IN RECENT ONSET DILATED CARDIOMYOPATHY

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Form: poster
Background: Recovery of left ventricular (LV) systolic function in patients with recent-onset dilated cardiomyopathy (RODCM), defined as new-onset heart failure with reduced LV ejection fraction (LVEF) below 40% without a history of chronic coronary syndrome, is difficult to predict. We explored whether plasma and myocardial proteomic profiles identify biological signals associated with 6-month recovery.
Methods: Mass-spectrometry proteomics was performed in two distinct RODCM cohorts: a retrospective (105 patients; 49/56 recovery/non-recovery [R/NR]) and a prospective cohort (47 patients; 23/24 R/NR). Plasma samples were either non-depleted (152 patients; 72/80 R/NR from both cohort) or abundant-plasmatic-protein-depleted (66 randomly selected patients; 32/34 R/NR). Endomyocardial biopsy (EMB) proteomics was available in the prospective cohort (47 patients; 23/24 R/NR). Recovery was defined as left ventricular ejection fraction (LVEF) ≥40% at 6 months after RODCM onset. Proteomic profiles were compared according to recovery status, accounting for baseline LVEF and inflammatory status where applicable, followed by exploratory GO enrichment analyses.
Results: In the retrospective cohort, baseline LVEF was 24.5 ± 6.9%; at 6 months, LVEF was 44.7 ± 4.8% in R versus 26.8 ± 5.9% in NR. In the prospective cohort, baseline LVEF was 25.5 ± 9.5%; at 6 months, LVEF was 48.6 ± 8.1% in R versus 29.2 ± 6.2% in NR. No individual plasma protein remained significant after proteome-wide false discovery rate (FDR) correction. Nevertheless, extracellular superoxide dismutase (SOD3) showed a consistent directional association with recovery in non-depleted plasma and EMB; however, the plasma association was attenuated after adjustment for baseline LVEF (meta-analysis log2FC –0.23, $p = 0.062$), while the association in EMB remained nominally significant (log2FC –0.44, $p = 0.034$). Plasma pathway analyses suggested differences in coagulation and inflammatory processes. In contrast, EMB demonstrated enrichment of cell adhesion (FDR = 0.005), ATP synthesis (FDR = 0.005), and mitochondrial ATP synthesis (FDR = 0.025), alongside extracellular matrix/remodeling-related processes.
Conclusions: LVEF recovery was not characterized by a single robust circulating or endomyocardial protein but was associated with exploratory proteomic signals involving inflammation, tissue remodeling, cell adhesion, and mitochondrial energetics. SOD3 emerged as a consistent candidate across plasma and myocardial tissue.

LOW SVO_2, BUT NOT ELEVATED LACTATE, IDENTIFIES PATIENTS WHO MAY BENEFIT FROM ECMO IN CARDIOGENIC SHOCK: A POST-HOC ANALYSIS OF THE MULTICENTER RANDOMIZED ECMO-CS TRIAL

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Form: oral presentation, **theme:** acute cardiovascular care
Introduction: Immediate initiation of extracorporeal membrane oxygenation (ECMO) has not demonstrated consistent clinical benefit in patients with cardiogenic shock diagnosed primarily on the basis of hypotension and signs of tissue hypoperfusion. In the Extracorporeal Membrane Oxygenation in the Therapy of Cardiogenic Shock (ECMO-CS) trial, tissue hypoperfusion was defined by either low venous oxygen saturation (SvO_2) or elevated lactate levels. However, the ability of these parameters to identify patients who may benefit from immediate ECMO initiation remains uncertain. We therefore evaluated the effect of immediate ECMO initiation according to baseline SvO_2 and lactate levels.
Methods: In the ECMO-CS trial, 117 patients with cardiogenic shock were randomized to immediate ECMO initiation or an early conservative strategy. In this post-hoc analysis, the effect of immediate ECMO was assessed in subgroups defined by baseline SvO_2 <50% and lactate ≥3 mmol/L. The primary endpoint was all-cause mortality at one year. The secondary endpoint was a composite of all-cause mortality or hemodynamic deterioration requiring ECMO.

Results: Baseline SvO_2 was available in 95 patients. Among patients with SvO_2 <50% (n = 40), immediate ECMO initiation was associated with a significantly lower risk of one-year all-cause mortality (HR 0.35; 95% CI 0.15–0.81; $p = 0.014$) and of the composite secondary endpoint (HR 0.34; 95% CI 0.15–0.76; $p = 0.003$). Baseline lactate was available in all 117 patients. Among patients with lactate ≥3 mmol/L (n = 92), immediate ECMO initiation was not associated with a significant reduction in one-year all-cause mortality (HR 0.86; 95% CI 0.53–1.41; $p = 0.538$) or the composite secondary endpoint (HR 0.65; 95% CI 0.41–1.05; $p = 0.067$).
Conclusions: In this post-hoc analysis of the randomized ECMO-CS trial, immediate ECMO initiation was associated with improved clinical outcomes in patients with low baseline SvO_2, whereas elevated lactate did not identify a subgroup with a significant treatment effect. These findings suggest that SvO_2 may provide greater discriminatory value than lactate for identifying patients with cardiogenic shock who may benefit from early mechanical circulatory support.

TARGETING THE SARCOMERE IN HCM AND HFPEF: MECHANISTIC AND THERAPEUTIC PERSPECTIVES ON CARDIAC MYOSIN INHIBITION

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Form: oral presentation, **theme:** heart failure I
Hypertrophic cardiomyopathy (HCM) and heart failure with preserved ejection fraction (HFpEF) share a common pathophysiological substrate at the level of the cardiac sarcomere, making myofilament-targeted pharmacotherapy a rational therapeutic strategy. Both conditions are characterized by impaired diastolic function, altered myofilament regulation, and compromised Frank–Starling reserve. Cardiac myosin inhibitors (CMIs) modulate the actomyosin cross-bridge cycle by stabilizing myosin in its autoinhibited super-relaxed state, reducing force-generating cross-bridges without interfering with intracellular calcium handling. This mechanism distinguishes CMIs from conventional negative inotropic agents and offers the potential to reduce hypercontractility while preserving intrinsic sarcomeric regulatory properties.
In HCM, clinical trials with mavacamten and aficamten have demonstrated effective reduction of LVOT gradients, improved functional capacity, and symptom relief. Mechanistic studies suggest these benefits are mediated through restoration of diastolic chamber volumes and recruitment of Frank–Starling reserve rather than simple contractility suppression. In HFpEF—increasingly recognized as a disorder of impaired myofilament relaxation, increased passive stiffness, and blunted preload responsiveness—early proof-of-concept studies suggest that myosin inhibition may ameliorate elevated filling pressures and impaired Frank–Starling reserve, though definitive clinical evidence remains forthcoming.
Our recent work examined myofilament-level effects of aficamten in healthy rat and canine myocardium using integrated *in vivo*, intact-cell, and skinned fiber approaches. Aficamten-induced negative inotropy was compensated by increased diastolic chamber volumes and recruitment of length-dependent force generation, maintaining stroke volume and cardiac output. Diastolic indices (E/e', IVRT) reflected hemodynamic consequences of increased preload rather than primary myofibrillar dysfunction, suggesting that CMI therapy engages intrinsic cardiac compensatory mechanisms.
Accumulating evidence positions CMIs as a promising therapeutic class capable of targeting sarcomere-level dysfunction while preserving Frank–Starling reserve and hemodynamic stability. Future studies in disease-relevant models and well-defined patient populations will be essential to fully characterize their therapeutic potential across the spectrum of HCM and HFpEF.

WHY REHABILITATION FAILS IN PERIPHERAL ARTERIAL DISEASE: CT-DERIVED MUSCLE QUALITY IDENTIFIES THE MYOSTEATOTIC BARRIER

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Form: oral presentation**, theme:** peripheral vascular and aortic disease
Introduction: Supervised exercise therapy is first-line treatment for intermittent claudication, yet a proportion of patients fail to achieve adequate symptom relief and ultimately require revascularization. Conventional frailty assessment focuses on muscle mass and body mass index, potentially overlooking muscle quality and visceral adiposity as more relevant determinants of rehabilitative capacity. We investigated whether CT-derived body composition parameters can explain and predict this variability in exercise therapy response.
Methodology: Prospective pilot series of 8 PAD patients (Fontaine IIa–IIb; mean age 62 ± 8 years) completing a 3-month structured supervised exercise program. Automated CT body composition analysis (AutoMATiCA) at the L3 vertebral level was performed on existing angiographic imaging, measuring skeletal muscle index (SMI, cm^2/m^2), muscle density (HU), and visceral (VAT) and subcutaneous adipose tissue areas—all without additional cost or patient burden. Functional assessments: 30-second chair stand test, handgrip dynamometry, 2-minute walk test. Primary outcome: conservative management success versus revascularization. Correlations: Spearman rank coefficient.

Results: Five patients (62.5%) achieved treatment success; three (37.5%) required revascularization. Rehabilitation failures paradoxically showed greater muscle mass (SMI 59.7 vs 48.6 cm²/m²) but lower muscle density (35.9 vs 41.1 HU) and higher BMI (29.6 vs 24.9 kg/m²). Muscle density correlated strongly with chair stand performance (ρ = +0.829, p = 0.021), whereas SMI showed no functional associations. VAT demonstrated the strongest relationships with walking distance (ρ = –0.900, p = 0.037) and handgrip strength (ρ = –0.775, p = 0.041). A positive VAT–SMI correlation (ρ = +0.821, p = 0.023) confirmed sarcopenic obesity as the dominant risk phenotype.
Conclusion: Rehabilitation failure in PAD is characterized by a myosteatotic obesity phenotype—preserved muscle volume coexisting with poor quality and elevated visceral adiposity—entirely undetected by conventional anthropometric methods. CT-derived muscle density and VAT area, extractable from angiographic imaging routinely obtained for vascular planning, distinguish rehabilitation responders from non-responders more accurately than BMI or muscle mass. These pilot findings support prospective validation of CT-based metabolic phenotyping as a patient selection tool for conservative claudication management.

ADAPTIVE BIOELECTRONIC PACING: RESTORING PHYSIOLOGICAL HEART RATE VARIABILITY

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Form: oral presentation, **theme:** arrhythmia and electrophysiology I
Natural cardiorespiratory synchronization, primarily manifested as respiratory sinus arrhythmia (RSA), is guided by autonomic brainstem activity and is fundamental for homeostatic cardiovascular regulation. Aging and pathological cardiac remodeling often degrade or abolish this natural heart rate variability (HRV). Conventional cardiac rhythm management devices operate monotonically and lack the capacity to dynamically adapt inter-chamber delays or cycle lengths to match real-time respiratory feedback. CresPace predeceasing project developed an adaptive bioelectronic platform, whin now as CResPulse utilizes real-time thoracic bioimpedance sensing to feed a digital twin of brainstem neuronal circuits (central pattern generators, CPGs), thereby enabling autonomous, physiologically responsive, closed-loop cardiac pacing.
Methods: A custom bioimpedance sensing configuration was utilized to capture continuous, time-varying thoracic impedance changes reflecting lung volume displacement during breathing. To evaluate the performance, signal stability, and noise margins of this sensing solution, we performed extensive validations across animal cadaver systems and dynamic *in-vivo* porcine models during acute pacing trials. The extracted respiratory envelope was processed to serve as a direct drive input to the neuromorphic CPG algorithm.
Results: The custom bioimpedance platform demonstrated high sensitivity in capturing respiratory cycles and tracking breathing phases in real time, exhibiting strong resistance to baseline drift and signal artifacts. This robust sensing allowed the integrated neuronal digital twin to autonomously calculate and deliver synchronized, beat-to-beat ventricular and atrial pacing sequences without cycle dropouts.
Conclusions: Integrating bioimpedance-derived respiratory feedback with neuromorphic digital twins of autonomic circuits establishes a viable pathway for personalized, closed-loop cardiac resynchronization therapy. This bioelectronic approach offers a highly clinically translatable strategy to restore natural cardiorespiratory coupling and improve hemodynamic performance in chronic cardiac disease.

PROCEDURAL AND CLINICAL OUTCOMES AFTER INTRAVASCULAR LITHOTRIPSY ACCORDING TO TARGET VESSEL: INSIGHTS FROM THE BENELUX-IVL REGISTRY

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Form: oral presenation, **theme:** intravascular lithotripsy (IVL)
Background: Coronary artery calcification (CAC) may limit optimal percutaneous coronary intervention (PCI). Intra-

vascular lithotripsy (IVL) improves vessel compliance, but whether outcomes differ by target vessel location remains unclear.
Methods: This retrospective analysis of data from the multicenter BENELUX-IVL registry included consecutive patients undergoing IVL-assisted PCI for calcified coronary lesions between May 2019 and January 2025. Patients treated with IVL in multiple vessels during the same procedure were excluded. Patients were stratified by IVL-treated target vessel into LM, LAD, LCx, and RCA groups. The primary endpoint was technical success. Secondary endpoints included procedural and device success, periprocedural complications, and clinical outcomes up to 2 years.
Results: Among 555 patients, IVL was performed in the RCA in 212, LCx in 83, LAD in 241, and LM in 19. Target vessel location was associated with distinct lesion and procedural characteristics, including bifurcation lesions, long segment lesions, in-stent lesions, balloon dimensions, and number of IVL pulses. Technical success was comparable across target vessels (89.5%; $p = 0.64$), as were procedural success (88.5%; $p = 0.56$), device success (97.4%; $p = 0.53$), and overall complications (6.1%; $p = 0.65$). Clinical outcomes did not differ, including cumulative 1-year MACE (10.7%; $p = 0.83$) and 2-year MACE (1.3%; $p = 0.38$).
Conclusions: IVL-assisted PCI demonstrated consistent procedural efficacy, safety, and clinical outcomes across coronary target vessels, supporting IVL as a versatile calcium modification strategy for calcified lesions irrespective of target vessel.

LONG-TERM INCIDENCE OF FIRST HOSPITAL-RECORDED ATRIAL ARRHYTHMIAS AND ALL-CAUSE MORTALITY AFTER PERCUTANEOUS PATENT FORAMEN OVALE CLOSURE: A SINGLE-CENTRE COHORT LINKED TO NATIONWIDE REGISTRIES

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Form: oral presentation, **theme:** cardiovascular interventions and cardiac arrest
Introduction: Long-term arrhythmic outcomes and population-standardized survival after percutaneous patent foramen ovale (PFO) closure remain incompletely defined. To our knowledge, relative survival has not previously been applied in this setting. We evaluated first hospital-recorded atrial arrhythmias (AAs), catheter ablation and mortality during nationwide follow-up. The endpoint captured clinically recognised events requiring hospital-based evaluation or treatment, not total AA burden.
Methodology: Adults undergoing PFO closure at a Czech tertiary centre between 2010 and 2024 were deterministically linked to nationwide hospital and mortality registries. The primary endpoint was first hospital-recorded AA after closure in patients without clinically documented pre-procedural AA. A qualifying event required an inpatient, day-case or hospital-based outpatient encounter with AF, atrial flutter or atrial tachyarrhythmia recorded as a primary or secondary diagnosis; encounters could include hospitalization, acute cardiology assessment or ambulatory electrical cardioversion. Cumulative incidence was estimated by Kaplan–Meier and Fine–Gray methods. Relative survival was calculated using the Pohar–Perme estimator against age-, sex- and calendar year-matched Czech population life tables.
Results: Overall, 715 procedures were performed in 707 patients (median age 49.4 years; 48.0% male), contributing 4,329 patient-years (maximum 14.9 years). Thirteen patients had a first hospital-recorded AA: 11 AF/flutter and two supraventricular tachycardia. Ten-year cumulative incidence was 2.9% (95% CI 1.2–4.6%) by Kaplan–Meier and 2.8% (95% CI 1.2–4.4%) by Fine–Gray analysis. Five events occurred within one year and six beyond three years. Affected patients were predominantly male with a high cardiovascular risk burden; with only 13 events, these patterns are descriptive and hypothesis-generating. Catheter ablation was performed in 11 patients (10-year incidence 2.0%). Thirty-four patients died (10-year mortality 7.5%).
Conclusion: Clinically recognized, hospital-recorded AAs after PFO closure were uncommon, although some occurred years after the procedure. The estimate excludes asymptomatic, self-limited and primary-care-only arrhythmias, and late events cannot be causally attributed to closure. Observed survival closely matched population expectations, with no observed excess mortality at 10 years (relative survival 1.023, 95% CI 0.982–1.065).

OPTICAL COHERENCE TOMOGRAPHY--EVALUATED CHOROIDAL BRIGHTNESS: A NOVEL INDEPENDENT NON-INVASIVE PREDICTOR OF CORONARY ARTERY DISEASE

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Form: oral presentation, **theme:** coronary artery disease
Purpose: To evaluate whether OCT-derived normalized choroidal brightness represents a novel non-invasive imaging biomarker for personalized prediction of coronary artery disease beyond traditional risk factors.
Design: Prospective frequency-matched case-control study.
Subjects: Eighty-four adults with a high pre-test probability of coronary artery disease referred for invasive coronary angiography were included. The study population comprised 60 consecutive patients with angiographically confirmed coronary artery disease and 24 controls without significant coronary artery disease, frequency-matched for age, diabetes mellitus, and biological sex.
Methods: All participants underwent coronary angiography and standardized macular OCT imaging using the Spectralis HRA+OCT system with a standardized volumetric scan protocol (61 B-scans, ART 9) centered on the fovea. Identical acquisition settings were applied to all participants to ensure measurement consistency.
Main outcome measures: Regional thickness and brightness quantified by calculating the mean values within each ETDRS subfield and global choroideal brightness as the mean of subfields brightnesses.
Results: Lower global choroidal brightness was significantly associated with CAD. Each 1-unit decrease in brightness was associated with 38% higher odds of angiographically confirmed CAD (odds ratio [OR], 1.38; 95% CI, 1.18–1.67). A prespecified threshold (<10) was associated with increased odds of CAD (OR, 21.1; 95% CI, 3.6–123.5). Discrimination using global choroidal brightness was good (AUC, 0.83; 95% CI, 0.73–0.92), with 78% sensitivity and 79% specificity. In contrast, conventional retinal and choroidal thickness parameters did not differ between age-, sex-, and diabetes-matched groups.
Conclusions: Normalized choroidal brightness derived from routine OCT imaging was independently associated with angiographically confirmed CAD, whereas conventional retinal and choroidal thickness metrics were not. These findings support the potential of choroidal brightness as a scalable, non-invasive imaging biomarker for CAD risk stratification.

COMPARISON OF CONVENTIONAL RIGHT VENTRICULAR PACING VERSUS CONDUCTION SYSTEM PACING USING ECHOCARDIOGRAPHICALLY ASSESSED MYOCARDIAL WORK

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Form: oral presentation, **theme:** arrhythmia and electrophysiology II
Introduction: Conventional pacing prolongs left ventricular (LV) electrical activation, manifesting as QRS widening, which in turn produces mechanically dyssynchronous contraction and reduces contractile efficiency. This can be quantified as a changer in myocardial work, which can be assessed non-invasively by echocardiographic speckle-tracking analysis. Conduction system pacing (CSP), by contrast, achieves faster LV activation that more closely approximates physiological conduction, and may therefore be expected to yield superior myocardial work efficiency. This study aimed to compare myocardial work efficiency between conventional pacing and CSP.
Methodology: Patients undergoing permanent pacemaker implantation who provided informed consent were prospectively enrolled. In each patient, transthoracic echocardiographic loops were acquired intraprocedurally at three time points: during native conduction, following lead positioning on the interventricular septum (conventional pacing), and after advancement of the lead to its final subendocardial position within the left bundle branch area (CSP). These loops were subsequently analyzed using dedicated myocardial work software.
Results: A total of 16 patients have been enrolled to date; echocardiographic data were of insufficient quality for analysis in 7 patients, leaving 9 evaluable patients (mean age 75.7 years, 55% male). CSP outperformed conventional pacing across all evaluated parameters: global LV strain (–11.1 ± 1.2 vs. –8.2 ± 2.1%, p = 0.014), global work index (1104.6 ± 186.2 vs. 896.8 ± 175.1 mmHg%, p = 0.027), and global work efficiency (79.3 ± 4.8 vs. 71.2 ± 5.3%, p = 0.025).
Conclusion: These preliminary data suggest that physiological pacing achieves more favorable myocardial work parameters than conventional pacing. Further enrollment is ongoing to confirm these findings in a larger patient cohort.

INTERNATIONAL EXPERIENCE WITH IMPLANTABLE CARDIOVERTER-DEFIBRILLATORS FOR THE PREVENTION OF SUDDEN DEATH IN HIGH-RISK PATIENTS WITH HYPERTROPHIC CARDIOMYOPATHY

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Form: oral presentation, **theme:** cardiomyopathy and cardiac fibrosis

Background: Implanted cardioverter-defibrillators (ICDs) have been used in patients with hypertrophic cardiomyopathy (HCM) to prevent sudden death and have proven lifesaving for many patients. However, experience with ICD therapy has largely been derived from relatively small HCM cohorts confined to specific countries or regions of the world. Therefore, we sought to determine the effectiveness of ICDs in preventing sudden death due to life-threatening ventricular arrhythmias in a large international multicenter HCM population.

Methods: Databases from 25 HCM centers (8 in the United States, 9 in Europe, 4 in Asia, and 1 each in Australia, Africa, Mexico, and South America) were retrospectively interrogated to identify consecutive patients with HCM with ICDs (1992 to 2024) followed for 7±6 years (up to 32 years) for clinical outcomes.

Results: A total of 3387 patients were identified (63% men). They had a mean left ventricular thickness of 22 ± 7 mm. The participants had received ICDs at a mean age of 47 ± 17 years. Over follow-up, 550 patients (16%) experienced ≥1 appropriate ICD therapy (2.6%/y), including 86 of the 247 implanted for secondary prevention (35% [6.4%/y]) and 464 of the 3140 implanted for primary prevention (15% [2.2%/y]). Appropriate therapy occurred in the 464 primary prevention patients at a mean age of 49 ± 17 years, with a median time to first appropriate therapy of 4 years after ICD implantation; 16% of these received their first appropriate therapy ≥10 years after implantation, and 47% experienced multiple interventions. Independent predictors of appropriate ICD therapy included unexplained syncope, left ventricular apical aneurysms, left ventricular systolic dysfunction, and nonsustained ventricular tachycardia on ambulatory monitoring. Of the 3140 primary prevention patients, 2946 survived (94%) and 194 died (6%) (0.8%/y), including 68 due to HCM (0.3%/y), predominantly of end-stage heart failure (n = 43) or stroke (n = 9). In contrast, 11 patients (0.4%) died suddenly, with device failures occurring in 2.4% of those with life-threatening ventricular tachyarrhythmias. Survival free from HCM-related mortality at 10 and 20 years was 97% and 91%, respectively.

Conclusions: In this international multicenter study, the largest to date, including >3300 consecutive patients with HCM and ICDs, device therapy terminated potentially lethal ventricular tachyarrhythmias in 1 of 6 patients, with low HCM-related mortality rates for patients with ICDs.

FUNCTIONAL AND APPLIED MORPHOLOGY OF HUMAN ATRIA

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Form: oral presentation, **theme:** arrhythmia and electrophysiology I
Purpose: This study aimed to quantify macroscopic variations in pectinate muscle architecture in human atrial appendages and evaluates their visualization using clinical imaging modalities. Then, we compared the morphology with commonly used experimental animal species (mouse and chicken).
Methods: We examined 153 cadaveric donor hearts (103 specified sex: 61 female, 42 male; plus 50 unspecified), employing gross dissection, *ex vivo* micro-CT, histology, and *in vivo* clinical imaging (CT angiography, MRI, and transthoracic/transesophageal echocardiography).
Results: Left atrial appendage morphologies comprised chicken-wing (41%), cactus (23%), cauliflower (22%), and windsock (14%) types, and contained 11 ± 4 (mean ± SD) main pectinate muscles (length: 47 ± 16 mm). Right atrial appendage pectinate muscle arrangements were dominated by type IV (41%), followed by types I/II (22%/20%), with 17 ± 4 muscles (length: 75 ± 17 mm); they were larger (p <0.001) but showed lower muscle density (p = 0.007) than the left atrial appendages. All modalities resolved main pectinate bundles, with angio-CT providing superior LAA detail; transesophageal echocardiography excelled for both sides. Atrial appendages are much more prominent structures in the mouse and chick atria, and the left-right differences are less pronounced, probably due to a lesser degree of pulmonary vein incorporation in the left atrium. Knowledge of atrial appendage morphology is important for catheter ablation, electrical pacing, and stroke risk stratification, bridging anatomical and clinical perspectives.

TRENDS AND OUTCOMES OF CARDIOGENIC SHOCK OF DIFFERENT ETIOLOGIES: RESULTS FROM A SINGLE-CENTRE REGISTRY (2001–2024)

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Form: oral presentation, **theme:** acute cardiovascular care
Background: Demographic changes and advances in cardiogenic shock (CS) management are reshaping the clinical spectrum of CS. The study investigates temporal trends in CS etiologies, as well as outcomes of CS stratified by the presence or absence of acute myocardial infarction (AMI).
Methods: Consecutive CS patients were included at one University Medical Centre from 2001 to 2024. Time trends regarding the distribution of CS etiologies and risk of 30-day in-hospital all-cause mortality were investigated stratified by 5-year periods. The prognostic impact of AMI-CS as compared to non-AMI-CS was investigated using Kaplan-Meier and multivariable Cox proportional regression analyses.
Results: From a total of 805 CS patients, 47% (n = 378) had AMI-CS. Patients with AMI-CS had higher rates of cardiac arrest, mechanical ventilation and were more frequently treated with mechanical circulatory support devices. From 2001 until 2024, the proportion of AMI-CS was demonstrated to decrease (2001–2004: 71%; 2021–2024: 47%; p = 0.001). AMI-CS was associated with an increased risk of all-cause mortality at 30 days (hazard ratio (HR) = 1.280; 95% confidence interval (CI) 1.077–1.520; p = 0.005), which was still observed after multivariable adjustment (adjusted HR = 1.220; 95% CI 1.019–1.461; p = 0.030). From 2001 until 2024, crude 30-day mortality rates increased (2001–2004: 52%, 2021–2024: 64%; p = 0.001) both for AMI-CS and non-AMI-CS, in line with an increase in comorbidity burden.
Conclusion: Although the rates of AMI-CS were demonstrated to decrease within the past two decades, AMI-CS was independently associated with a higher risk of 30-day all-cause mortality.

LONG-TERM PROGNOSIS OF PATIENTS WITH INFARCT-LIKE ACUTE MYOCARDITIS: A RETROSPECTIVE REGISTRY ANALYSIS

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Form: poster or oral presentation, **theme:** cell therapy, myocarditis, amyloidosis

Introduction: Acute myocarditis is an inflammatory heart disease with highly variable clinical presentation. A substantial subgroup of patients presents with chest pain mimicking acute coronary syndrome (ACS). This presentation is often referred to as infarct-like myocarditis. Published data on the long-term prognosis of these patients remain limited and inconsistent, with most studies reporting follow-up periods of up to five years. The aim of this study was to assess long-term outcomes in patients with infarct-like acute myocarditis, focusing on overall mortality, cardiovascular mortality, and cardiovascular rehospitalizations.

Methods: We conducted a retrospective single-center registry study including 137 patients hospitalized with a final diagnosis of acute myocarditis between January 1, 2010 and December 31, 2024. Clinical characteristics, laboratory findings, echocardiographic parameters, and results of coronary angiography or CT coronary angiography and cardiac magnetic resonance imaging were analyzed. Long-term follow-up data were obtained in collaboration with the National Registry of Reimbursed Health Services (NRHZS). Survival analyses were performed using Kaplan–Meier models for overall survival, cardiovascular survival, and survival free from cardiovascular death or cardiovascular hospitalization. Follow-up was censored on December 31, 2024. The maximum follow-up period approached 15 years.

Results: During long-term follow-up, only two deaths (1.5%) occurred among the 137 patients. Neither death was attributable to cardiovascular causes. Kaplan–Meier analysis demonstrated excellent overall survival, with survival probabilities of 98.9% (95% CI: 96.7–100%) at 4 years and 95.7% (95% CI: 89.4–100%) at 9 years. Cardiovascular mortality was 0%. In contrast, analysis of the composite endpoint (cardiovascular death or cardiovascular hospitalization) revealed a gradual decline in event-free survival over time. Approximately 10 years after the index hospitalization, the probability of remaining free from cardiovascular events decreased to approximately 41%.

Conclusion: Patients with infarct-like acute myocarditis have an excellent long-term prognosis in terms of overall and cardiovascular mortality, even with extended follow-up approaching 15 years. However, despite the very low mortality risk, a substantial proportion of patients experience subsequent cardiovascular hospitalizations over time.

SEX DIFFERENCES IN MANAGEMENT AND OUTCOMES AFTER OUT-OF-HOSPITAL CARDIAC ARREST WITH ANGIOGRAPHICALLY CONFIRMED OBSTRUCTIVE CORONARY ARTERY DISEASE

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Form: oral presentation, **theme:** cardiovascular interventions and cardiac arrest

Background: Previous studies have reported sex-based disparities in care, often disadvantaging women. Large registry analyses are limited by incomplete characterization of OHCA etiology and coronary angiography (CAG) findings. We aimed to evaluate sex differences in management and outcomes among patients with angiographically confirmed obstructive coronary artery disease (CAD) following OHCA.

Methods: We performed a post hoc analysis of consecutive adult OHCA patients with angiographically confirmed obstructive CAD admitted between 2012 and 2023 using a prospective OHCA registry. Obstructive CAD was defined as ≥70% coronary stenosis identified by coronary angiography and considered the most likely cause of OHCA. The primary outcome of this analysis was 30-day survival. The secondary outcome was favorable neurological outcome at hospital discharge. Multivariable logistic regression was used to assess the independent association between sex and 30-day survival.

Results: Among 1,219 OHCA admissions, 634 (52%) patients had angiographically confirmed CAD, and 580 (91.5%) were included in the final analysis (median age 61 years, 15.3% women). ST-segment elevation myocardial infarction was the predominant etiology in both groups (64% in women vs. 59.9% in men). Men more frequently had a history of CAD and prior myocardial infarction. No significant sex-related differences were observed in PCI utilization, procedural success, targeted temperature management (TTM), or extracorporeal cardiopulmonary resuscitation (ECPR) use. Thirty-day survival was 66.2% in women and 61.5% in men (log-rank $p = 0.33$), while favorable neurological outcome (CPC 1–2) occurred in 55.1% and 53.8%, respectively ($p = 0.82$). Men more frequently exhibited multivessel CAD, whereas women more often had identifiable culprit lesions. In multivariable analysis, sex was not significantly associated with 30-day survival.

Conclusions: In this cohort of patients with angiographically confirmed obstructive CAD following OHCA, no statistically significant sex-related differences were observed in management, procedural success, 30-day survival, or neurological outcomes. Men exhibited more extensive CAD.

ANGINA PECTORIS AFTER PERCUTANEOUS CORONARY INTERVENTION: THE ROLE OF COMPLEX FUNCTIONAL TESTING

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Form: oral presentation, **theme:** cardiovascular interventions and cardiac arrest
Introduction: Percutaneous coronary intervention (PCI) represents the predominant modality of myocardial revascularization, improving prognosis in patients with acute coronary syndrome (ACS) and quality of life in those with chronic coronary syndrome (CCS). However, up to 50% of patients continue to experience angina pectoris (AP) following PCI (post-PCI AP).
Purpose: Persistent or recurrent AP after PCI has become a clinically relevant issue. Despite its frequency, many aspects remain insufficiently understood, and current guidelines provide limited recommendations regarding diagnosis, prevention, and management. Complex functional testing (CFT) may help identify underlying mechanisms in patients without significant epicardial stenosis and guide targeted therapy to attenuate symptoms and improve quality of life.
Methods: Patients presenting with AP in the absence of significant epicardial stenosis are evaluated using CFT. Microvascular function is assessed by continuous thermodilution, and coronary vasoreactivity is evaluated by intracoronary acetylcholine testing. There is a substudy of a national based CFT registry CoroPhys-CZECH, that was approved by the institutional Ethics Committee (EK-151.23/25); all patients provided written informed consent. The study was prospectively registered at ClinicalTrials.gov (NCT07103317).
Results: In January 2026, selective coronary angiography was performed in 210 patients in our center; 21 (10%) had a history of prior PCI. Among these post-PCI AP patients, repeat PCI was performed in 4 patients (19%), fractional flow reserve (FFR) assessment in 4 (19%), CFT in 1 (5%), and no further diagnostic or interventional procedure was undertaken in 12 patients (57%).
Since December 2024, CFT has been performed in 55 patients in our center, including 7 with prior PCI. Among these patients, vasospastic angina (VSA) was diagnosed in 5 cases, coronary microvascular dysfunction (CMD) in 1 case, and 1 patient showed no evidence of epicardial stenosis, VSA, or CMD. Pharmacological therapy was subsequently adjusted according to the identified endotype.
Conclusion: Recurrent angina after apparently successful PCI represents a clinically significant problem. CFT enables identification of the underlying mechanism of post-PCI AP and may facilitate individualized therapeutic strategies.

CARDIOVASCULAR TOXICITY AND HYPERTENSION CAUSED BY ANTI-CANCER THERAPIES

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Form: oral presentation, **theme:** heart failure and cardio-oncology
Cancer survival rates are improving as a result of novel targeted and immune therapies. However, this improved survival is at the risk of cardiovascular disease, in large part due to cardiovascular toxicity of oncology therapy. VEGF inhibitors (VEGFi) are anti-angiogenic and effective anti-cancer drugs. They are the cornerstone of treatment targeted against many solid organ malignancies and have prolonged cancer survival. However, these drugs have potent cardiovascular side effects including severe hypertension and cardiac dysfunction. According to the American College of Cardiology, hypertension is the most prevalent adverse event associated with VEGFi treatment. In addition to the morbidity and potential mortality directly attributable to the cardiovascular toxicity of these drugs, their presence often results in VEGFi dose-reduction and/or interruption or cessation of treatment, thus denying patients of optimal anti-cancer therapy. As well as concerns about acute unwanted VEGFi effects, the longer-term implications of cardiovascular toxicity become more relevant as patients treated for cancer live longer, receive drugs over longer periods and treatment is administered to patients with an increasingly large burden of co-morbidity. There are major gaps in the understanding of how VEGFi (and other anti-cancer drugs) cause hypertension. The molecular mechanisms remain unclear although abnormal NO signaling, endothelin-1, and rarefaction have been implicated. We showed that oxidative stress and RhoA/Rho kinase are also important. This presentation will provide new insights into the molecular and vascular mechanisms whereby anti-cancer drugs cause hypertension.

HIGH-SUCROSE DIET-INDUCED ARRHYTHMIA AND CARDIO-HEPATIC AXIS DYSFUNCTION ACROSS THE EJECTION FRACTION SPECTRUM OF HEART FAILURE

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Form: oral presentation, **theme:** arrhythmia and electrophysiology II
Metabolic syndrome (MetS) is a major contributor to cardiovascular morbidity, while metabolic dysfunction-associated steatotic liver disease (MASLD) has emerged as an important cardiovascular risk factor. Since circulating cardiac and metabolic biomarkers may provide mecha-

nistic insights into arrhythmogenesis and early myocardial injury associated with cardiac arrest, we investigated alterations in cardiac arrest-related blood biomarkers in a rat model of MetS induced by chronic high-sucrose consumption. Compared with controls, MetS rats exhibited significantly increased serum K^+ concentrations and reduced Mg^{2+} levels. Oxidative stress markers were markedly elevated in the serum as well as in cardiac and liver tissues, accompanied by depleted ATP levels. Serum concentrations of cardiac injury and neurohormonal biomarkers, including cardiac troponin T (TnT), cardiac troponin I (TnI), B-type natriuretic peptide (BNP), N-terminal pro-B-type natriuretic peptide (NT-proBNP), interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), neuropeptide Y (NPY), and homocysteine, were significantly increased in MetS rats. In contrast, tissue levels of TnT, TnI, BNP, NT-proBNP, cyclic adenosine monophosphate (cAMP), and cyclic guanosine monophosphate (cGMP) were significantly decreased in both the heart and liver. Electrocardiographic analysis demonstrated QT interval prolongation and reduced heart rate in MetS rats. Echocardiographic assessment further revealed increased left ventricular end-systolic volume (LVESV) and left ventricular end-diastolic volume (LVEDV), together with reduced left ventricular ejection fraction (LVEF), stroke volume, and cardiac output, indicating impaired cardiac function. Importantly, the elevations in circulating biomarkers paralleled the observed electrical and functional cardiac abnormalities, suggesting a close association between systemic biomarker alterations and the development of arrhythmogenic remodeling in MetS. Collectively, these findings demonstrate a mechanistic link between circulating biomarker dysregulation, hepatic and myocardial injury, oxidative stress, and neurohormonal activation, all of which may contribute to cardiac remodeling and increase susceptibility to arrhythmias in the setting of MetS.

INVASIVE RIGHT VENTRICULAR DP/DT AS A MARKER OF RIGHT VENTRICULAR CONTRACTILITY: RELATIONSHIP WITH PULMONARY ARTERY PRESSURE AND PAPI

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Form: oral presentation, **theme:** heart failure I
Background: Assessment of right ventricular (RV) function remains challenging, particularly under varying loading conditions. The maximal rate of RV pressure rise (dP/dt_max), derived directly from the invasive RV pressure waveform, reflects the rate of pressure generation during systole and may provide a direct measure of RV contractile performance. However, its relationship with established invasive hemodynamic indices of RV function remains incompletely characterized.
Purpose: To investigate the relationship between invasively measured RV dP/dt_max, pulmonary artery systolic pressure (PASP), and pulmonary artery pulsatility index (PAPi), and to determine whether these associations differ according to RV filling pressures.
Methods: Eighty-four patients undergoing invasive hemodynamic assessment were included. RV dP/dt_max, pulmonary artery systolic and diastolic pressures (PASP and PADP), right atrial pressure (RAP), and RV end-diastolic pressure (RVEDP) were obtained directly from invasive pressure tracings. PAPi was calculated as (PASP-PADP)/RAP. Associations between RV dP/dt_max and hemodynamic variables were assessed using Spearman correlation analysis. Prespecified subgroup analyses were performed according to RVEDP and RAP.
Results: Mean PASP was 40.9 ± 15.6 mmHg, PADP 20.1 ± 10.4 mmHg, RAP 8.45 ± 6.09mmHg, and RVEDP 9.82 ± 6.16 mmHg. RV dP/dt_max showed a significant positive correlation with PASP (Spearman r = 0.590, 95% CI 0.424–0.717; p <0.0001) and a weaker but significant correlation with PAPi (r = 0.248, 95% CI 0.029–0.444; p = 0.023). No significant association was observed between RV dP/dt_max and either RAP or RVEDP. In patients with RVEDP ≥9 mmHg the relationship between RV dP/dt_max and PAPi was markedly stronger (r = 0.634, 95% CI 0.404–0.788; p <0.0001), accompanied by a strong correlation with PASP (r = 0.785, p <0.0001). In contrast, among patients with RVEDP <9 mmHg, RV dP/dt_max remained correlated with PASP (r = 0.526, p = 0.0005), whereas no significant association with PAPi was observed.
Conclusions: Invasive RV dP/dt_max demonstrates a strong and consistent association with pulmonary artery systolic pressure, supporting its role as a marker of RV pressure-generating capacity and contractile performance. Its relationship with PAPi appears to depend on RV filling pressure, becoming particularly strong in patients with elevated RVEDP. These findings suggest that invasive RV dP/dt_max may provide direct information on RV contractile performance, while PAPi reflects the resulting interaction between RV contractility, pulmonary vascular load, and filling pressure. Further studies are warranted to determine the incremental clinical value of RV dP/dt_max over conventional invasive indices of RV function.

PREHOSPITAL TRIAGE WITH HIGH-SENSITIVITY POINT-OF-CARE TROPONIN FOR SUSPECTED NON-ST-ELEVATION ACUTE CORONARY SYNDROME: THE HARTC 2.0 RANDOMIZED TRIAL

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Form: oral presentation, **theme:** acute cardiovascular care

Background: Patients with suspected non-ST-elevation acute coronary syndrome (NSTE-ACS) frequently undergo hospital evaluation despite ACS being excluded in most cases. Prehospital point-of-care high-sensitivity cardiac troponin I (POC hs-cTnI) testing may improve risk stratification and reduce unnecessary hospital transport.
Purpose: To determine whether an integrated prehospital triage strategy combining the modified HEART score, POC hs-cTnI testing, and expert consultation increases home management after ambulance assessment compared with standard care.
Methods: HARTc 2.0 was a multicenter, open-label, randomized controlled trial in adults assessed by ambulance for suspected NSTE-ACS with symptom onset ≥2 h before first medical contact. Patients were assigned to a prehospital strategy using the modified HEART score with POC hs-cTnI or standard care using the HEAR score without prehospital troponin testing. The primary outcome was home management following ambulance assessment. Secondary outcomes included 30-day major adverse cardiac events (MACE), healthcare utilization, and within-trial costs.
Results: Among 1,368 analyzed patients, 727 were assigned to the intervention group and 641 to the control group. Home management occurred in 358/727 (49.2%) versus 265/641 (41.3%), an absolute difference of 7.9 percentage points (95% CI, 2.6–13.2); RR 1.19 (95% CI, 1.06–1.34; p = 0.003). Thirty-day MACE occurred in 61/727 (8.4%) and 52/641 (8.1%), respectively (absolute difference, 0.3 percentage points; 95% CI, –2.7 to 3.2; p = 0.852). Among patients managed at home, no MACE occurred in the intervention group versus 1 event in the control group. In the cost-analysis population, mean initial diagnostic-phase costs were €101 lower per participant in the intervention group (€1,739 vs €1,840; 95% CI, –€184 to –€20; p = 0.017), while total 30-day healthcare costs were not significantly different (€2,404 vs €2,575; difference –€171; 95% CI, –€550 to €199; p = 0.373). Additional prespecified analyses of healthcare utilization and secondary outcomes are ongoing and will be available at the time of presentation.
Conclusion: An integrated prehospital triage strategy using the modified HEART score, high-sensitivity POC troponin testing, and expert consultation increased home management without a significant difference in 30-day MACE. Initial diagnostic costs were lower, while total healthcare costs through 30 days were not significantly different.

EVOLVING PERSPECTIVES ON ARTIFICIAL INTELLIGENCE IN INTERVENTIONAL CARDIOLOGY: A FIVE-YEAR REPEATED ASSESSMENT

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Form: oral presentation, **theme:** cardiovascular interventions and cardiac arrest
Introduction: Artificial intelligence (AI) is increasingly studied in interventional cardiology, but clinical implementation remains limited. We compared AI-related knowledge, expectations, perceived applications, and barriers among the same interventional cardiologists in 2020 and 2025.
Methodology: Ten Dutch interventional cardiologists completed semi-structured interviews in 2020 and the same assessment items online in 2025. Assessments included self-rated AI knowledge, expectations of clinical impact, willingness to engage with AI, and perceived applications and implementation barriers. Because of the small sample size, analyses were descriptive and responses were summarized as counts and medians.
Results: Median self-rated AI knowledge increased from 2 to 3 on a 5-point scale. The proportion expecting AI to play a role in interventional cardiology increased from 6/10 to 9/10, while uncertainty decreased from 4/10 to 1/10. Expected short-term impact remained unchanged, whereas median expected impact at 5–10 years increased from 3 to 4 and at >10 years from 3.5 to 5. In both years, 9/10 respondents were willing to learn more about AI, contribute to model development, and use AI clinically. Reported barriers shifted from lack of confidence, limited generalizability, and costs in 2020 toward digital infrastructure, ethical/legal issues, and safety in 2025. Perceived clinical value also became more focused on selected applications such as lesion assessment, complication-risk prediction, stent selection, and procedural planning.
Conclusion: Within this small repeated cohort, respondents reported greater AI knowledge and confidence in its future role in 2025, while expectations became more differentiated and barriers more implementation-focused. These exploratory findings suggest a shift from broad expectations toward more targeted clinical applications and practical implementation concerns.

SODIUM-GLUCOSE COTRANSPORTER 2 INHIBITOR INITIATION IN SYSTEMIC RIGHT VENTRICULAR FAILURE: A PRE-POST ANALYSIS FROM THE INTERNATIONAL ACHIEVE-SGLT2I REGISTRY

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Form: oral presentation, **theme:** heart failure I
Background: Sodium-glucose cotransporter 2 inhibitor (SGLT2i) therapy is an established pillar in the management of left ventricular heart failure. Evidence on its clinical impact in adult congenital heart disease patients with a systemic right ventricle is limited.
Methods: Adult congenital heart disease patients from nine centers initiating SGLT2i therapy were enrolled in the ACHIEVE-SGLT2i registry (NCT06932081); data from those with a systemic right ventricle and heart failure were analyzed. The primary outcome was the change in heart failure events; longitudinal NT-proBNP levels, echocardiographic parameters, and safety were assessed.
Results: A total of 104 patients with a systemic right ventricle were included (mean age 47 years, 37% female); 61% transposition of the great arteries (TGA) after atrial switch and 39% congenitally corrected TGA. Median follow-up was 398 days (IQR 190–719). Comparing 12 months post- and pre-initiation periods, heart failure event rates reduced by 74% (IRR 0.26, 95% CI 0.08 to 0.93, $p = 0.038$). NT-proBNP levels decreased by 22% (95% CI 5% to 35%, $p = 0.013$) within 100 days, with apparent stabilization afterwards. A 2.4-fold higher odds of improved systolic right ventricular function was observed after initiation (OR 2.39, 95% CI 0.93 to 6.15, $p = 0.07$), although non-significant. Ten patients (10%) reported side effects; 7 (7%) discontinued therapy.
Conclusion: SGLT2i therapy appears safe and well tolerated. Initiation was associated with lower heart failure event rates and reduced NT-proBNP levels within the first 100 days. These findings support a role for SGLT2i in this population and warrant further evaluation of its effectiveness.

A NOVEL ECG MARKER TO DISTINGUISH PROXIMAL LEFT BUNDLE BRANCH BLOCK FROM INTRAVENTRICULAR CONDUCTION DELAY IN CANDIDATES FOR CARDIAC RESYNCHRONIZATION THERAPY

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Form: oral presentation, **theme:** arrhythmia and electrophysiology II
Introduction: Distinguishing proximal left bundle branch block (LBBB) from intraventricular conduction delay (IVCD) is essential for cardiac resynchronization therapy (CRT) candidate selection, but remains challenging using standard 12-lead ECG. Calculation of negative-derivative activation times (NDAT) enables non-invasive assessment of epicardial ventricular activation from conventional ECG signals. We hypothesised that NDAT may provide a practical marker for differentiating proximal LBBB from IVCD and aimed to assess its diagnostic performance in CRT candidates compared with Strauss criteria.
Methodology: Consecutive CRT candidates with non-RBBB morphology and QRS ≥130 ms from the multicenter UHF-BLOCK trial (NCT07057544) were included. Invasive electrophysiological study with left ventricular (LV) septal activation mapping was performed to classify conduction disorder as proximal LBBB or IVCD. NDATs were measured as the interval from the QRS onset to the steepest negative downslope of the QRS complex in leads V_1–V_8 during spontaneous rhythms. The presence of LBBB according to the Strauss criteria on standard 12-lead ECG was assessed by an independent investigator.
Results: Among 125 patients (84 male; LVEF 28 [23–34]%; QRS 167 ± 18 ms), invasive LV septal mapping identified proximal LBBB in 84 (67%) and IVCD in 41 (33%) cases. Patients with proximal LBBB presented with longer QRS duration than those with IVCD (175 ± 16 vs 153 ± 12 ms; $p < 0.001$). Strauss criteria differentiated proximal LBBB from IVCD with an AUC of 0.67, sensitivity of 98%, and specificity of 37%. A specific activation pattern was observed in patients with proximal LBBB and IVCD: NDATs in right ventricular and septal leads (V_1–V_4) were similar, while NDATs in leads associated with LV lateral wall activation (V_5–V_8) were more prolonged in patients with proximal LBBB than with IVCD. Receiver operating characteristic (ROC) analysis showed that NDAT in leads V_5, V_6, V_7, and V_8 all outperformed Strauss criteria for differentiating proximal LBBB from IVCD, with AUCs of 0.78, 0.93, 0.91, and 0.90 ($p = 0.03$ for V_5 and $p < 0.001$ for V_6–V_8 vs. Strauss criteria), optimal cutoffs of 89, 110, 116, and 122 ms, with sensitivity/specificity (se/sp) of 74/83%, 83/98%, 89/90%, and 88/85%, respectively.
Conclusion: NDAT in leads V_5–V_8 differentiates proximal LBBB from IVCD more accurately than the Strauss criteria and provides a clinically available tool to improve the results of CRT.