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Abstracts SSC/SSCS: Orals

Basel (Switzerland), June 10–12, 2026

JOINT ANNUAL MEETING OF THE SWISS SOCIETY OF CARDIOLOGY (SSC), THE SWISS SOCIETY FOR HEART AND THORACIC VASCULAR SURGERY (SSCS), THE SWISS SOCIETY OF PNEUMOLOGY (SSP) AND THE SWISS SOCIETY FOR THORACIC SURGERY (SSTS)

BASEL, JUNE 10–12, 2026

ORALS SSC AND SSCS

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The posters from this conference have been published in Supplementum 299 of the Swiss Medical Weekly (https://doi.org/10.57187/5577).

RAPID FIRE ABSTRACTS: HEART FAILURE

O001

Decoding Lung Fibroblast–Endothelial Dialogue Driving Pulmonary Hypertension in Cardiometabolic HFpEF

Marialucia Telesca[1], Valeria Masciovecchio[1], Iliana Papadopoulou[1], Shafeeq Mohammed[1], Era Gorica[1], Alessia Mongelli[1], Christian Matter[1], Francesco Paneni[1], Burkhard Ludewig[1], Frank Ruschitzka[1], Sarah Costantino[1]

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Introduction: Pulmonary hypertension (PH) is a common complication of heart failure with preserved ejection fraction (HFpEF), associated with worse clinical outcomes than HFpEF alone. Beyond hemodynamic stress, maladaptive remodeling of the lung microenvironment-driven by dysfunctional cellular populations may promote pulmonary microvascular injury and PH progression. This study aimed to characterize lung microenvironmental alterations in cardiometabolic HFpEF-associated PH (HFpEF-PH) and to assess whether targeting the c-Fos–Mediator axis could restore endothelial homeostasis.

Material & Methods: C57BL/6J mice were fed a control-diet (CD) or a high-fat diet combined with L-NAME for 21 weeks to induce HFpEF-PH. Cardiac and pulmonary function were evaluated by high-resolution echocardiography. Lung tissue underwent single-nucleus RNA sequencing (snRNA-seq). Primary pulmonary endothelial cells (mECs) and fibroblasts (mFBs) were isolated for molecular, metabolic, and autophagic profiling. Paracrine signaling was investigated using co-culture assays and Olink proteomic analysis of fibroblast-conditional media to define secreted mediators. Autophagic flux was assessed by transmission electron microscopy and protein analysis. The functional role of c-Fos was tested by siRNA-mediated silencing in mFBs.

Results: HFpEF mice developed PH, with elevated pulmonary pressures, right ventricular hypertrophy, and reduced pulmonary acceleration time. snRNA-seq revealed fibroblasts and endothelial cells as the most transcriptionally dysregulated populations, with expansion of activated myofibroblast subsets and upregulation of AP-1 complex genes (c-Fos, c-Jun). HFpEF fibroblasts exhibited a profibrotic, metabolically reprogrammed phenotype, while mECs displayed inflammatory activation and impaired autophagy. Proteomic analysis of HFpEF fibroblast secretomes identified key inflammatory and profibrotic mediators driving endothelial injury. Exposure of CD-mECs to HFpEF-conditioned media reproduced endothelial dysfunction, which was rescued by c-Fos silencing in mFBs-normalizing S100A4 expression and restoring autophagic flux.

Conclusion: Fibroblast-endothelial communication via secreted molecules drives microvascular dysfunction in HFpEF-PH. Targeting this signaling pathway and its downstream secreted effectors offers a novel therapeutic opportunity to prevent pulmonary vascular remodeling in HFpEF-PH.

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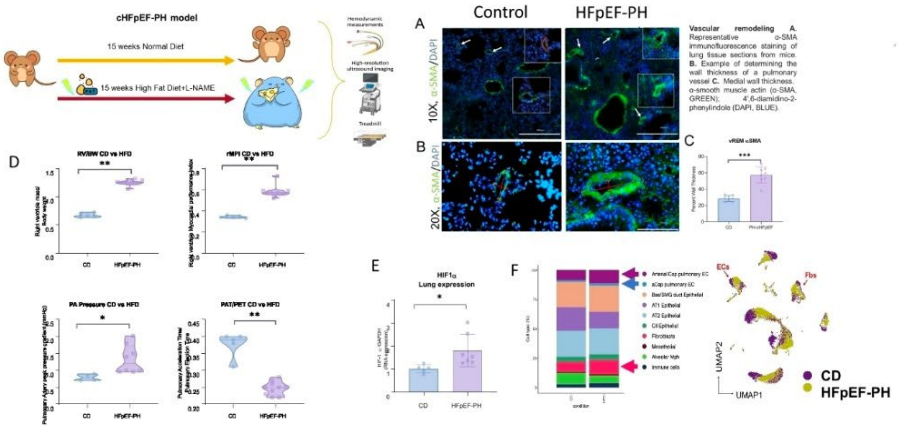

FIG.1 Schematic representation of the cardiometabolic HFpEF-PH mouse model. Mice were fed a normal chow diet (CD) or a high-fat diet combined with L-NAME (HFpEF-PH) for 15 weeks, followed by hemodynamic measurements, high-resolution echocardiography, and treadmill testing.(A-C) Representative immunofluorescence images of lung sections stained for α-smooth muscle actin (α-SMA, green) and DAPI (blue) showing increased pulmonary vessel wall thickness in HFpEF-PH compared to CD mice. Scale bars, 50 µm (10×) and 25 µm (20×).(D) Hemodynamic assessment demonstrating increased right ventricular hypertrophy (RV/LV+S ratio), elevated RV systolic pressure (RVSP), pulmonary arterial pressure, and prolonged PAT/PET ratio in HFpEF-PH mice. (E) qPCR analysis of lung tissue showing upregulation of HIF1α mRNA expression in HFpEF-PH mice compared to CD.(F) Single-cell RNA sequencing analysis of lung tissue highlighting distinct cellular clustering (UMAP) and relative cell-type distribution. Fibroblast (Fbs) and endothelial cell (EC) populations display marked transcriptomic shifts in HFpEF-PH versus CD conditions.*Data are presented as mean ± SEM. Statistical significance was determined by unpaired Student's t-test; *p < 0.05, **p < 0.01, **p < 0.001.

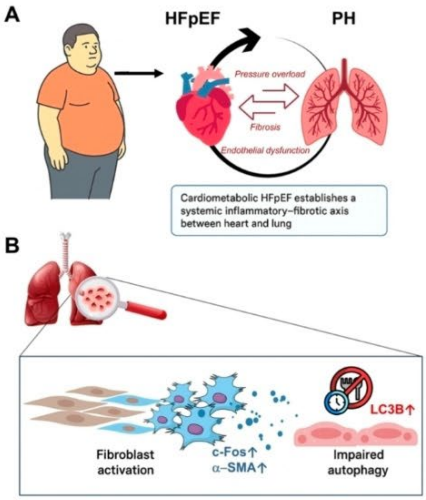

Fig.2.(A) *Cardiometabolic HFpEF — Heart and Lung as Active Players.* Schematic representation of the cardiometabolic HFpEF model showing the systemic inflammatory–fibrotic axis between heart and lung. Obesity and metabolic stress promote cardiac and pulmonary dysfunction, establishing cross-organ communication that contributes to pulmonary hypertension development. **(B)** *Fibroblast–Endothelial Interaction and Autophagy Impairment.* Illustration of fibroblast–endothelial cell crosstalk in the HFpEF lung microenvironment. Activated fibroblasts secrete inflammatory and profibrotic mediators that impair endothelial cell autophagy, promoting vascular remodeling and dysfunction.

O002

CSF2-Driven Myeloid Reprogramming Connects Metabolic Stress to Pulmonary Vascular Remodeling in HFpEF and PH

Valeria Masciovecchio[1], Marialucia Telesca[1], Iliana Papadopoulou[1], Yu-Jen Wang[1], Alessia Mongelli[1], Jing Liu[1], Pascale Enz[1], Shafeeq Mohammed[1], Christian Matter[1 2], Francesco Paneni[1 2], Burkhard Ludewig[1 3], Frank Ruschitzka[1 2], Sarah Costantino[1]

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Introduction: Pulmonary hypertension (PH) is a common and prognostically adverse complication in obese patients with heart failure with preserved ejection fraction (HFpEF). Cardiometabolic HFpEF-associated PH (PH-cHFpEF) is characterized by systemic inflammation and metabolic dysregulation. Alterations in pulmonary immune cell populations, particularly monocytes and macrophages, may amplify inflammation and promote maladaptive pulmonary vascular remodeling. However, the contribution of immune cell activation to PH-cHFpEF pathogenesis remains unclear.

Material & Methods: A murine model of PH-cHFpEF was established using high-fat diet combined with L-NAME treatment for 15 weeks. Right ventricular function and pulmonary pressures were assessed by echocardiography and invasive hemodynamics. Pulmonary immune cell populations were characterized by single-nucleus RNA sequencing (snRNA-seq). Primary mouse lung fibroblasts (mFbs) were isolated, and their secretome analyzed by proteomics. In vitro studies were conducted in THP-1 monocytes exposed to palmitic acid (PA).

Results: PH-cHFpEF mice developed elevated pulmonary pressures and right ventricular dysfunction. SnRNA-seq revealed expansion and transcriptional reprogramming of pulmonary immune cells. Patrolling monocytes (pMos) exhibited increased *Srgn* and *Dusp1* and reduced of *Dram1* expression, consistent with enhanced granule-mediated secretion. CD206^{+}MHC-II^{+} macrophages showed increased *Atf3* and reduced *Fkbp5* expression, suggesting activation of stress-resolution programs and heightened glucocorticoid sensitivity. PA induced a pro-inflammatory phenotype in THP-1 cells. Interactome analyses identified mFbs as the primary source of intercellular signaling in PH-cHFpEF lungs. Conditioned media from PH-cHFpEF mFb recapitulated macrophage activation in THP-1 cells, supporting a paracrine mechanism. Proteomics of mFb-conditioned media demonstrated increased secretion of CSF2, a cytokine that promotes pro-inflammatory myeloid differentiation, activates MAPK signaling, and suppresses autophagy.

Conclusion: mFb-derived CSF2 emerges as a central paracrine effector linking metabolic stress to myeloid dysfunction in PH-cHFpEF. CSF2-driven transcriptional programs promote pro-inflammatory activation, suppress autophagy, and alter tissue-repair responses, contributing to maladaptive pulmonary vascular remodeling. Targeting fibroblast–immune cell crosstalk may represent a novel therapeutic strategy in PH-cHFpEF.

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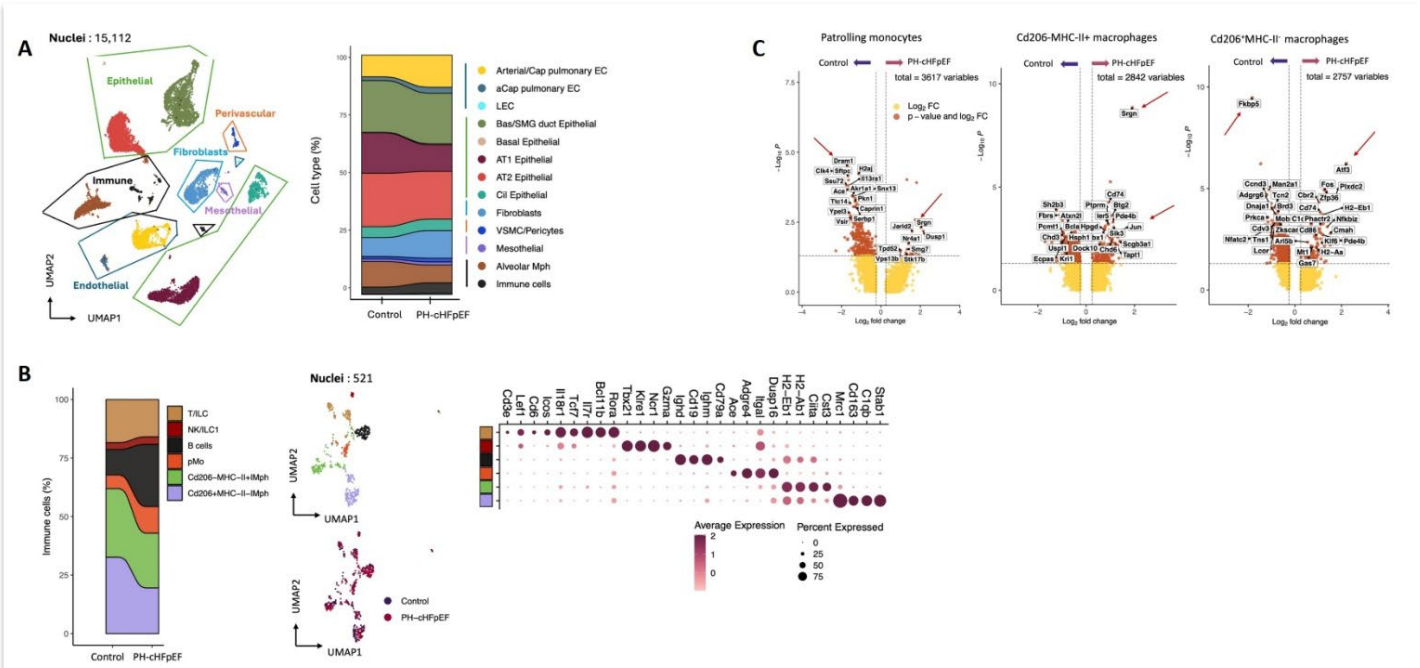

Figure 1. Molecular phenotyping of PH-cHFpEF and control lungs.

A) UMAP representation of pulmonary cells colored by cell type and clustering.

B) Immune cell clustering and marker genes of clustering.

C) Differentially expressed genes in patrolling monocytes, Cd206-MHC-II^{+} macrophages, and Cd206^{+}MHC-II^{-} macrophages.

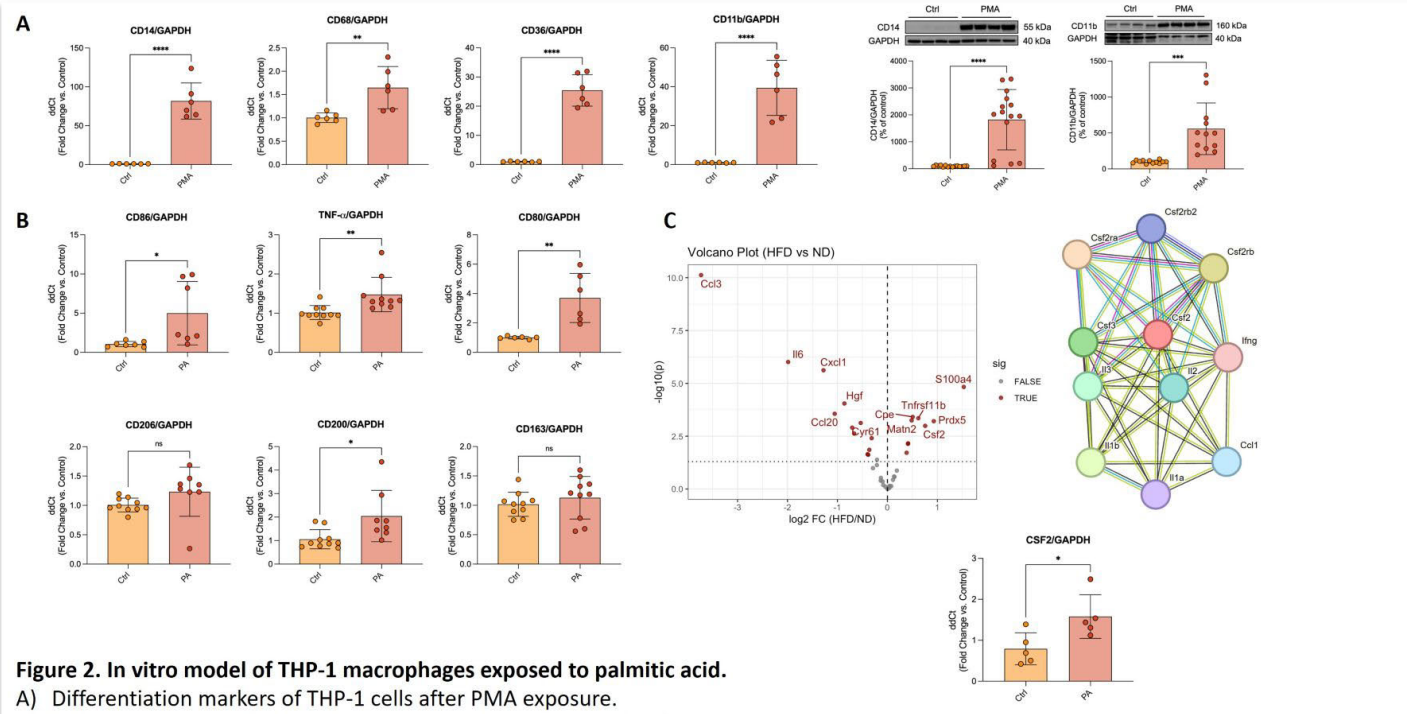

Figure 2. In vitro model of THP-1 macrophages exposed to palmitic acid.

A) Differentiation markers of THP-1 cells after PMA exposure.

B) Polarization of THP-1 after PA treatment mRNA expression levels.

C) Volcano plot of proteomic analysis of primary lung fibroblasts, Protein-Protein interaction and mRNA levels after PA treatment. Ctrl: control. PMA: phorbol myristate acetate. PA: palmitic acid.

O003

Real World Safety and Efficacy of Mavacamten for Hypertrophic Obstructive Cardiomyopathy: the SWISS-MAVA Cohort

Dennis Jensen[1], Niccolo Maurizi[2], Annina Vischer[3], Simon Stämpfli[4 5], Pierre Monney[2], Christiane Gruner[1]

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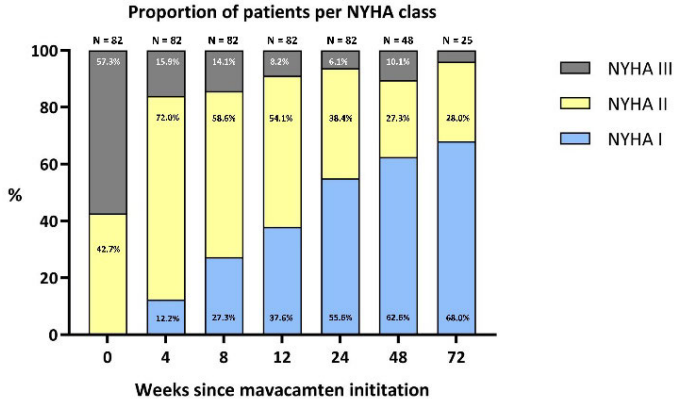
Introduction: Evidence of real-world use and challenges related to Mavacamten are much needed. The SWISS-MAVA Cohort was established to evaluate the real-world effectiveness and safety of mavacamten in the treatment of Obstructive Hypertrophic Cardiomyopathy (oHCM) in Switzerland.

Material & Methods: Consecutive patients with oHCM treated with Mavacamten across 4 Swiss referral centers were included. Clinical, echocardiographic, and laboratory data were collected at baseline and during follow-up. Primary outcomes included changes in New York Heart Association (NYHA) class and left ventricular outflow tract gradients (LVOTG). Secondary outcomes addressed the evolution of LV ejection fraction (LVEF), treatment discontinuation and new-onset atrial fibrillation (AF).

Results: Eighty-two patients (59.5±13.4 years, 37% female) were included with a mean follow-up of 52.4±21.4 weeks. At baseline, 47/82 patients (57%) were in NYHA class III. Sustained reductions from baseline to last follow-up occurred in LVOTG (resting -26.5 [IQR -8;-61]mmHg; Valsalva -61 [IQR -36.8;-95]mmHg), left atrial volume indexed (-7.5±4.9ml/m^2), maximal LV wall thickness (-2.4±1.7mm) and N-terminal pro B-type natriuretic peptide (Nt-proBNP, -1076 [IQR -1325.5,-208]ng/L). Mean LVEF decreased from 66.7% (baseline) to 62.5% (week 24) and remained stable (62.6%) up to last visit. At last follow-up, 65 (79.3%) had a Valsalva LVOTG <30mmHg and 71 (86.6%) improved by at least one NYHA class from baseline with a Valsalva LVOTG <50mmHg. Over 85.3 patient-years exposure, 5 patients (exposure-adjusted incidence: 5.9/100 patient-years) experienced transient reductions in LVEF to <50% resulting in temporary treatment interruption (all recovered LVEF of ≥50% and Nt-proBNP remained stable in 4/5 cases). Six patients developed new-onset AF (7.1/100 patient-years). No patients died.

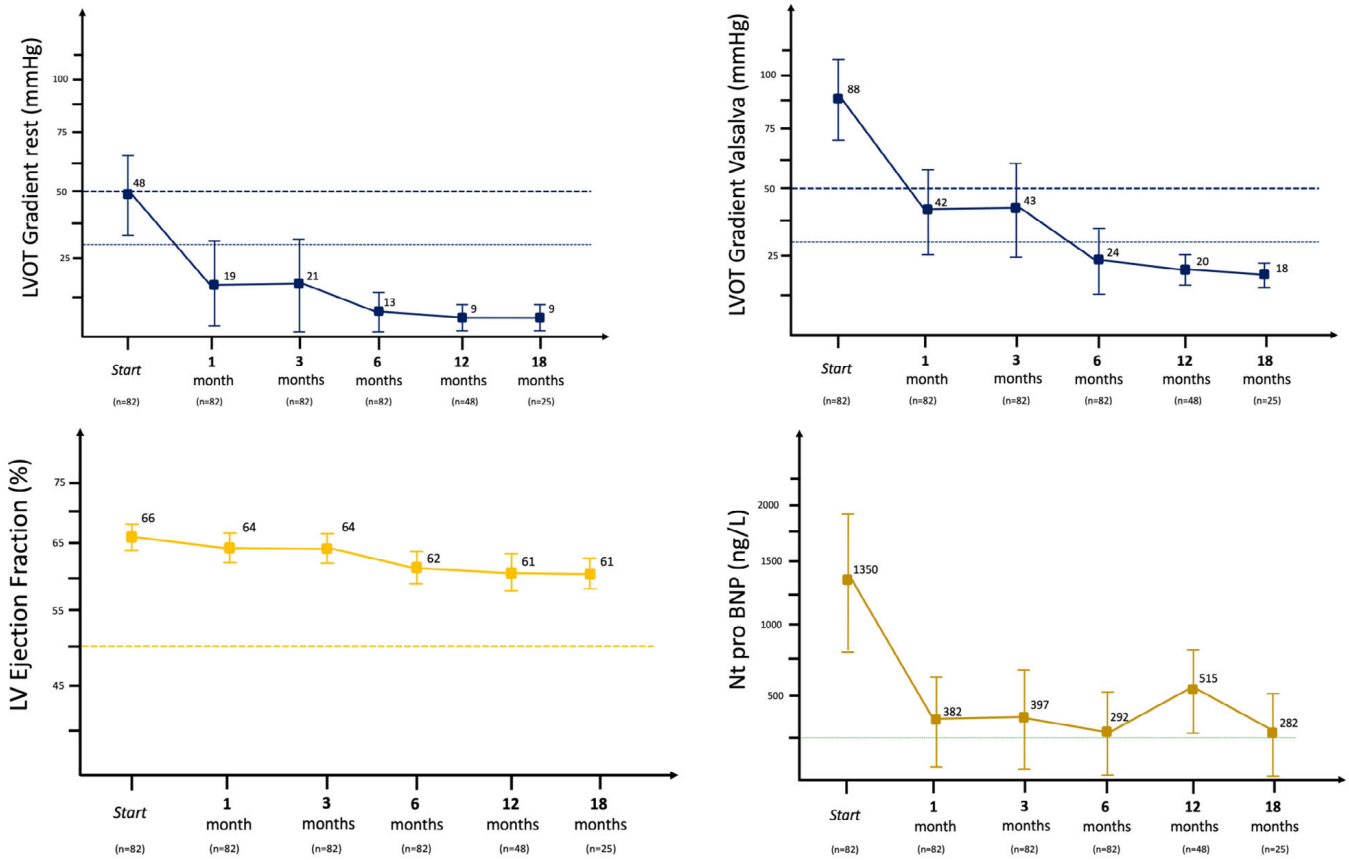
Conclusion: In this first European real-world cohort, mavacamten demonstrated sustained symptomatic and hemodynamic relief with an acceptable safety profile over >1 year follow-up. These findings support the effectiveness and feasibility of mavacamten therapy in routine clinical practice and complement contemporary trials evidence.

Fig. 1 Proportion of patients per NYHA class



COI: NM, SS and PM served as consultants for Bristol Myers Squibb. AV has received speaker and advisory board honoraria, as well as research and travel grants, from the following companies: Bristol Myers Squibb, Amarin, Servier, Medtronic, Vifor, NovoNordisk, Norvartis, AstraZeneca, MSD. All agreements are strictly between the University Hospital Basel and the respective companies. There are no personal contracts. All honoraria received are transferred directly to the Research Fund of the Medical Outpatient Department, University Hospital Basel. CG has received consulting/presenter fees from Bristol Myers Squibb, Cytokinetics, GE healthcare.

Fig. 2 Hemodynamic relief and biological effect of mavacamten therapy in the SWISS-MAVA Cohort



O004

Renin-Angiotensin-Aldosterone System Blockade in Patients with Chronic HeartFailure and End-Stage Kidney Disease : A Systematic Review and Meta-Analysis

Aarthiga Sritharan[1], Matteo Marchetti[1], Panagiotis Antiochos[1], Léa Iten[1], Menno Pruijm[2], Philippe Meyer[3], Thibaut Charlemagne[4], Baris Gencer[1], Olivier Muller[1], Peter Vollenweider[5], Patrick Yerly[1], Roger Hullin[1], Henri Lu[1]

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Introduction: Despite established benefits of renin–angiotensin–aldosterone system (RAAS) inhibition in heart failure with reduced ejection fraction (HFrEF), evidence in dialysis-dependent patients is limited. We performed a systematic review and meta-analysis to assess the efficacy and safety of angiotensin receptor–neprilysin inhibitors (ARNis) and angiotensin-converting enzyme inhibitors/angiotensin receptor blockers (ACEi/ARBs) in patients with chronic HFrEF undergoing maintenance dialysis.

Material & Methods: Outcomes of interest included all-cause mortality and cardiovascular mortality. Eligible studies included adult patients with chronic HFrEF on maintenance dialysis (hemodialysis and peritoneal dialysis), comparing ARNI versus non-ARNI regimens and ACEi/ARB versus regimens without ACEi/ARB. Literature searches were conducted in PubMed, EMBASE, Google Scholar, and Web of Science, covering the period from inception to September 2025. Hazard ratios (HRs) and their respective 95% confidence intervals (CIs) were pooled and meta-analyzed across studies.

Results: Six studies including dialysis-dependent patients with chronic HFrEF evaluated ARNIs (3,818 treated; 4,344 controls), and three studies evaluated ACEi/ARBs (3,293 treated; 1,982 controls). Use of ARNIs and ACEi/ARBs was associated with a significant reduction in all-cause mortality (HR 0.78, 95% CI 0.71–0.87 and HR 0.56, 95% CI 0.34–0.92, respectively; Figure 1). In contrast, neither ARNI nor ACEi/ARB therapy was associated with a statistically significant reduction in cardiovascular mortality (HR 0.84, 95% CI 0.57–1.24 and HR 0.57, 95% CI 0.32–1.02, respectively; Figure 2). Regarding safety, ARNI use

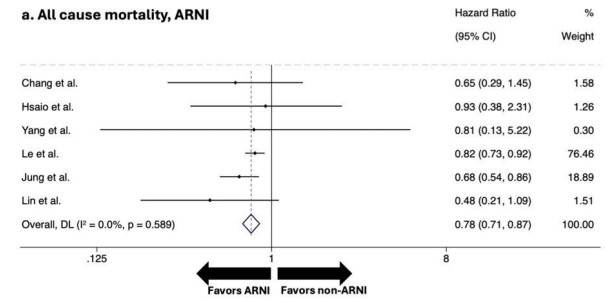
was associated with a lower incidence of hyperkalemia, with no significant difference in hypotension risk compared with non-ARNI regimens; safety data for ACEi/ARB therapy were unavailable.

Conclusion: ACEI/ARBs and ARNIs were associated with lower all-cause mortality in dialysis-dependent patients with HFrEF, despite no clear reduction in cardiovascular mortality. These findings suggest a potential overall benefit of RAAS inhibition in this population, although residual confounding cannot be excluded and underlying mechanisms remain uncertain.

COI: No

Figure 1. All-Cause Mortality Associated with Use of (a) ARNIs, (B) ACEis/ARBs, in HFrEF Patients Undergoing Maintenance Dialysis

Abbreviations: ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker ; ARNI= angiotensin receptor-neprilysin inhibitor; HFrEF = heart failure with reduced ejection fraction.



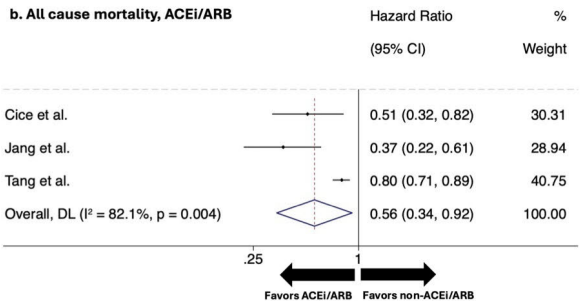
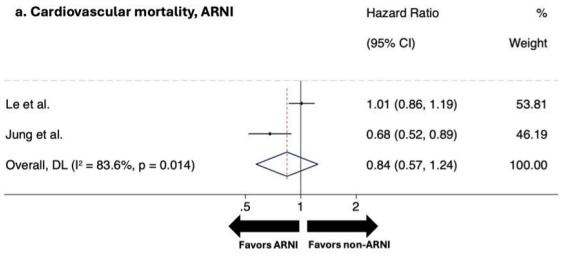
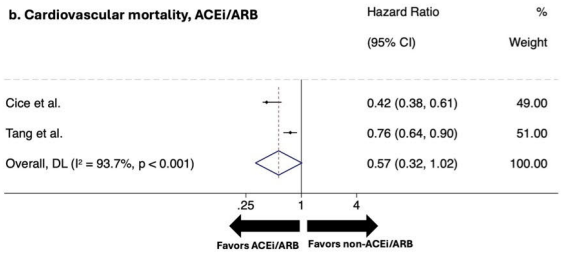


Figure 2. Cardiovascular Mortality Associated with Use of (a) ARNIs, (b) ACEis/ARBs, in HFrEF Patients Undergoing Maintenance Dialysis

Abbreviations: ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker ; ARNI = angiotensin receptor-neprilysin inhibitor; HFrEF = heart failure with reduced ejection fraction.





O005

Incremental prognostic value of four chamber strain for death in arrhythmogenic right ventricular cardiomyopathy: a practical stepwise approach

Rodrigo Carrasco[1], Thierry Donati[1], Qi Chen[1], Neria E. Winkler[1], Monika Hebeisen[1 2], Alexandros Protonotarios[3], Konstantinos Savvatis[3], Robert Bigseth[4], Paul A.S. Olsen[4], Firat Duru[1], Ardan Saguner[1], Perry Elliott[3], Kristina H. Haugaa[4], Felix C. Tanner[1]

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Introduction: Arrhythmogenic right ventricular cardiomyopathy (ARVC) is linked to progressive myocardial dysfunction and the risk of death or heart transplantation. We assessed the incremental prognostic value of left ventricular global longitudinal strain (LVGLS) and right (RAGLS) as well as left (LAGLS) atrial strain beyond right ventricular free wall strain (RVFWS) and developed a practical stepwise four-chamber strain approach for risk stratification.

Material & Methods: This retrospective study included patients fulfilling the 2010 Task Force Criteria for definite ARVC from three prospective registries. RVFWS, RAGLS, LVGLS, and LAGLS were measured from apical views. Chamber involvement was defined using international consensus-derived strain cut-offs. The primary outcome was death/heart transplantation. Associations with outcome were evaluated using age- and sex-adjusted Cox models. Incremental prognostic value of four-chamber strain was assessed by likelihood ratio tests. A stepwise strain-based decision algorithm incorporating 5-year event rates derived from Kaplan-Meier was constructed for classification into low- and high-risk groups.

Results: Among 243 ARVC patients (mean age 46.9±15.5 years; 56% male; median follow-up of 7.4 [4.0-10.6] years), all four strain parameters were significantly associated with death or heart transplantation in univariable Cox models: reduced LVGLS (HR 4.95, 95%CI 1.98-12.35, p <0.001), LAGLS (HR 7.58, 95%CI 2.74-21.00, p <0.001), RVFWS (HR 4.69, 95%CI 1.77-12.44, p = 0.002), and RAGLS (HR 4.21, 95%CI 1.91-9.28, p <0.001). Sequential addition of four-chamber strain progressively enhanced prediction by likelihood ratio tests with improved model fit: RVFWS (p = 0.0008), RAGLS (p = 0.012), LVGLS (p = 0.016), and LAGLS (p = 0.004), consistent with reductions in −log-likelihood and AIC. The stepwise algorithm was able to identify a high-risk group with markedly increased risk (HR 13.82, 95%CI 5.08–37.54; p <0.001; C-index = 0.79).

Conclusion: Four-chamber strain provides incremental prognostic value in ARVC, and its evaluation through a practical stepwise approach identifies patients at high risk of death/heart transplantation, supporting efficient risk stratification in routine clinical practice.

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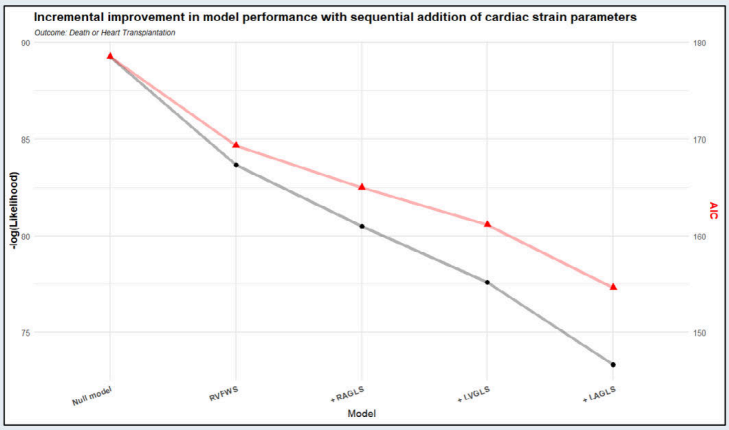
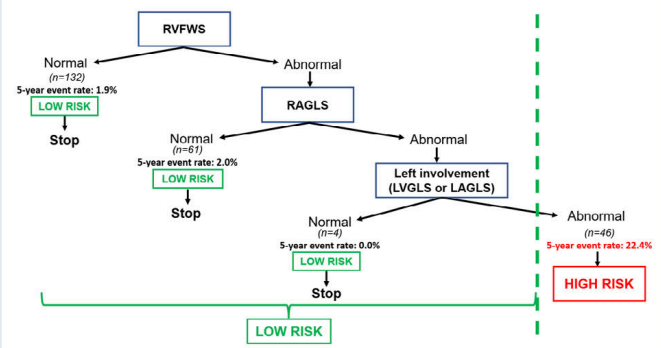
Figure 1. Incremental prognostic value of sequential addition of four-chamber strain parameters for death and heart transplantation.

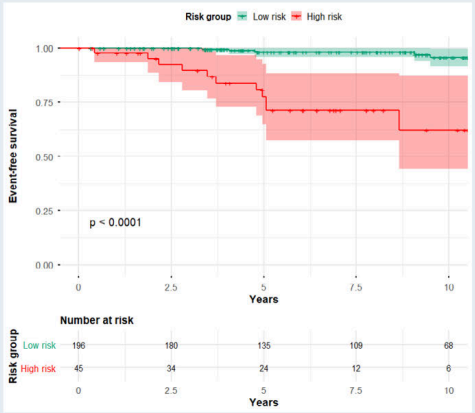


Black circles and lines represent –log-likelihood values, and red triangles and lines represent AIC (rescaled), across Cox models with sequential addition of RVFWS, RAGLS, LVGLS, and LAGLS. Progressive reductions in –log-likelihood and AIC indicate incremental improvement in prediction of death or heart transplantation, as confirmed by likelihood ratio tests.

Figure 2. Stepwise four-chamber strain-based risk stratification for death or heart transplantation risk in ARVC.



(A) Sequential assessment of RVFWS, RAGLS, and LVGLS/LAGLS using a strain-based decision algorithm. Terminal nodes show estimated 5-year event rates derived from Kaplan–Meier analysis.



(B) Kaplan–Meier curves for event-free survival according to strain-based risk classification. Patients classified as high risk showed significantly lower event-free survival compared with low-risk patients (log-rank p < 0.001).

O006

CMR-derived parameters increase H2FPEF Score diagnostic performance

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[1]Heart & Vessels Department, Cardiology Division, University Hospital of Lausanne, Lausanne, Switzerland, [2]Centre for Diagnosis and Treatment of Cardiomyopathies, Cardiovascular Department, Azienda Sanitaria Universitaria Giuliano-Isontina (ASUGI), University of Trieste, Trieste, Italy, Trieste, Italy, [3]Lausanne University Hospital (CHUV) and University of Lausanne (UNIL), Lausanne, Switzerland

Introduction: Heart failure with preserved ejection fraction (HFpEF) is a heterogeneous syndrome with challenging diagnosis. Current diagnostic scores mainly rely on clinical and echocardiographic parameters. We evaluated the diagnostic performance of H_2FPEF score after integration of cardiac magnetic resonance (CMR)-derived parameters

Material & Methods: From a retrospective cohort of 19,535 CMR scans performed at CHUV (2010-2024, Magnetom Siemens 1.5T-3T), we identified 613 patients with left ventricular ejection fraction (LVEF) >50%,calculable H_2FPEF score and echocardiography within ±3 months from CMR. Patients were classified as low–intermediate (0–5) or high probability (6–9). Clinical HF was identified using ICD codes. Univariable and multivariable logistic regression identified parameters associated with clinical HF.HF. Three models were evaluated: H_2FPEF score alone, H_2FPEF score plus native septal T1 at 1.5T, and H_2FPEF score plus mean extracellular volume (ECV) at 1.5T. Diagnostic performance was assessed using the area under the curve (AUC), with model comparison by DeLong test and Akaike information criterion.

Results: Among 613 patients, 67 had clinical HF. At univariable analysis, an H_2FPEF score ≥6 was associated with an eightfold higher likelihood of clinical HF (OR 8.10, p <0.001). LV morpho-functional parameters were not associated with HF. In those with calculable T1 mapping at 1.5T (n = 269), septal native T1 and ECV were independently associated with HF. Integration of mean ECV significantly improved diagnostic performance compared with H_2FPEF score alone (AUC 0.73 vs 0.63; p = 0.007,AIC: 192.6 vs 397.0), whereas native T1 did not.

Conclusion: In patients with CMR-LVEF >50% and high HFpEF likelihood (H_2FPEF >5), native septal T1 and mean ECV were independently associated with clinical HF. Integrating mean ECV into the H_2FPEF score improved diagnostic accuracy, supporting the role of CMR tissue characterization in refining noninvasive HFpEF diagnosis.

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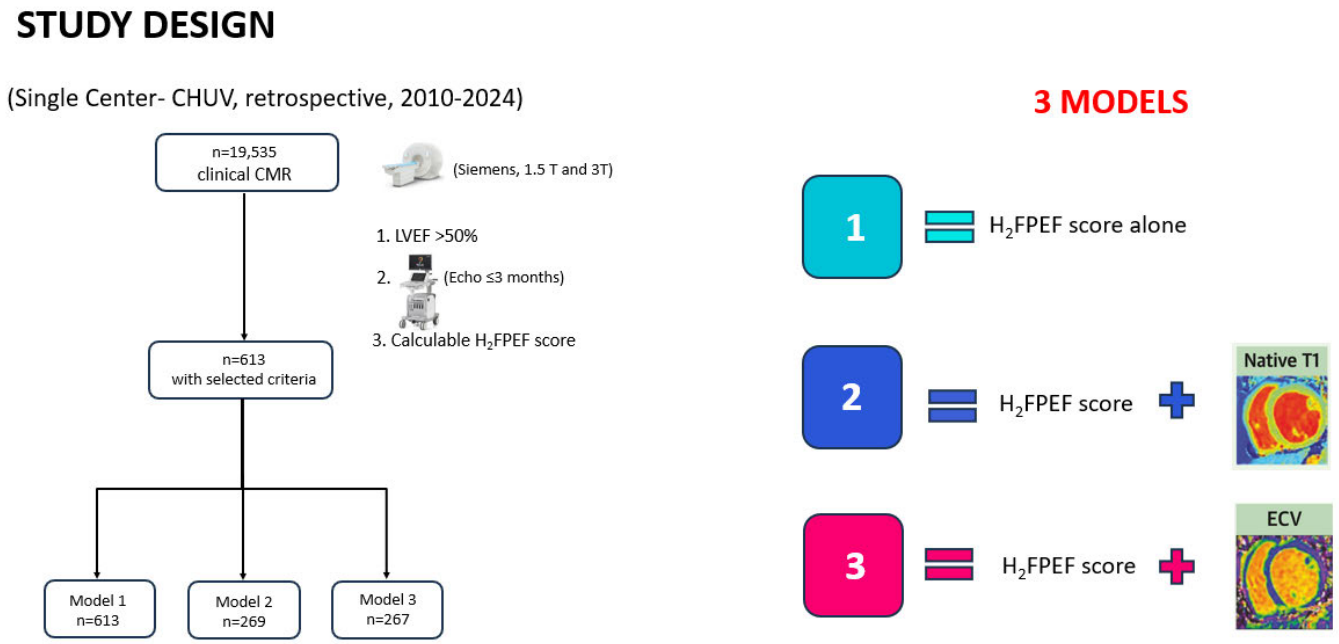

Table 1. Univariable and multivariable binary logistic regression analysis for prediction of clinical HFpEF

Variables	Univariable OR (95% CI)	p-value	Multivariable OR (95% CI)	p-value	Multivariable OR (95% CI)	p-value
H2PEF Score>5	**8.1 (4.0-16.2)**	**<0.001**	**16.1 (3.8-67.3)**	**<0.001**	**18.0 (4.4-73.2)**	**<0.001**
BMI>30	1.1 (0.6-2.1)	0.717				
AF	9.6 (4.7-19.4)	<0.001				
PASP >35 mmHg	5.5 (3.1-9.9)	<0.001				
HTN (2 or more drugs)	2.6 (1.5- 2.9)	<0.002				
E/e' >9	2.9 (1.3-3.9)	0.003				
LVSV (1 ml)	0.9 (0.9-1.0)	0.243				
LVSVi (1 ml)	0.9 (0.9-1.0)	0.619				
LVEDV (1 ml)	1.0 (0.9-1.0)	0.847				
LVEDVi (1 ml)	1.0 (0.9-1.0)	0.608				
LVESV	1.0 (0.9-1.0)	0.329				
LV mass/LVEDV	1.1 (0.6-2.1)	0.688				
Septal nT1 (10 ms)	**1.1 (1.0-1.2)**	**0.006**	**1.1 (1.0-1.2)**	**0.021**		
Lateral nT1 (10 ms)	1.0 (0.9-1.0)	0.910				
Mean nT1 (10 ms)	1.0 (0.9-1.0)	0.498				
Mean ECV (1%)	**1.1 (1.0-1.1)**	**0.011**			**1.0 (1.0-1.1)**	**0.010**

Abbreviation BMI Body Mass Index, AF Atrial fibrillation, PASP Pulmonary Artery Systolic Pressure, HTN hypertension, LVSV left ventricular stroke volume, LVSVi left ventricular stroke volume indexed, LVEDV left ventricular end-diastolic volume, LVEDVi left ventricular end-diastolic volume indexed, LVESV left ventricular systolic volume, LVESVi left ventricular systolic volume indexed, nT1 Native T1, ECV Extra Cellular Volume

Figure 1. Receiver-operating characteristic (ROC) curves for the clinical prediction of clinical HFpEF using different models.: Model 1 (H_2FPEF score alone); Model 2 (H_2FPEF score plus septal native T1); Model 3 (H_2FPEF score plus mean ECV) at 1.5T.

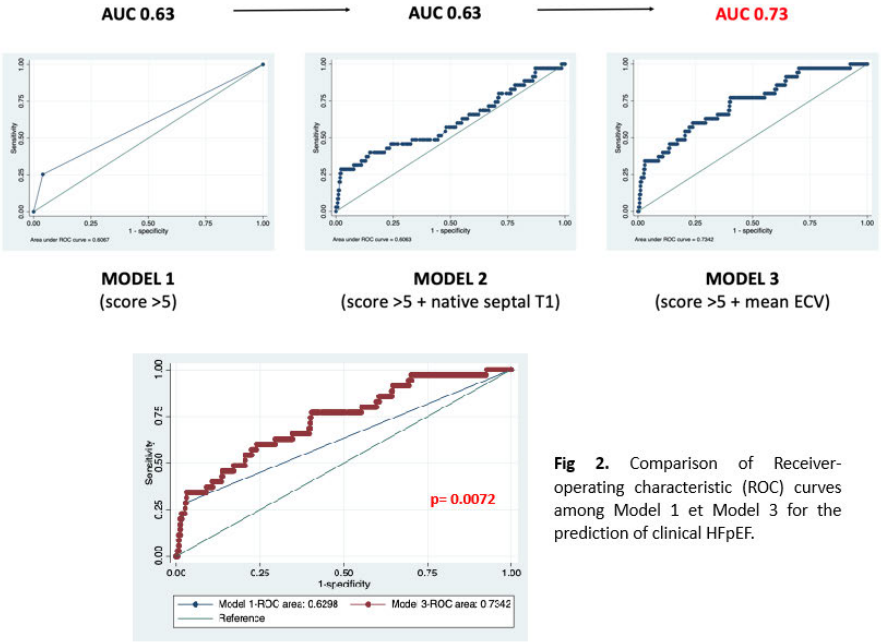

Fig 2. Comparison of Receiver-operating characteristic (ROC) curves among Model 1 et Model 3 for the prediction of clinical HFpEF.

RAPID FIRE ABSTRACTS: MYOCARDITIS & MECHANISMS IN HEART FAILURE

O007

Prognostic Factors and Impact of Immunosuppression Regimens in Giant Cell Myocarditis: A Systematic Review and Individual Patient Data Meta-Analysis

Alma Donlagic[1], Andy Geiser[1], Thanh-Danh Ly[1], Georgios Tzimas[1], Pierre Monney[1], Patrizio Pascale[1], Olivier Muller[1], Henri Lu[1], Panagiotis Antiochos[1]

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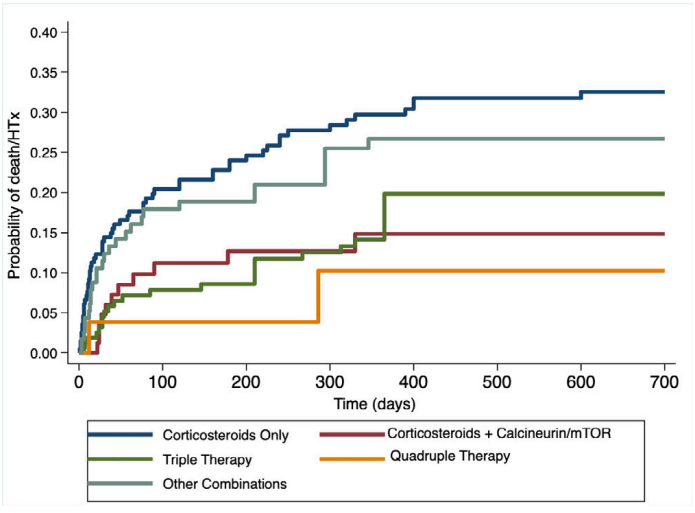
Introduction: Giant cell myocarditis (GCM) is a rare inflammatory cardiomyopathy. While historical data suggest a poor prognosis, contemporary predictors of death/heart transplantation (HTx) and the optimal immunosuppressive regimen remain unclear. We performed the largest individual patient data (IPD) meta-analysis to date to investigate both.

Material & Methods: We conducted a systematic review of all available published GCM cases to build a unified IPD database. The primary endpoint was a composite of death or HTx. Multivariable Cox regression with forward stepwise selection was utilized to identify independent predictors.

Results: We included 909 patients (49±16 years; 51% female). Over a mean follow-up of 20 months, 574 (63%) met the primary endpoint. Multivariable analysis identified a specific high-risk phenotype driven by autoimmune comorbidities (HR 1.46, p = 0.017), cardiogenic shock (HR 1.67, p = 0.010), and high-risk ECG features including LBBB (HR 1.91, p = 0.021), ST-elevation (HR 1.52, p = 0.013), or ST- depression (HR 3.36, p = 0.012). Notably, admission biomarkers (Troponin T, NT-proBNP) were not associated with outcomes, while higher baseline LVEF was protective (HR 0.98 per%, p = 0.003). Regarding management, the absence of immunosuppression was the strongest predictor of death or HTx (HR 2.12, p <0.001). We observed a distinct interaction with regimen composition: while triple-therapy (corticosteroids + calcineurin-inhibitors + anti-metabolites – such as azathioprine, mycophenolate) was associated with higher risk (HR 1.68, p = 0.008) compared to standard therapy (corticosteroids + calcineurin-inhibitors alone), quadruple regimens incorporating targeted biologic immunomodulators – such as muromonab – reversed this risk, conferring the highest transplant-free survival (HR 0.36, 95% CI 0.15–0.85, p = 0.020).



Conclusion: GCM remains a fulminant disease with high mortality and transplantation rates predicted by hemodynamic and electrical instability rather than biomarker elevation. Our data suggest that the integration of adjunctive biologic immunomodulators to standard treatment regimens is essential to improve transplant-free survival in GCM, a finding that warrants further study in prospective trials.

COI: No

O008

Atrial Function by Long-Axis Shortening Enhances Risk Stratification Beyond Traditional CMR Makers in Possible Myocarditis

Nicola Ciocca[1], Delia Brändle[1], Lars Riederer[1], Kady Fischer[1], Jonathan Schütze[1], Andreas Wahl[1], Giulin Tanner[1], Dominik P. Guensch[1], Kiril Stoyanov[1], Chrysoula Garefa[1], Maryam Pavlicek-Bahlo[1], Moritz Hundertmark[1], Isaac Shiri[1], Benedikt Bernhard[2], Raymond Y. Kwong[2], Christoph Gräni[1]

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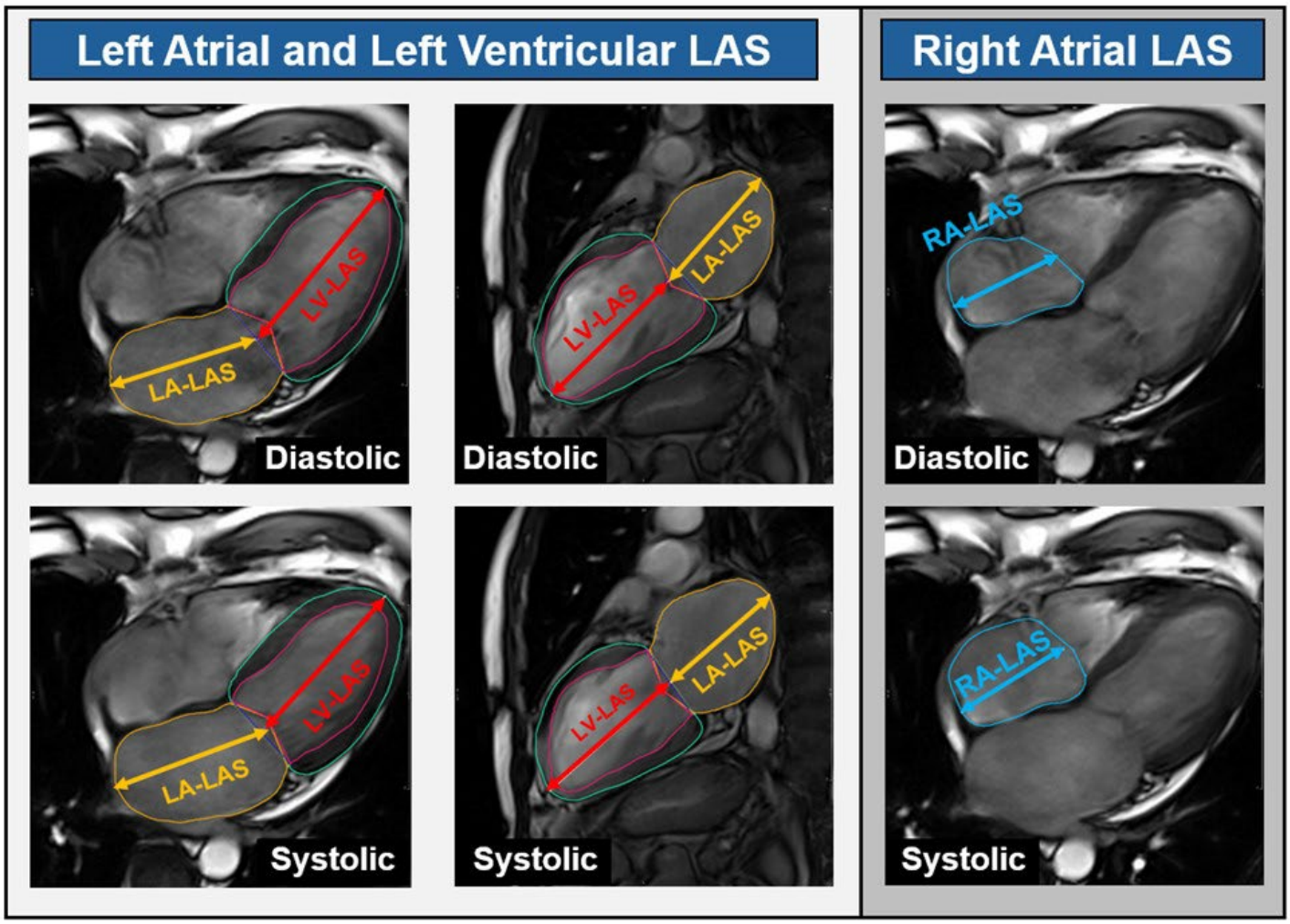
Introduction: Left ventricular ejection fraction (LVEF) and cardiac magnetic resonance (CMR) late gadolinium enhancement (LGE) are established risk markers for patients with myocarditis. Whether atrial functional impairment may occur in this clinical setting, and whether this finding offers incremental prognostic value, remains unknown. We aimed to evaluate the association of left/right- atrial long axis shortening (LAS) assessed in CMR with major cardiovascular adverse events (MACE) in patients with possible myocarditis.

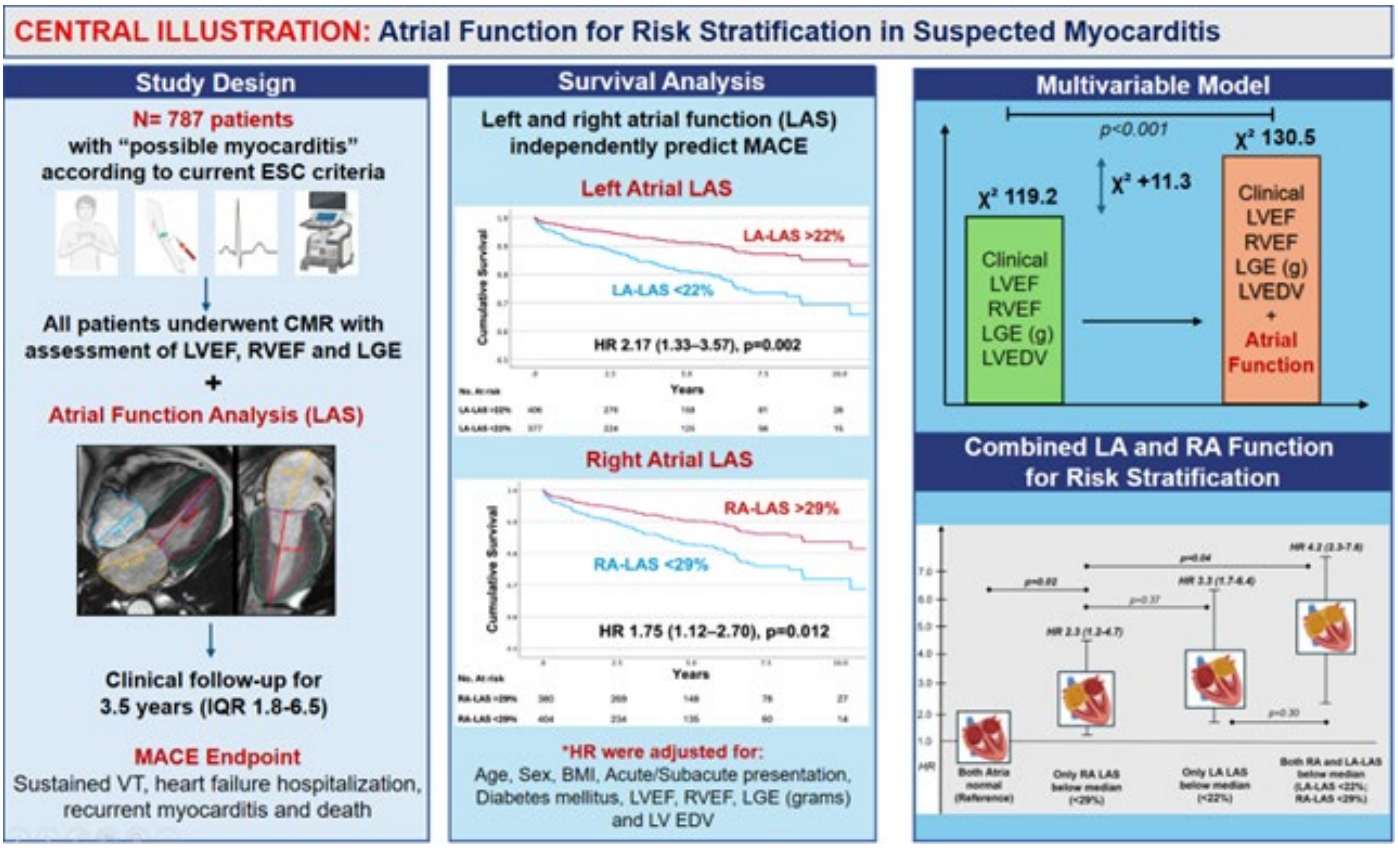
Material & Methods: Patients meeting 2025 ESC criteria for 'possible myocarditis' undergoing CMR at two tertiary centers without coronary artery disease or alternative diagnosis were included. LAS was associated with a composite of first MACE (heart failure hospitalization, sustained ventricular tachycardia, recurrent myocarditis, and all-cause mortality).

Results: A total of 787 patients (48±16years, 36% female) were included. Left atrial (LA)-LAS was 21.5±10.1% and right atrial (RA)-LAS 29.5±12.1%. After a median follow-up of 3.5 [1.8-6.5 years], 17.5% experienced a MACE. In the univariate analysis, both LA-LAS (per 5% decrease) (HR 1.37, 95% CI: 1.23-1.52; p <0.001) and RA-LAS (HR 1.27, 95% CI: 1.19-1.37; p <0.001), were associated with MACE. In multivariable models, LA-LAS below the median (22%) (HR 2.17,95% CI: 1.33-3.57; p = 0.002) and RA-LAS below the median (29%) (HR 1.75, 95% CI: 1.12-2.70; p = 0.012) both independently predicted MACE. Adding LA-LAS and RA-LAS to the baseline model (including clinical variables, LVEF, LVEDV and LGE) resulted in improved risk stratification for MACE (Chi² increase by 11.3, p <0.001; C-index from 0.72 to 0.75).

Conclusion: Atrial dysfunction can be present in possible myocarditis and atrial LAS was independently and incrementally associated with outcomes beyond traditional CMR markers and may refine future risk stratification in this clinical setting.

COI: No





O009

A metabolome-wide Mendelian randomization study reveals causal links of urinary 2-hydroxybutyrate/2-hydroxyisobutyrate with heart failure in patients with hypertension

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[1]*Shanghai Institute of Cardiovascular Diseases, Zhongshan Hospital and Institute of Biomedical Sciences, Fudan University, Shanghai, China*

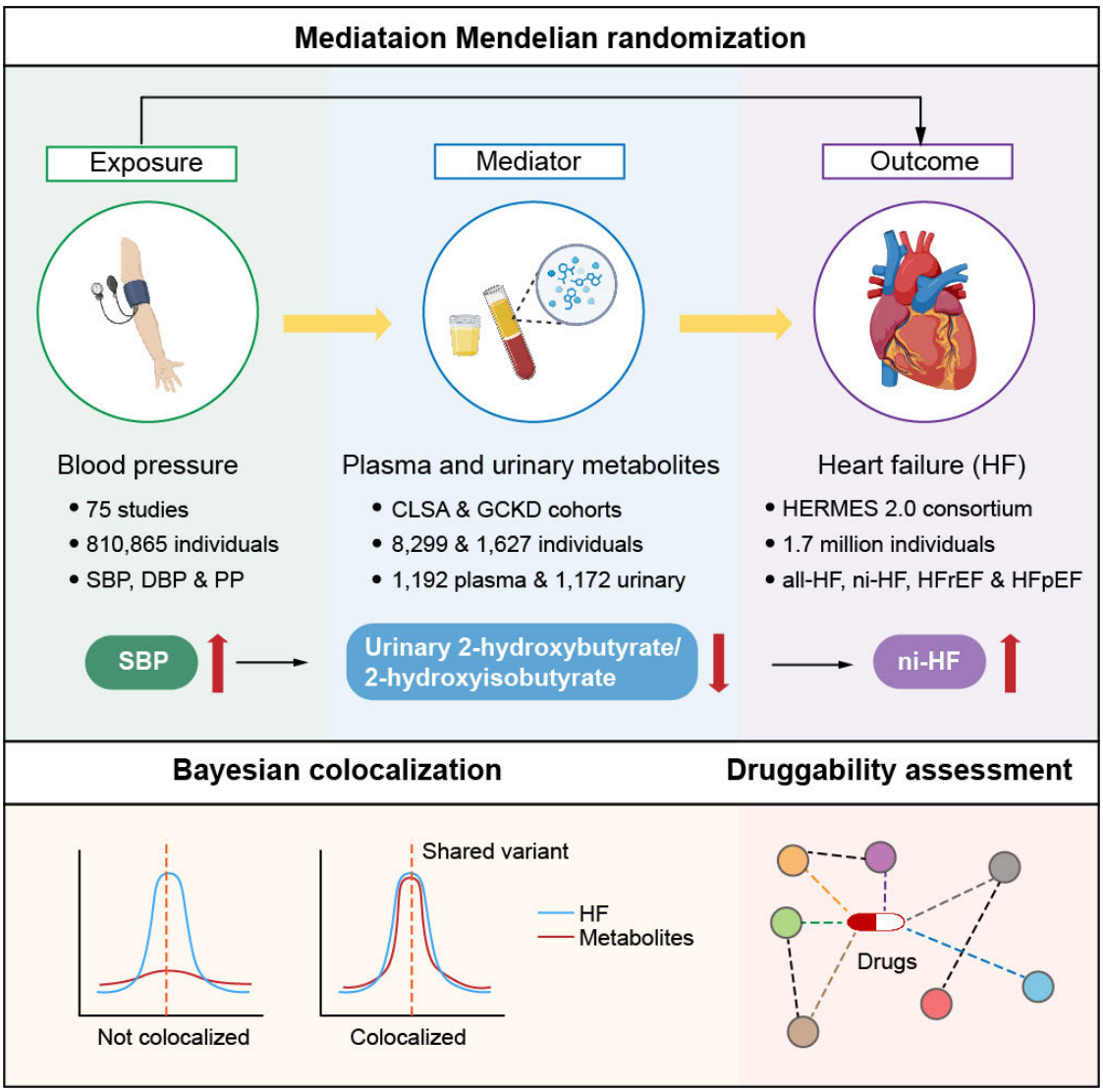
Introduction: Hypertension is a major risk factor for heart failure (HF), yet the metabolic mechanisms linking elevated blood pressure (BP) to HF remain poorly understood. This Mendelian randomization (MR) study aims to identify specific metabolic mediators that explain this causal link, with a focus on uncovering potential preventive targets.

Material & Methods: We employed a two-step MR framework to assess the mediating effects of plasma and urinary metabolites in the relationship between systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse pressure (PP), and all-cause heart failure, non-ischemic HF (ni-HF), heart failure with reduced ejection fraction (HFrEF), and heart failure with preserved ejection fraction (HFpEF), with CMR traits used to characterize cardiac remodeling. MR estimates, combined with Bayesian colocalization analysis, were used to prioritize metabolites linked to HF.

Results: Hypertension was associated with extensive metabolic reprogramming in plasma and urine, showing both shared and blood pressure (BP) trait-specific associations with metabolites. These metabolites were further associated with HF, revealing subtype-specific relationships and distinct metabolic profiles for HFrEF and HFpEF, with stronger associations observed in HFpEF. These HF-related metabolites were associated with left ventricular strain, often without corresponding changes in ejection fraction. By integrating MR and colocalization analyses, we established a tiered framework for metabolite prioritization for drug discovery. Mediation MR analyses revealed five negative mediators (e.g. hexadecanedioate (C16-DC)* and prolyglycine) and one positive mediator (2-hydroxybutyrate/2-hydroxyisobutyrate) linking elevated BP to HF in the tier 1 metabolites. Notably, urinary 2-hydroxybutyrate/2-hydroxyisobutyrate emerged as the strongest metabolic mediator, explaining 21.6% of the effect of SBP on ni-HF (P = 0.0195).

Conclusion: This mediation MR study identifies specific plasma and urinary metabolites as metabolic mediators linking hypertension to HF. Notably, urinary 2-hydroxybutyrate/2-hydroxyisobutyrate emerges as a novel mediator of ni-HF in patients with hypertension, offering promising insights for early preventive intervention strategies.

COI: No



O010

Prognostic value of artificial intelligence-derived echocardiographic measurements in cardiac amyloidosis

Amely Walser[1], Andreas Flammer[1], Moritz Hundertmark[2], Isaac Shiri[2], Rahel Schwotzer[3], Frank Ruschitzka[1], Felix C. Tanner[1], Christoph Gräni[2], Dominik C. Benz[1 4]

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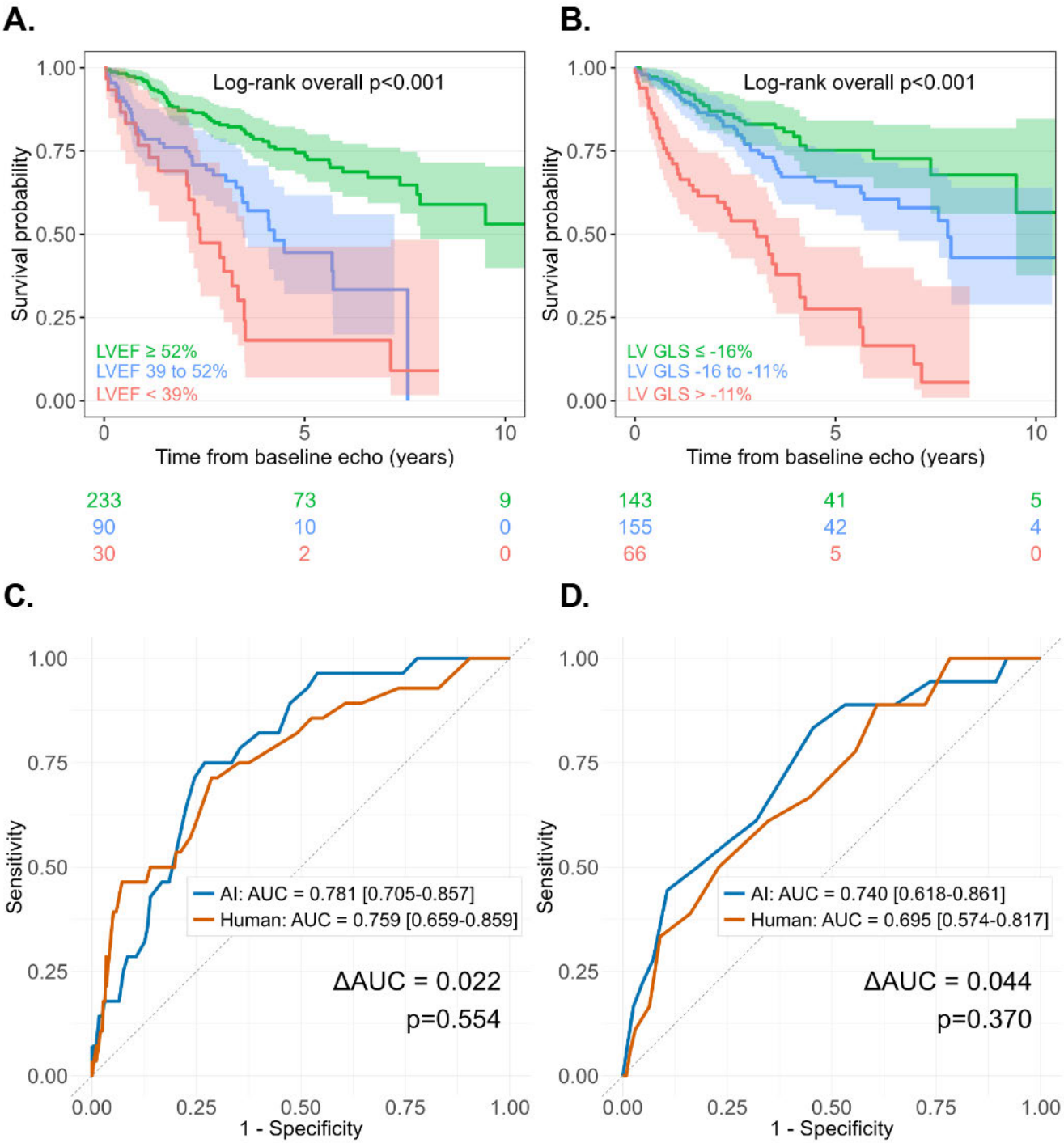
Introduction: Cardiac amyloidosis is a progressive and potentially fatal disease, requiring accurate risk stratification. Echocardiography is the first-line imaging modality, and AI-based tools are increasingly applied for automated analysis. However, the prognostic value of these measurements remains unclear.

Material & Methods: This retrospective cohort study included patients from two registries with transthyretin amyloid cardiomyopathy (ATTR-CM), immunoglobulin light-chain amyloid cardiomyopathy (AL-CM) and rare forms of cardiac amyloidosis. The AI-based software US2AI quantified fully automated interventricular septal thickness (IVSd), left ventricular ejection fraction (LVEF), and global longitudinal strain (LV-GLS) from baseline echocardiogram. Prognostic performance was assessed using Kaplan–Meier analysis and Cox regression along with ROC curves for 1-year mortality. Cutoffs stratifying abnormal values into mild and severe were determined by log-rank maximization. An echo staging was developed by combining LV-GLS and LVEF, stratifying patients into low (both normal), intermediate (one abnormal), and high risk (both abnormal).

Results: The cohort includes 434 patients (86% male, median age 76 years) with 347 (80%) ATTR-CM, 64 (15%) AL-CM and 24 (5%) rare forms. During follow-up of 2.9 [1.5–4.8] years, 147 patients (34%) died. In univariate Cox regression, AI-IVSd showed a prognostic trend (HR 1.05 [0.99-1.11], p = 0.077), while AI-LVEF (0.95 [0.93-0.96], p <0.001) and AI-LV-GLS (1.16 [1.10-1.21], p <0.001) provided significant prognostic stratification. Kaplan-Meier analysis stratified by optimal cutoffs confirmed discrimination across three risk groups (overall p <0.001; Panels A-B). Echo staging stratified risk with HRs of 2.36 and 4.38 (intermediate and high vs. low; p <0.001) and provided incremental value over NAC staging in ATTR-CM (χ^2 59 vs. 46, p <0.001). One-year mortality prediction did not differ between AI and human measurements for IVSd (p = 0.775), LVEF (p = 0.554; Panel C) and LV-GLS (p = 0.370; Panel D).

Conclusion: AI-derived echocardiographic measurements provide robust prognostic stratification in cardiac amyloidosis, with performance comparable to manual measurements, supporting their clinical utility for risk stratification.

COI: No



Kaplan-Meier survival curves for AI-derived LVEF (**Panel A**) and LV-GLS (**Panel B**). Receiveroperating curve (ROC) with area under the curve (AUC) comparing AI-derived versus human measured LVEF (**Panel C**) and LV-GLS (**Panel D**) for 1-year mortality prediction.

O011

Identification of Vascular Phenotypes in Heart Failure by Unsupervised Hierarchical Clustering using Retinal Vessel Analysis

Philip Sommer[1], Konstantinos Bitos[1], Flavia Piciacchia[1], Natallia Laptseva[1], Matthias Nägele[1 2], Frank Ruschitzka[1], Isabella Sudano[1], Andreas Flammer[1]

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Introduction: Unsupervised hierarchical clustering classifies patients based on similarity and may allow more precise differentiation of heart failure (HF) patients. Vascular function is a paramount aspect in HF and target of different therapeutic strategies, e.g. Renin-Angiotensin-Aldosterone system (RAAS) inhibition. Despite its important role, vascular function has never been used for HF phenotyping. This explorative study aimed to identify vascular phenotypes in HF patients.

Material & Methods: In this observational study program, HF patients of different etiologies were prospectively enrolled. Vascular function was assessed using retinal vessel analysis (RVA), flow-mediated dilatation (FMD), pulse-wave-velocity (PWV) and pulse-wave-analysis (PWA). Additionally, echocardiographic and laboratory parameters were collected. For the determination of vascular phenotypes an unsupervised hierarchical clustering was performed using 7 vascular variables. The number of clusters was determined via silhouette-score and model stability was assessed by bootstrap-validation.

Results: In total 306 HF patients were included. The hierarchical clustering identified two vascular HF phenotypes (HFPT-1; HFPT-2). HFPT-1 was characterized by significantly better microvascular retinal function shown by higher flicker-light-induced arterial dilatation [FIDart] HFPT-1: 3.02 ± 2.5 vs. HFPT-2: 0.91 ± 1.1; p <0.001 and venous dilatation [FIDven] HFPT-1: 3.81 ± 1.7 vs. HFPT-2: 2.61 ± 1.7; p <0.001). On the contrary, HFPT-1 had significantly higher PWV (HFPT-1: 10.3 ± 2.6 vs. HFPT-2: 8.17 ± 2.5). No difference was found for FMD. LVEF was significantly lower in HFPT-2 (HFPT-1: 45.1 ± 15.0 vs. HFPT-2: 34.5 ± 14.9, p <0.001) and inflammation more pronounced (hsCRP HFPT-1: 1.8 (8.8) vs. 3.5 (5.4), p = 0.01). Details shown in table 1.

Conclusion: Unsupervised hierarchical clustering identified 2 vascular HF-phenotypes. HFPT-1 was characterized by increased arterial stiffness and was more frequently associated with preserved/mildly-reduced LVEF, whereas HFPT-2 was characterized by impaired microvascular function and was associated with reduced LVEF and higher inflammatory activity.

COI: No

	HFPT-1 (n=75)	HFPT-2 (n=231)	p-value
Age, years	63.2 ± 11.7	63.6 ± 12.6	ns
BMI, kg/m²	28.1 ± 5.4	27.4 ± 5.4	ns
SBP, mmHg	132.6 ± 20.3	111.2 ± 18.6	<0.001
DBP, mmHg	82.5 ± 11.4	68.5 ± 11.0	<0.001
Female patients, N (%)	17 (22.7)	53 (22.9)	ns
Hypertension, N (%)	42 (56)	111 (48.3)	ns
Dyslipidemia, N (%)	29 (40)	101 (44)	ns
LVEF, % ± SD	45.1 ± 15.0	34.5 ± 14.9	<0.001
EDVI, ml/m²	66.8 ± 32.1	89.1 ± 41.8	<0.001
E-E' -Ratio	12.6 ± 6.4	15.3 ± 9.3	0.03
NTproBNP, ng/l (IQR)	948.5 (1618.75)	1060.0 (2149,5)	ns
hsCRP, mg/dl (IQR)	1.8 (8.8)	3.5 (5.4)	0.01
hsTroponin, ng/l ± SD	46.7 ± 63.3	59.6 ± 148.7	ns
FIDart, %	3.02 ± 2.5	0.91 ± 1.1	<0.001
FIDven, %	3.81 ± 1.7	2.61 ± 1.7	<0.001
AVR	0.82 ± 0.07	0.86 ± 0.08	<0.001
PWV, m/s	10.3 ± 2.6	8.17 ± 2.5	<0.001
FMD, %	4.93 ± 3.0	4.99 ± 3.3	ns

Table 1: Comparison of study characteristics between the vascular phenotypes. Normally distributed variables are shown as mean ± SD. Non-parametric variables are shown as median and corresponding Interquartilrange (IQR). Significance testing was carried out with Student's t-test or Mann-Whitney -U test where appropriate. LVEF – left ventricular ejection fraction; EDVI – end diastolic volume index; FIDart_ flicker light induced arterial dilatation; FIDven – flicker light induced venous dilatation; AVR – arterio venous ratio; PWV – pulse wave velocity; FMD – flow mediated dilatation.

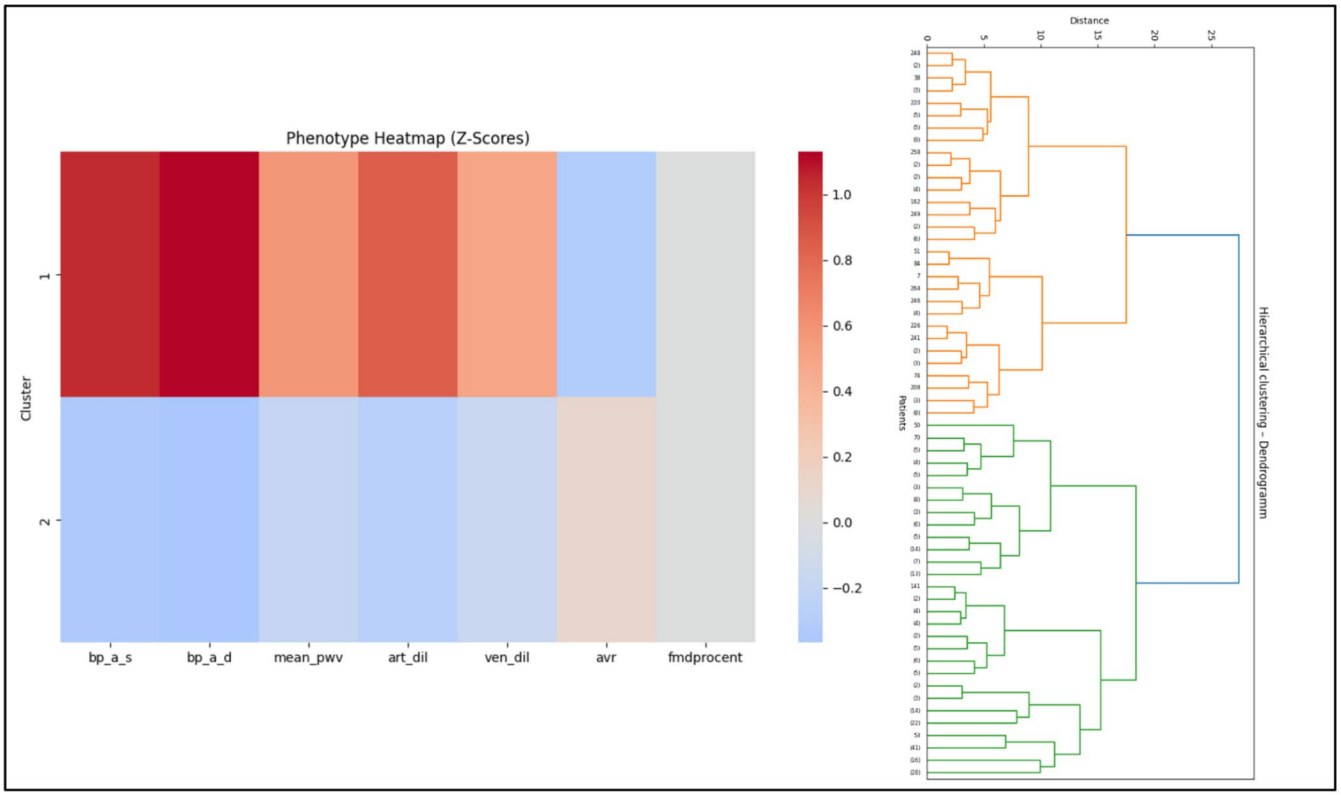

Figure 1: Heat map of hierarchical clustering. Included parameters are aortic blood pressure systolic/diastolic (bp_a_s/bp_a_d), PWV, FIDart (art_dil), FIDven (ven_dil), arterio-venous-ratio (AVR) and FMD (fmdprocent). Adjusted-Rand-Index (ARI) 0.54 indicating good model stability.

RAPID FIRE ABSTRACTS: RHYTHM DISORDERS

O012

Geographic and Community Responder Determinants of Return of Spontaneous Circulation in Out-of-Hospital Cardiac Arrest With Shockable Rhythm: A Nationwide Swiss Study

Maria Luce Caputo[1], Tommaso Scquizzato[2], Valentina Guerini Giusteri[1], Roman Burkart[3], André Wilmes[3], Ruggero Cresta[4], Claudio Benvenuti[4], Marco Bergonti[1], Tardu Özkartal[1], Catherine Klersy[5], Giulio Conte[1]

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Introduction: Out-of-hospital cardiac arrest (OHCA) remains associated with substantial morbidity and mortality, with wide variability in outcomes, even among bystander-witnessed OHCA with an initial shockable rhythm (Utstein comparator). Geographic and population-related factors may contribute to these disparities. To date, the impact of geographic and population-based factors on ROSC probability in OHCA has not been systematically evaluated at a national level in Switzerland.

The aim of this study was to assess the association between community responder network characteristics, geographic setting, and population density with ROSC achievement in Utstein comparator group.

Material & Methods: All consecutive OHCAs of presumed medical origin, witnessed by bystanders and presenting with an initial shockable rhythm (Utstein comparator group), recorded in the Swiss Registry of Cardiac Arrest (SWISSRECA) between January 1, 2019, and December 31, 2024, were included. According to arrest location, cases were classified into rural, intermediate, or urban settings based on geographic and population density criteria. Variables known to influence OHCA outcomes were analyzed using univariate and multivariable logistic regression.

Results: During the study period, 5,752 OHCA met Utstein comparator criteria. ROSC was achieved in 2,600 patients (49%). Median age was 67 years (IQR 58–77), 83% were male, and 49% of arrests occurred at home. ROSC probability was significantly lower in rural areas (36%; OR 0.67, 95% CI 0.54–0.82; p <0.001) compared with intermediate (46%) and urban settings (54%; OR 1.39, 95% CI 1.18–1.63; p <0.001). Nighttime arrest, EMS-initiated CPR, and refractory ventricular arrhythmias were negatively associated with ROSC, whereas first defibrillation delivered by a bystander was positively associated.

Conclusion: ROSC rates among OHCA with an initial shockable rhythm are high; however, significant geographic disparities persist. Lower ROSC probability in rural areas and during nighttime highlights the need to strengthen community responder networks, particularly in geographically disadvantaged regions, to improve outcomes.

COI: No

O013

Long-term outcomes after atrial appendage closure in frail and non-frail patients: A nationwide cohort study

Caroline Kleinecke[1], Cindy Bannwart[2], Rahel Laager[3], Steffen Gloekler[4], Alexander Kutz[5]

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Introduction: Left atrial appendage closure (LAAC) is a non-pharmacological option for stroke prevention in patients with atrial fibrillation (AF) who have contraindications to oral anticoagulation. Frailty is common in this population and may substantially influence patient outcomes. We examined frailty-associated differences in patient characteristics and long-term outcomes following LAAC in a nationwide Swiss cohort.

Material & Methods: We conducted a nationwide retrospective cohort study using administrative data of patients with AF undergoing LAAC between 2012 and 2023. Frailty was defined using a validated claims-based frailty index. Main outcomes were stroke, major bleeding, and all-cause mortality.

Results: Among 3,801 patients (34.1% women; median age, 78 years [IQR 72–82]; mean CHA_2DS_2-VASc score, 3.1±1.4), 547 (14.4%) were classified as frail. Frail patients were more often aged ≥75 years (70.7% vs 61.1% in non-frail patients) and had a higher prevalence of comorbidities, including prior stroke (10.2% vs 5.0%), chronic heart failure (37.7% vs 19.4%), and chronic kidney disease (55.4% vs 28.3%) (all p <0.001). In-hospital mortality was higher in frail than non-frail patients (4.2% vs 0.4%; p <0.01). Over a mean follow-up of 4.3 years (16,344 patient-years [py]), frail patients had higher incidence rates of stroke (6.58 vs 1.57 per 100 py), major bleeding (11.68 vs 5.02 per 100 py), and all-cause mortality (12.63 vs 6.97 per 100 py) (all p <0.001). One- and five-year cumulative incidences of stroke and major bleeding were consistently higher among frail patients.

Conclusion: In this nationwide cohort study, frailty in patients undergoing LAAC was associated with higher in-hospital mortality and substantially increased long-term risks of stroke, major bleeding, and all-cause death. Incorporating frailty assessment into clinical decision-making may improve risk stratification and inform tailored peri- and postprocedural management.

COI: No

O014

Cardiac Computed Tomography and Three-dimensional Transesophageal Echocardiography for Morphological Characterization of the Left Atrial Appendage

Marco Gamardella[1], Raouf Madhkour[1], Nicolas Brugger[1], Victor Kjær Eskildsen[2], Tommaso Bini[1], Antanas Gasys[1], Stefania Quintiliani[1], Alan Peters[3], Georgios Siontis[1], Roberto Galea[1], Christoph Gräni[1], Lorenz Räber[1]

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Introduction: Accurate preprocedural imaging of the left atrial appendage (LAA) is essential for procedural feasibility and optimal device sizing in percutaneous LAA closure (LAAC). Cardiac computed tomography (CT) and three-dimensional transesophageal echocardiography (3D-TEE) are the most commonly used modalities, yet systematic and comprehensive comparisons remain limited.

Material & Methods: We included 391 consecutive patients from the Bern LAAC registry who underwent preprocedural cardiac CT and 3D-TEE. LAA evaluation included ostium and landing zone (LZ) measurements, detection of trabeculations <10 mm in both width and depth), accessory lobes (≥10 mm in either dimension) and working depth. Device size prediction for Amulet was assessed according to manufacturer Instructions for Use. LAA contractility was evaluated on 3D-TEE and analyzed as a potential contributor to sizing discrepancies.

Results: Compared with CT, 3D-TEE yielded systematically smaller LAA measurements, and the magnitude of underestimation was more pronounced at the LZ than at the ostium (maximum diameter mean bias −1.81 mm vs −0.42 mm, respectively; p <0.01). CT detected more accessory lobes: 3D-TEE correctly identified 54.2%, misclassified 26.4% as trabeculations, and missed 19.4%. Working depth showed good agreement between modalities (mean bias +0.16 mm; p = 0.157).

Amulet device size prediction was concordant in 37.5% of cases; CT recommended a larger device in 58%, whereas 3D-TEE did so in 4.5%. Higher LAA contractility was associated with cases where 3D-TEE suggested a larger device (p = 0.016).

Conclusion: Cardiac CT provided larger LAA measurements than 3D-TEE, leading to selection of a larger device size in 58% of cases, and enabled more accurate identification of accessory lobes. 3D-TEE offered complementary value in patients with high LAA contractility, better capturing maximal LAA distension, which may not always be achieved with cardiac CT.

COI: No

O015

Clinical spectrum and phenotypic characteristics of patients with SCN5A variants and cardiomyopathy: early insights from an International Cohort

Ioannis Vouloagkas[1], Andrea Impellizzeri[2], Massimiliano Lorenzini[2], Zhongli Chen[3], Liang Chen[3], Christian Grebmer[4], Matthieu Proust[1], Alexis Hermida[5], Estelle Gandjbakhch[6], Argelia Medeiros Domingo[1], Siv Fokstuen[1], Felix C. Tanner[1], Corinna Brunckhorst[1], Firat Duru[1], Ardan Saguner[1]

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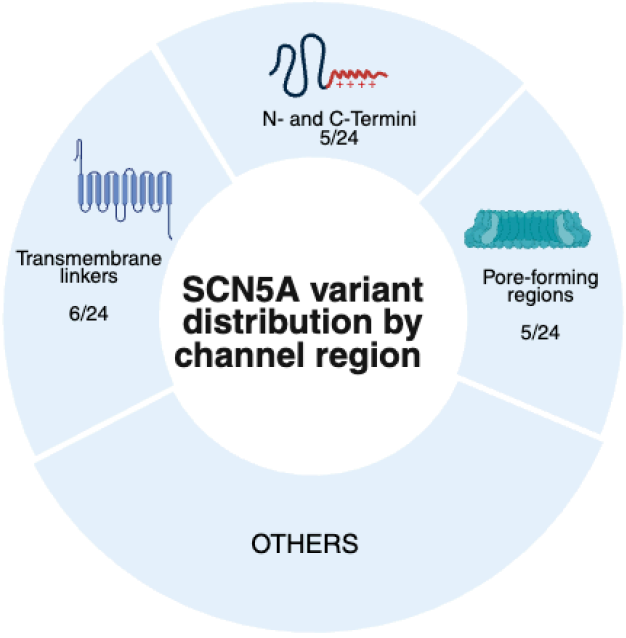
Introduction: Genetic variants in the SCN5A gene are primarily linked with electrical heart diseases such as Brugada syndrome. However, SCN5A variants have also been implicated in cardiomyopathies, and the clinical presentation of patients harbouring an SCN5A variant with structural heart disease remains poorly understood. Here, we present preliminary findings from our multicenter, international cohort.

Material & Methods: Results: We analyzed 34 patients (54% male; median age at onset 40 years) with heterozygous P/LP/VUS SCN5A variants presenting with cardiomyopathy. At baseline, 32% met criteria for dilated cardiomyopathy (DCM), 9% for arrhythmogenic cardiomyopathy (ACM, Padua 2020), 26% for ARVC (2010), 12% for hypertrophic cardiomyopathy (HCM), 15% had unclassified SCN5A-related cardiomyopathy, while single cases showed DCM/ACM overlap or an NDLVC phenotype. Among 24 patients with variants mapped to specific functional domains, variants localized to transmembrane linkers, N- or C-termini, and pore-forming regions. The most frequent ECG abnormality was intraventricular conduction delay (40%), with T-wave inversions mainly in anterior (V1–V3) and lateral leads (V4–V6). Conduction disturbances included right and left bundle branch block, and atrial fibrillation was rare. Median LVEF was 55% (IQR 37–59%), median FAC 45% (IQR 36–50%), and median LVEDD 52 mm (IQR 46.5–55). Dilated LV was present in 29%, and regional wall motion abnormalities were observed in both ventricles.



Conclusion: Patients with SCN5A variants and structural heart disease most commonly exhibited a DCM or ARVC phenotype. Despite preserved median LVEF and FAC at baseline, regional wall motion abnormalities were already detectable in a substantial fraction of patients, suggesting that early structural changes may precede global functional decline. Unlike classical channelopathies, where SCN5A variants often cluster in canonical voltage-sensor regions, variants in our cohort were shifted toward cytoplasmic termini and inter-segment linkers. Further studies are required to clarify whether structural and electrical SCN5A phenotypes arise from distinct variant topologies.

COI: No

O016

Diagnostic Accuracy of an AI-Based ECG Algorithm for Atrial Flutter: A Prospective Comparison with Electrophysiological Study

Fabian Jordan[1], Oguz Abdulhalik[2], Alexei Shvilkin[3], Lance Meyers[4], Uros Mitrovic[4], Nicola Formenti[1], Philipp Krisai[1], Nicolas Schärli[1], Sven Knecht[1], Felix Mahfoud[1], Michael Kühne[1], Christian Sticherling[1], Patrick Badertscher[1]

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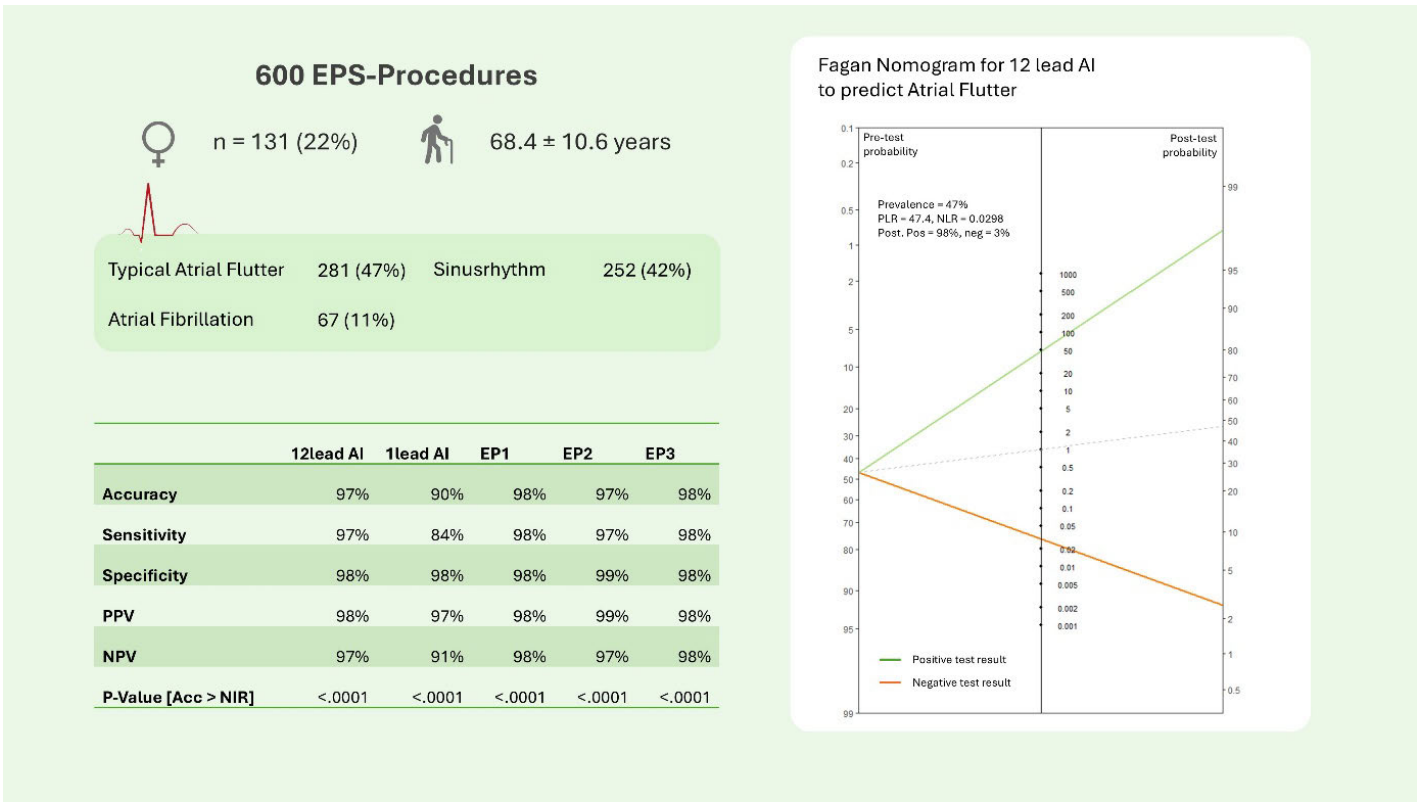
Introduction: Current automated electrocardiogram (ECG) algorithms reliably detect atrial fibrillation (AF) but perform poorly in identifying atrial flutter (AFL). We prospectively evaluated the diagnostic accuracy of an artificial intelligence (AI)-based ECG algorithm for typical AFL, using invasive electrophysiological study (EPS) as the reference standard.

Material & Methods: In this single-center diagnostic study, 600 patients referred for AFL ablation were enrolled. Standard 12-lead ECGs were obtained prior to EPS and analyzed by both the AI algorithm and two independent electrophysiologists.

Results: EPS confirmed typical AFL in 281 patients (47%), sinus rhythm in 252 (42%), and atrial fibrillation in 67 (11%). For detection of typical AFL, the AI algorithm achieved an accuracy of 97% (95% confidence interval [CI] 95–98), sensitivity 97%, specificity 98%, positive predictive value 98%, and negative predictive value 97%, demonstrating non-inferiority compared with manual expert interpretation. When applied to lead I only, simulating single-lead consumer devices, accuracy was 90% (CI 86–93), while manual interpretation by cardiologists reached 98% accuracy (κ = 0.95).

Conclusion: An AI-based ECG algorithm enables highly accurate detection of typical AFL from 12-lead ECGs, with diagnostic performance comparable to expert cardiologists and confirmed by EPS. Integration of such algorithms into smart devices and clinical ECG platforms could enhance rhythm classification and support earlier recognition of AFL in ambulatory care.

COI: No



	12lead AI	1lead AI	EP1	EP2	EP3
Accuracy	97%	90%	98%	97%	98%
Sensitivity	97%	84%	98%	97%	98%
Specificity	98%	98%	98%	99%	98%
PPV	98%	97%	98%	99%	98%
NPV	97%	91%	98%	97%	98%
P-Value [Acc > NIR]	<.0001	<.0001	<.0001	<.0001	<.0001

O017

Diagnostic Yield of Patient-Triggered Events During 14-days-Electrocardiographic Monitoring After Transcatheter Aortic Valve Implantation

Arianna Bettelini[1 2], Celine Moser[1 2], Jonas Brügger[1 2], Fabian Jordan[1 2], Sven Knecht[1 2], Teodor Serban[1 2], Reto Stump[1 2], David Spreen[1 2], Thomas Nestelberger[1 2], Christoph Kaiser[1 2], Beat Schär[1 2], Philipp Krisai[1 2], Nicolas Schärli[1 2], Christian Sticherling[1 2], Felix Mahfoud[1 2], Michael Kühne[1 2], Patrick Badertscher[1 2]

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Introduction: Wearable ECG patches used after transcatheter aortic valve implantation (TAVI) enable continuous rhythm monitoring and allow patients to report symptoms via trigger buttons and diary entries. However, the reliability of triggered events to identify clinically relevant arrhythmias after TAVI is uncertain. We aimed to systematically analyze patient-triggered events and their corresponding rhythm findings during 14-day ECG patch monitoring after TAVI.

Material & Methods: Adult patients undergoing TAVI at a Swiss tertiary centre were prospectively enrolled and monitored with a 14-day ECG patch before discharge. Patients were instructed to record any cardiac-related symptoms using the patch trigger button and/or diary function.

Results: A total of 260 patients (median age 82 years [78–86], 43% female) underwent ECG patch monitoring. Median monitoring duration was 13 days [11–14]. At least one patient-triggered event was recorded by 87 patients (33%), with a median of 2 triggered events per patient [1–3]. In 27 patients (10%) triggered events occurred exclusively during sinus rhythm without any associated arrhythmia. Atrial fibrillation was detected by continuous monitoring in 70 patients (27%), newly detected in 15 patients (6%). Among them, 18 patients (26%) reported symptoms during AF episodes. Pauses were identified in 54 patients (21%) and high-grade atrioventricular block in 31 patients (12%), with no triggered events during pauses and only one patient (3%) reporting associated symptoms during high-grade AV block. Ventricular tachycardia (≥3 beats) occurred in 130 patients (50%) and was not associated with any patient-triggered events. Supraventricular and ventricular ectopy were frequently detected and prompted patient activation in 12% and 10% of cases, respectively.

Conclusion: After TAVI, patient-triggered events during ECG patch monitoring poorly reflect objectively detected arrhythmias and substantially underestimate the burden of clinically relevant rhythm disturbances. Continuous rhythm analysis remains essential for reliable arrhythmia detection in this high-risk population.

COI: No

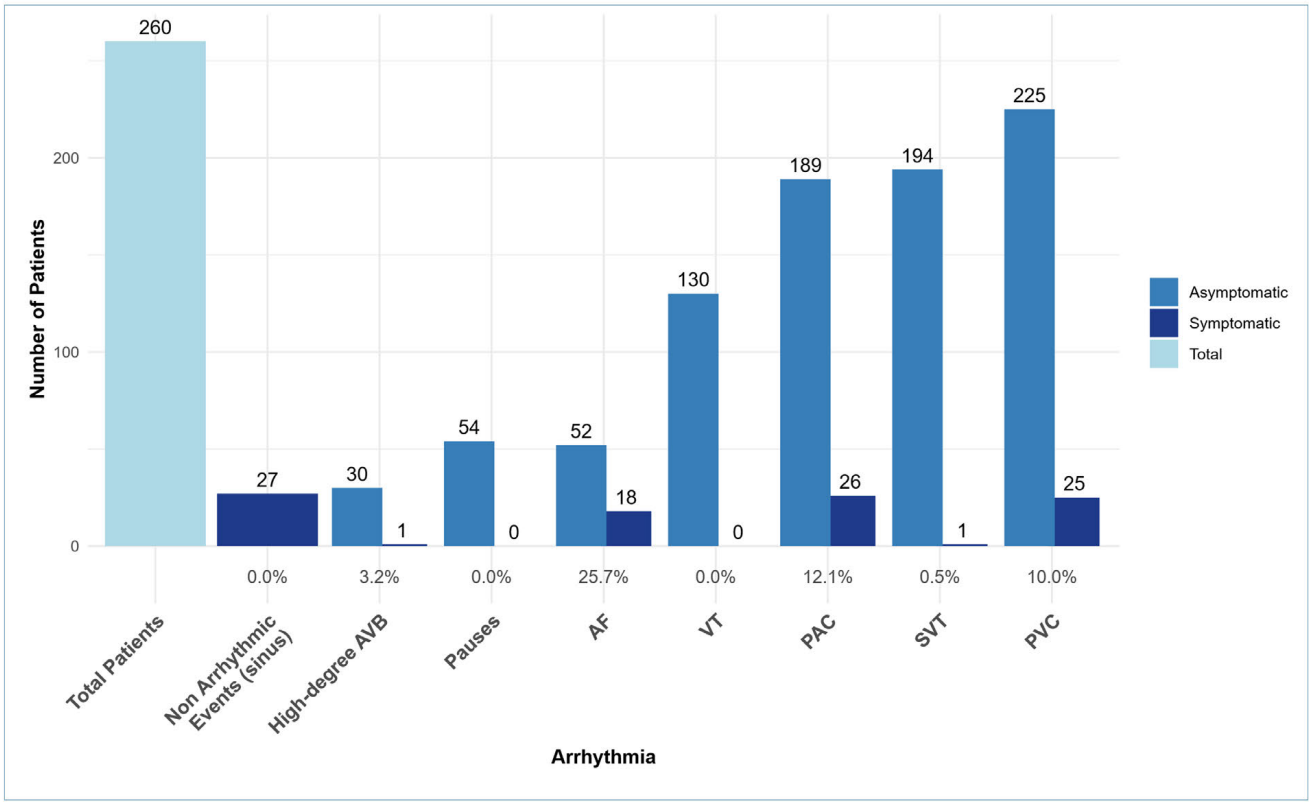
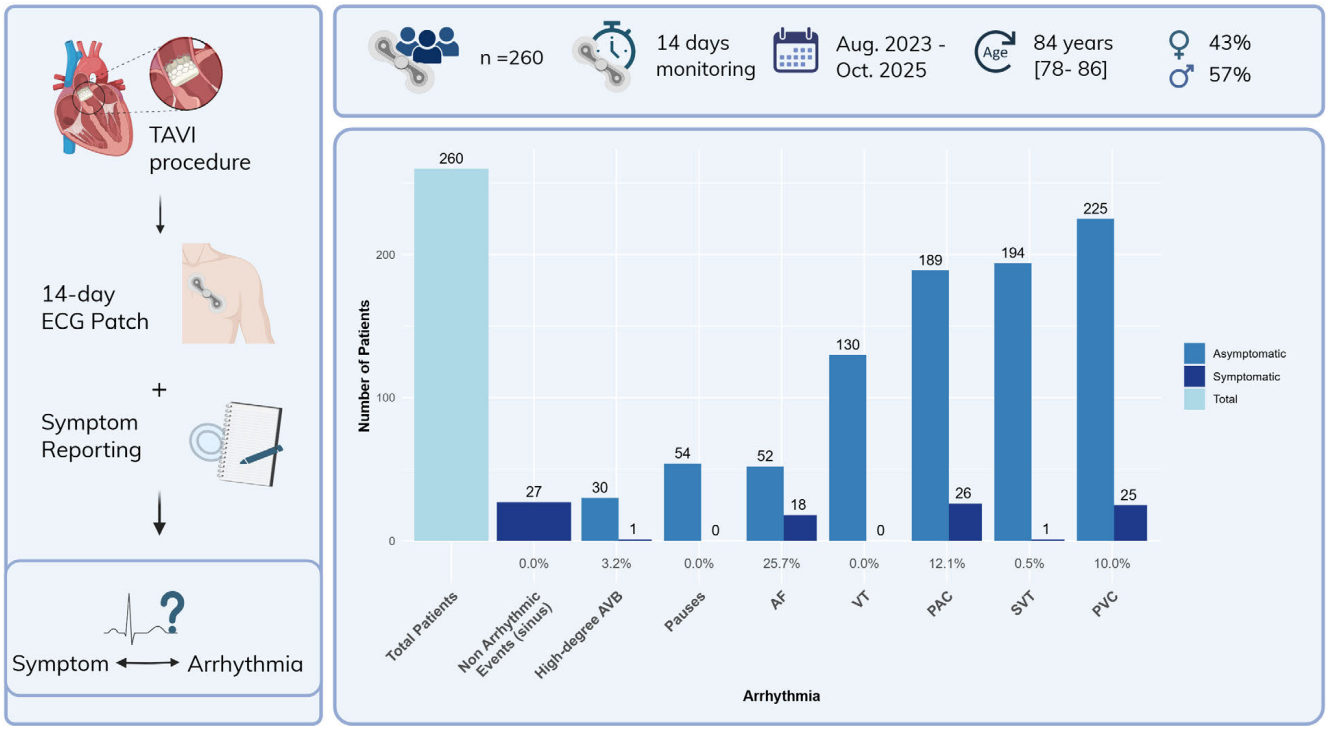


Figure 1. Distribution of arrhythmia types and symptom correlation during electrocardiographic patch monitoring

The figure shows the number of patients with at least one detected arrhythmia during electrocardiographic patch monitoring, stratified by symptomatic and asymptomatic presentation on a per-patient basis. Percentages indicate the proportion of patients with symptom-arrhythmia correlation for each arrhythmia category. Non-arrhythmic events (sinus) refer to patient-triggered symptoms without any corresponding arrhythmia detected. Abbreviations: AF = atrial fibrillation; AVB = atrioventricular block; VT = ventricular tachycardia; PAC = premature atrial complex; SVT = supraventricular tachycardia; PVC = premature ventricular complex.



O018

Clinical Presentation and Outcomes of Patients with Homozygous Cadherin Variants in Arrhythmogenic Cardiomyopathy (ACM): Insights from the Zurich ACM Registry

Stefanos Zafeiropoulos[1], Argelia Medeiros Domingo[2], Danaë Parianos[1], Deniz Akdis[1], Siv Fokstuen[1], Matthieu Proust[1], Felix C. Tanner[1], Ruth Biller[3], Andreas Giannopoulos[1], Marina Rieder[4], Andreas Flammer[1], Frank Ruschitzka[1], Corinna Brunckhorst[1], Firat Duru[1], Ardan Saguner[1]

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Introduction: Homozygous variants in desmosomal cadherins – desmoglein 2 (DSG2) and desmocollin 2 (DSC2) – constitute a rare but severe form of arrhythmogenic cardiomyopathy (ACM), typically associated with early-onset biventricular disease. Detailed phenotypic characterization and outcomes remain limited. We aimed to describe the clinical presentation and outcomes of ACM patients carrying homozygous cadherin variants.

Material & Methods: We retrospectively analyzed patients with homozygous DSG2 or DSC2 variants enrolled in the Zurich ACM Registry. Demographics, genetic data, ECG/Holter findings, echocardiography, 18F-FDG-PET/CT, endomyocardial biopsy, and clinical outcomes were extracted. Descriptive statistics were used.

Results: Six unrelated male patients were identified (median age at symptom onset 22 [IQR 17–26] years; median age at diagnosis 26 [19–28], Table 1). Four carried homozygous DSG2 and two homozygous DSC2 pathogenic/likely pathogenic variants. Three Swiss patients shared an identical DSG2 variant, suggesting a common founder variant. All patients presented with severe biventricular disease, including RV dilatation and dysfunction (median FAC 26 [23–30]%) and reduced LVEF (median 35 [33–51]%). Sustained ventricular arrhythmias occurred in three patients (50%), and all received ICDs. Myocardial inflammation was detected in three patients (50%): two with ^18F-FDG-PET/CT findings mimicking isolated cardiac sarcoidosis and one with borderline lymphocytic myocarditis on biopsy. One patient underwent heart transplantation, one died after declining transplantation, and one is currently evaluated for heart transplantation. Two patients (33%) experienced ischemic stroke without atrial fibrillation, likely due to LV thrombus formation, prior to anticoagulation; four patients received anticoagulation.

Conclusion: Homozygous cadherin-related ACM is an early-onset, severe biventricular disease with rapid heart failure progression, frequent myocardial inflammation, and high thromboembolic risk independent of atrial arrhythmias. These findings support liberal anticoagulation in advanced phenotypes and systematic evaluation for myocardial inflammation, alongside early referral to heart transplant centers.

COI: No

Table 1.

Variable	Homozygous *DSG2* (n = 4)	Homozygous *DSC2* (n = 2)
Male (%)	4 (100%)	2 (100%)
Origin/Ethnicity	3 Swiss, 1 Somali	1 Kurdish, 1 Turkish
Mutation	c.523+2T>C p.(?)[1] c.880A>G p.(Lys294Glu)	c.1A>G p.(Met1?) c.354+3A>G p.(Val52Glyfs*8^Val52Aspfs*15)[1]
Age at symptom onset (years)	17.0 [15.5–20.0]	30.5 [28.2–32.8]
Age at diagnosis (years)	21.0 [15.5–26.5]	30.5 [28.2–32.8]
Biventricular involvement	4 (100%)	2 (100%)
LVEF (%)	35.5% [34–41]	41.0% [28.0–54.0]
RV FAC (%)	28 [23.5–30]	29.5 [24–35]
Malignant ventricular arrhythmias (sustained VT/VF)	3 (75%)	0 (0%)
ICD implantation	4 (100%)	2 (100%)
Inflammatory findings (PET or biopsy)	2 (50%)	1 (50%)
Heart transplantation	1 (25%)	0 (0%)
Listed/evaluated for transplantation	1 (25%)	0 (0%)
Death due to heart failure	1 (25%)	0 (0%)
Stroke / systemic embolism	1 (25%)	1 (50%)
Anticoagulation	3 (75%)	1 (50%)

[1]novel mutations

RAPID FIRE ABSTRACTS: "AFTER ABLATION"

O019

Ventricular Fibrillation During Lattice-Tip Radiofrequency Ablation Adjacent to ICD Coils: A Multicenter Case Series

Boldizsar Kovacs[1], Celina Malyar[2], Kinan Kneizeh[3], Nicolas Schärli[4], Valon Spahiu[1], Thomas Kueffer[1], Andreas Haeberlin[1], Laurent Roten[1], Michael Kühne[4], Helmut Pürerfellner[3], Sing-Chien Yap[2], Tobias Reichlin[1]

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Introduction: The lattice-tip dual-energy catheter (LTC) delivers both pulsed-field (PF) and high-power, temperature-controlled radiofrequency (RF) ablation. Although generally effective, unexpected ventricular fibrillation (VF) during RF applications in patients with ICDs has been reported, and the mechanism remains uncertain. We describe a multicenter experience of inadvertent VF induction during ablation with the LTC near ICD high-voltage (HV) leads.

Material & Methods: Seven cases from four tertiary centers were identified. All procedures used temperature-controlled RF (target 60 °C, ~200 W) for ventricular tachycardia (VT) or cavotricuspid isthmus (CTI) ablation, during which VF occurred. Device interrogations were performed pre- and post-procedure and fluoroscopy documented catheter position at VF onset.

Results: Baseline characteristics appear in Figure 1A. In all cases, the catheter was close to an HV coil (3 right-ventricular inferoseptal, 3 left-ventricular inferoseptal, 2 CTI), with one patient experiencing events during both RV and CTI ablation. The catheter–coil distance was ~10 mm (5–30) (Figure 1B). Impedance and temperature behavior were normal. VF was terminated in all patients. Visible pectoral stimulation at the generator site occurred in one case. All ICD generators and HV leads were from the same manufacturer. Median HV lead age was 15 months (3–185); two patients had abandoned leads. Median preprocedural impedance was 68.5 Ω, with postprocedural changes of –2 to +6 Ω except in two patients who showed abnormal values and ultimately required generator replacement. No structural defects of the ablation catheter were found.

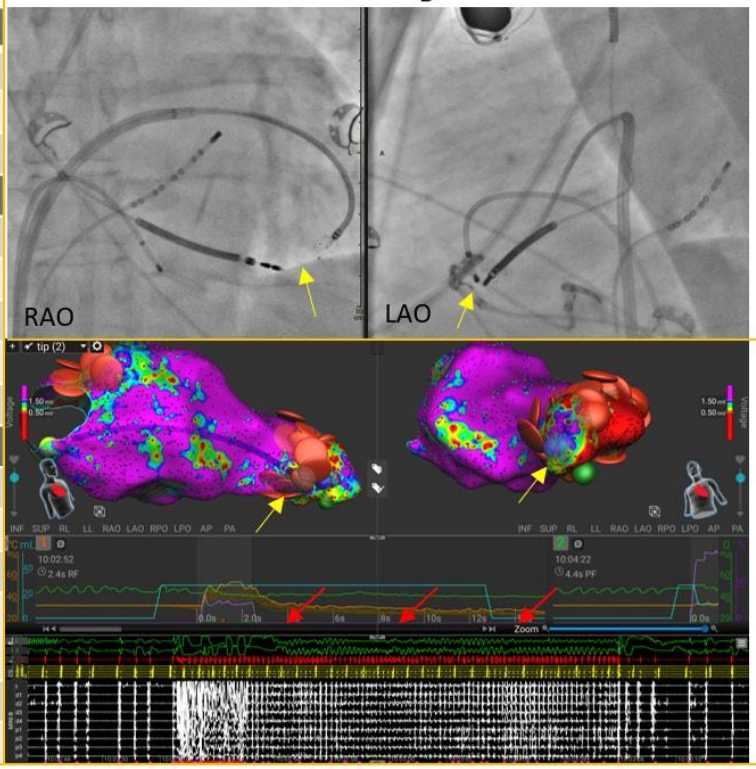
Conclusion: RF ablation with the LTC near an ICD HV coil can precipitate VF, likely through unintended coupling of the lead–can system into the RF circuit. Generator malfunction, possibly related to PF delivery, was also observed. Caution is warranted when ablating near ICD leads, and RF should be avoided directly on or adjacent to HV coils.

COI: No

A

Number of patients, n	**7 (100)**
Age, years — median (range)	55 (44–68)
Male sex, n (%)	7 (100)
BMI, kg/m² — median (range)	28 (20–36)
Clinical substrate	
ARVC, n (%)	1 (15)
— Ischemic, n (%)	3 (42.5)
Non-ischemic dilated, n (%)	3 (42.5)
LVEF, % — median (range)	45 (20–64)
Prior ICD-shocks for VT/VF. n (%)	5 (71)
Periprocedural characteristics	
General anesthesia, n (%)	4 (57.5)
Conscious sedation, n (%)	3 (42.5)
Site of RF delivery during VF induction	
Number of delivered shocks to terminate VF episodes — median (range)	1 (0–5)
Post-procedure device dysfunction, n (%)	2 (29)
Other adverse events, n (%)	0 (0)

B



O020

Catheter Ablation of Atrial Fibrillation in Transthyretin and Light-Chain Cardiac Amyloidosis: Results from the Multicenter AMYL-AF Study

Daniele Faccenda[1], Tom De Potter[2], Michel Haissaguerre[3], Johan Saenen[4], Carlo de Asmundis[5], Giuseppe Ciconte[6], Marta De Riva[7], Michela Casella[8], Daniel Scherr[9], Mikael Laredo[10], Patrizio Mazzone[11], Avi Sabbag[12], Matteo Anselmino[13], Claudio Tondo[14], Giulio Conte[1]

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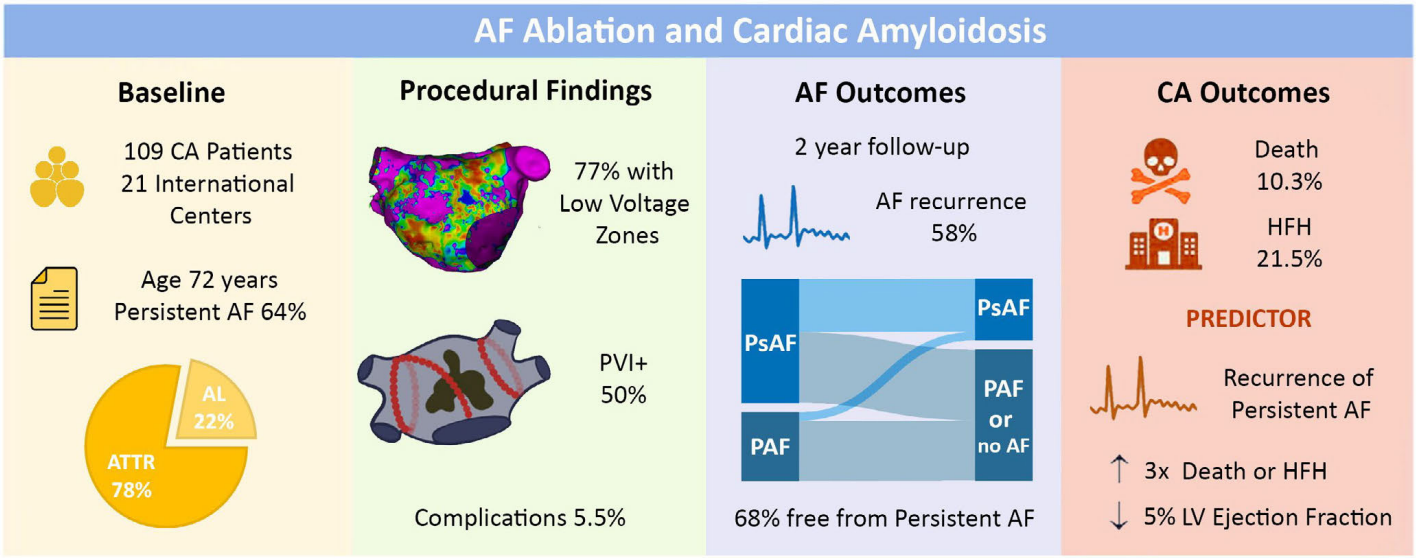
Introduction: Atrial fibrillation (AF) is highly prevalent among cardiac amyloidosis (CA) patients and contributes significantly to their morbidity and mortality. Evidence regarding AF ablation efficacy and safety in CA patients is limited. The aim of this study was to evaluate baseline characteristics and outcomes of AF ablation in a series of patients with transthyretin (ATTR) or light-chain (AL) CA.

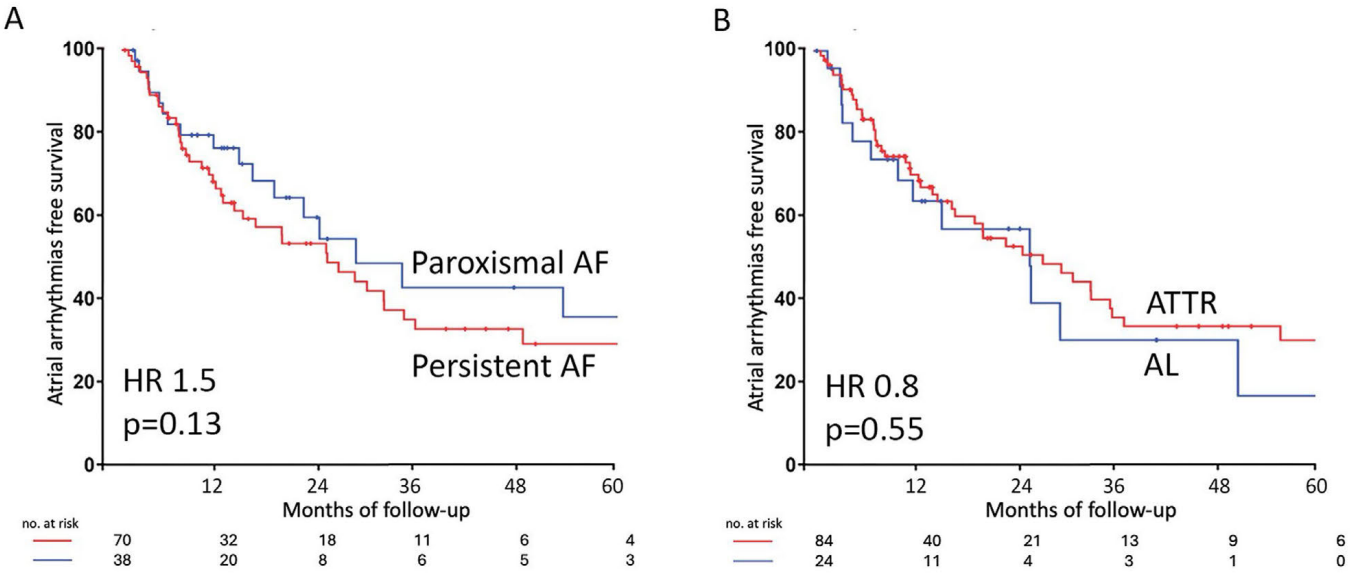
Material & Methods: This was a multicenter, retrospective study including consecutive CA patients undergoing AF ablation in 21 international centers. Co-primary endpoints were: (1) atrial arrhythmia (AA) recurrence; (2) a composite endpoint of all-cause mortality and heart failure (HF) hospitalization.

Results: A total of 109 patients (mean age 72.4±7.4 years, females 17.4%, persistent AF 64.2%, ATTR 78%, AL 22%) were included. Radiofrequency, cryo-balloon and pulsed-field ablation were performed in 67%, 15% and 18% of patients, respectively; 49.5% received pulmonary vein isolation plus additional ablations. Low voltage zones were documented in 34 out of 44 patients undergoing electro-anatomical mapping (77.3%). During a median follow-up of 22.7 months, 63 patients (58.3%) experienced AA recurrence (32.4% persistent AF recurrence), with no significant differences between CA subtypes (ATTR 59.5% vs AL 54.2%, log-rank p = 0.55, figure 1), and no baseline characteristic was associated with lower recurrences. The composite endpoint of HF hospitalization and all-cause death occurred in 27 patients (25.0%). At univariate logistic regression analysis, recurrence of persistent AF was associated with three-fold higher risk (OR 2.9, p = 0.02, figure 2) of the composite endpoint. Patients without persistent AF recurrence had a higher left ventricular ejection fraction (55% vs. 50%, p = 0.02) at follow-up.

Conclusion: CA patients undergoing AF ablation present high prevalence of persistent AF and advanced atrial remodeling. Freedom from AA after AF ablation is achieved in 42% of patients after a median follow-up of two years. Patients with persistent AF recurrence have three times higher risk of HF hospitalization and death.

COI: No





O021

Impact of Follow-up Strategy After Catheter Ablation of Atrial Fibrillation on Repeat Procedures

Celine Moser[1 2], Corinne Isenegger[1 2], Jonas Brügger[1 2], Fabian Jordan[1 2], Arianna Bettelini[1 2], Sven Knecht[1 2], Philipp Krisai[1 2], Gian Voellmin[1 2], David Spreen[1 2], Nicolas Schärli[1 2], Reto Stump[1 2], Beat Schär[1 2], Felix Mahfoud[1 2], Christian Sticherling[1 2], Michael Kühne[1 2], Patrick Badertscher[1 2]

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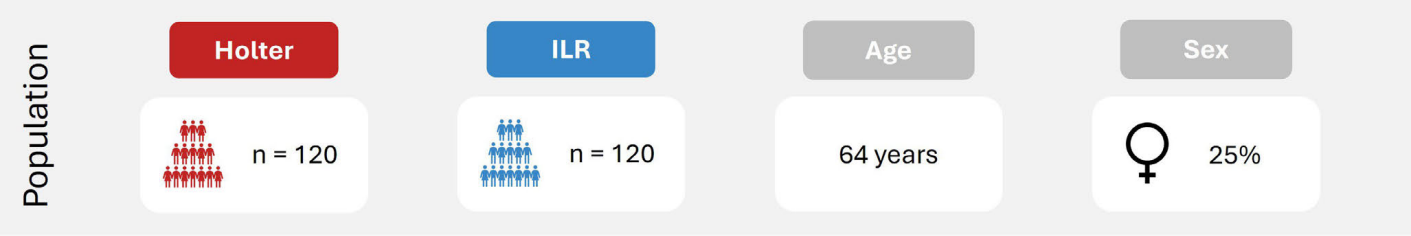
Introduction: The intensity and type of rhythm monitoring after catheter ablation for atrial fibrillation (AF) affects arrhythmia detection. However, it is unclear whether more intensive rhythm surveillance also translates into higher rates of repeat ablation. Our aim was to compare the impact of systematic Holter ECG follow-up versus implantable loop recorder (ILR) monitoring on the incidence of repeat catheter ablation procedures after first-time pulmonary vein isolation (PVI).

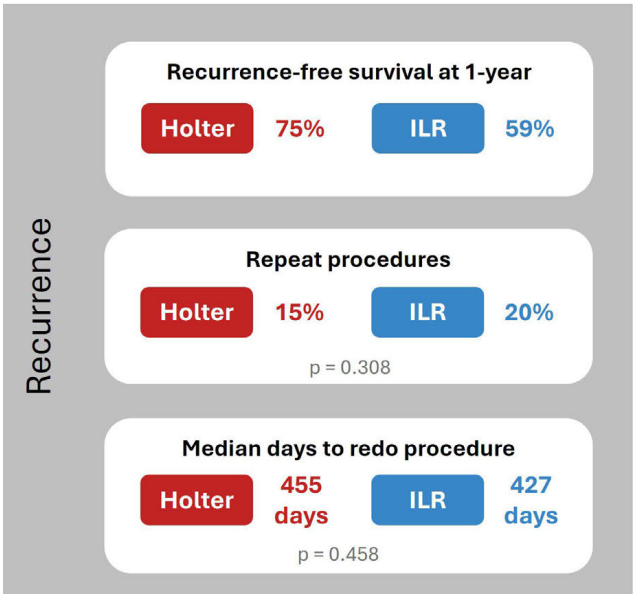
Material & Methods: Patients with paroxysmal AF undergoing single-shot PVI at a tertiary center were enrolled prospectively. Follow-up consisted of standardized Holter recordings at 3, 6, and 12 months (plus symptom-triggered ECGs) or continuous ILR monitoring. Recurrence was defined as any atrial arrhythmia lasting >30 seconds after a 60-day blanking period. Redo procedures performed within 3 years after index PVI were analyzed. Cohorts were propensity-score matched for age, sex, AF type, and cardiovascular comorbidities.

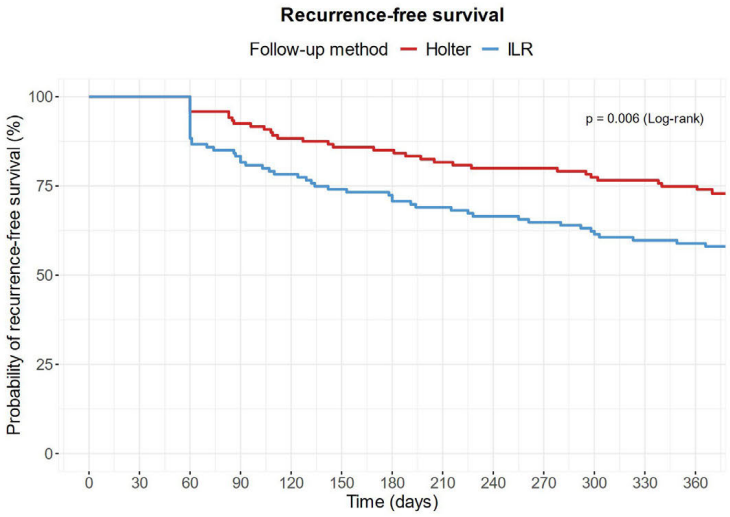
Results: A total of 240 patients (median age 64 [56–71] years, 25% female) were included; 120 (50%) underwent Holter-based follow-up and 120 (50%) ILR-based follow-up. Ablation modality was similar (Cryo 75% vs. 79%; pulsed-field ablation 25% vs. 21%; p = 0.443). At 1-year, atrial arrhythmia recurrence was significantly more frequently detected in ILR patients compared to Holter (41% vs. 25%; log-rank p = 0.006). Redo procedures were performed in 20% vs. 15% of ILR vs. Holter, respectively (p = 0.308). Median time to redo was comparable (455 vs. 427 days; p = 0.458), and redo-free survival did not differ (log-rank p = 0.196). Results were consistent across ablation modalities.

Conclusion: Despite markedly higher arrhythmia detection with continuous rhythm monitoring, follow-up modality did not significantly impact redo rates after PVI. These data suggest that more intensive follow-up using ILRs does not necessarily alter clinical decision-making regarding repeat ablation.

COI: No







O022

Device-Dependent Variability of Activated Clotting Time in Left-Sided Electrophysiology Procedures

Valon Spahiu[1], Ivan Zeljkovic[2], Henning Nilius[1], Sophie Dütschler[1], Thomas Kueffer[1], Berat Gerguri[3], Varis Gjergjizi[1], Claudia Herrera Siklody[1], Nikola Kozhuharov[1], Helge Servatius[1], Hildegard Tanner[1], Laurent Roten[1], Michael Nagler[1], Tobias Reichlin[1], Boldizsar Kovacs[1]

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Introduction: Current guidelines recommend maintaining an activated clotting time (ACT) >300 s during left-sided electrophysiology procedures to minimize thromboembolic risk. However, it remains largely unknown whether different routinely used ACT-devices and cartridge-types measure the same biological signal.

Purpose: To quantify inter-device and intra-device variability of ACT-measurements during left-sided-EP procedures under "real-world" anticoagulation conditions.

Material & Methods: We prospectively compared ACT-values obtained simultaneously from the same whole blood samples during left-sided-EP procedures using three different commercially available ACT-devices (Optical-OP; Electromechanical-EM; Electrochemical-EC) and cartridge (high-HR and low-range-LR) types. We included 185 consecutive patients undergoing left-atrial (n = 146) or left-ventricular (n = 23) ablations. All patients received a heparin bolus (150-200 IU/kg) to maintain an ACT of 300-350s as measured by the reference ACT-device (EM, HR-cartridge). ACT was measured 5 minutes after heparin administration and every 30 minutes. For statistical analysis, Pearson's correlation coefficients were calculated among the three ACT-devies and for HR and LR separately.

Results: Correlations between the test systems were moderate, ranging from 0.52 (EM/OP) to 0.58 (EM/EC) with the HR-cartridges (Figure1 B and D). Similar results were observed for the LR-cartridges, with correlations of 0.55 (EM vs. OP) and 0.49 (EM vs. EC) (Figure2, B and D). PB-analysis demonstrated statistically significant bias in both intercept and slope across all comparisons (Figures1 and 2, B and D). The absolute difference between measured ACT ranged from –115 to 130 s with the HR-cartridges (Figure1 A and C) and from –40 to 630 s with the LR-cartridges (Figure2 A and C). In particular, comparisons involving the EM-device + LR cartridges showed pronounced systematic bias as demonstrated on Figure2, B and D.

Conclusion: ACT results differ substantially between devices and cartridge types, indicating that different devices do not measure the same thing. Defining device-specific therapeutic ACT ranges or applying validated correction factors, could improve consistency and patient safety.

COI: No

Figure 1: Upper panels shows absolute difference between the EM platform + HR cartridge and OP platform + HR cartridge (Bablok Regression Fit); The lower panels shows absolute difference between the EM platform + HR cartridges and the EC platform (Bablok Regression Fit). Number of simultaneous comparisons is shown below the x axis.

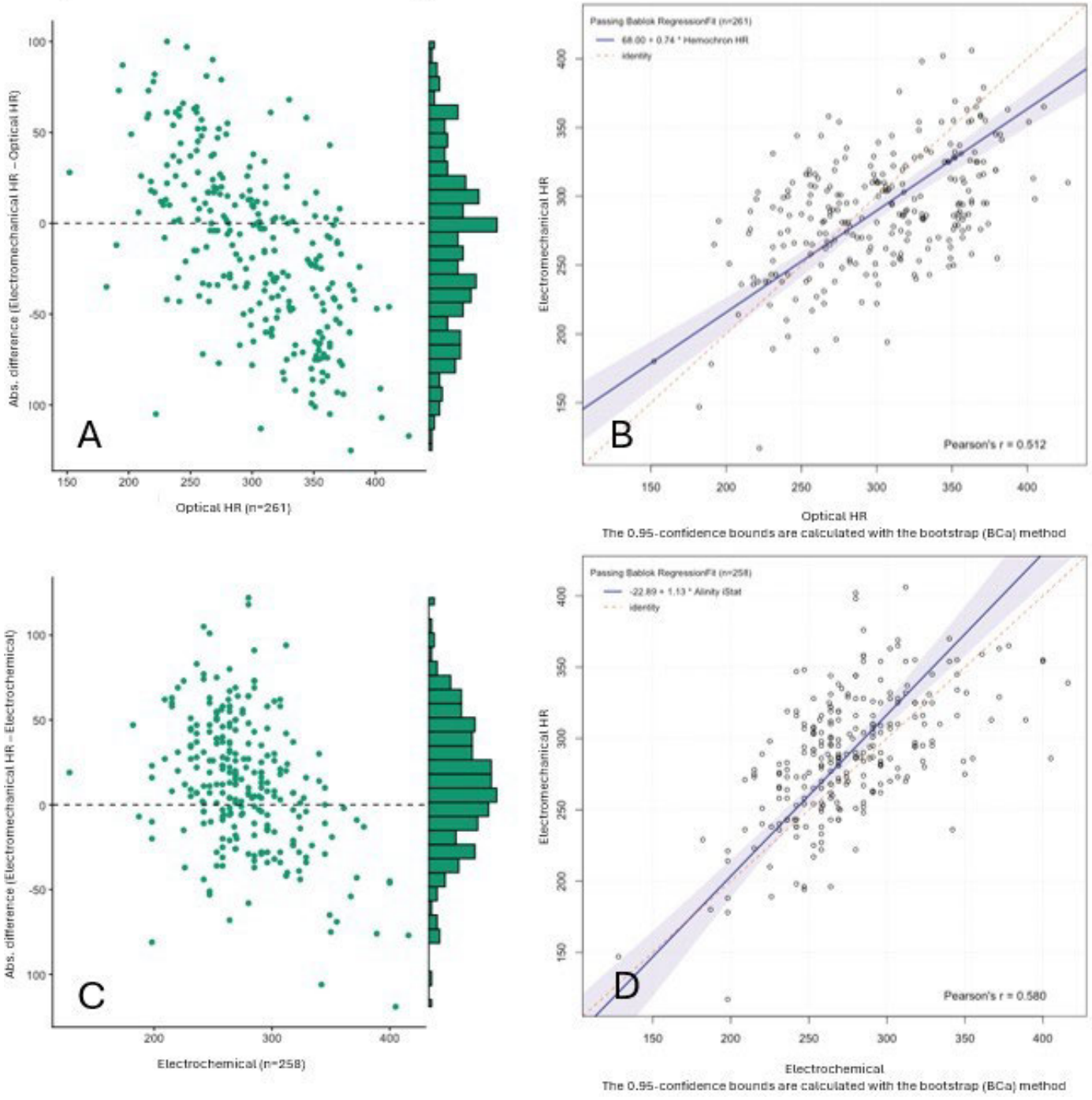
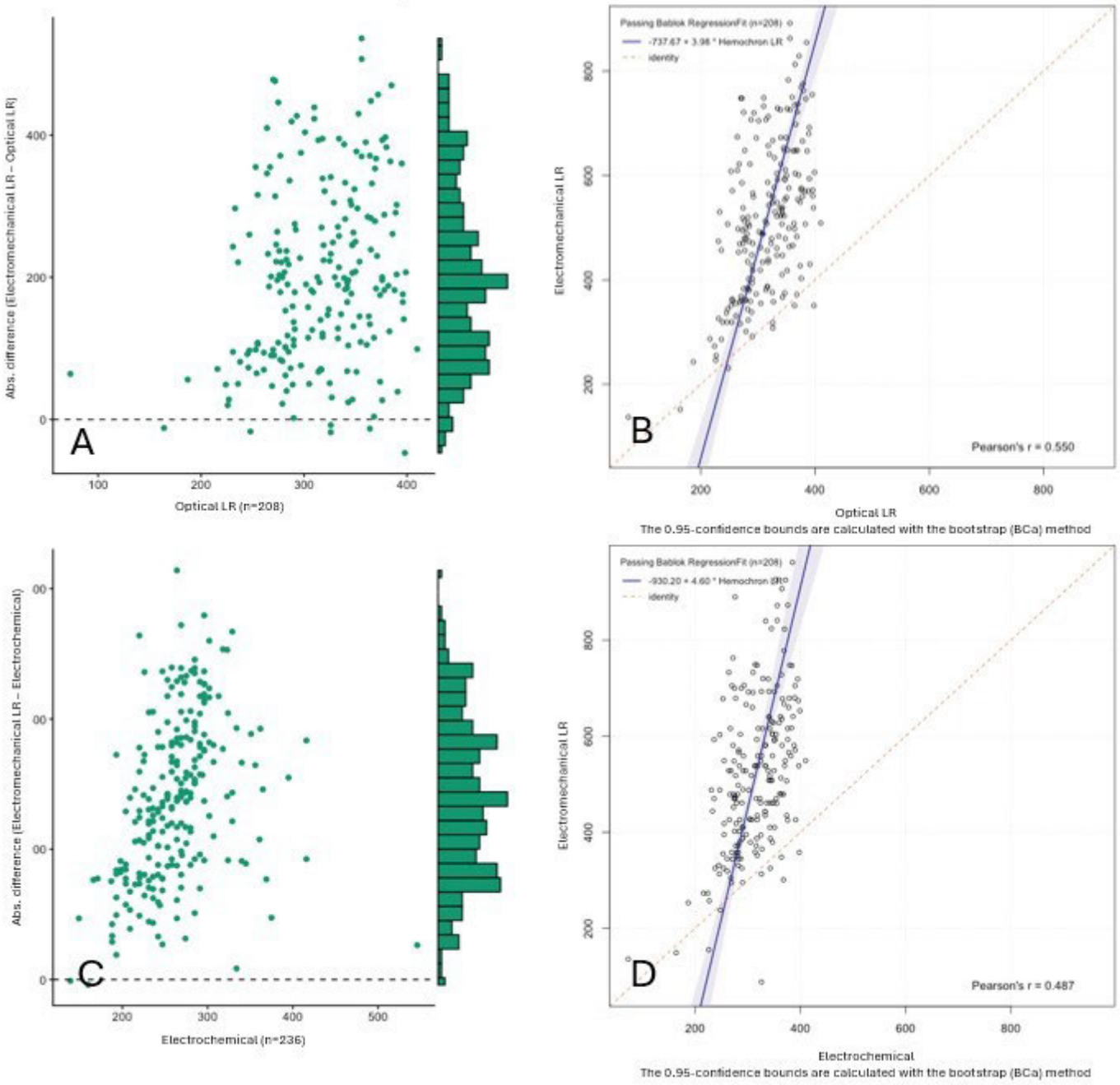


Figure 2: Upper panel shows absolute difference between the EM platform + LR cartridge and OP platform + LR cartridge (Bablok Regression Fit); Lower panel shows absolute difference between the EM platform + LR cartridge and EC platform (Bablok Regression Fit). Number of simultaneous comparisons is shown below the x axis.



O023

Quality of Life After Atrial Fibrillation Ablation: A Comparison of Pulsed-field versus Radiofrequency Energy

Corinne Isenegger[1,2], Arianna Bettelini[1,2], Jonas Brügger[1,2], Fabian Jordan[1,2], Celine Moser[1,2], Sven Knecht[1,2], Philipp Krisai[1,2], David Spreen[1,2], Nicolas Schärli[1,2], Reto Stump[1,2], Felix Mahfoud[1,2], Christian Sticherling[1,2], Michael Kühne[1,1], Patrick Badertscher[1,2]

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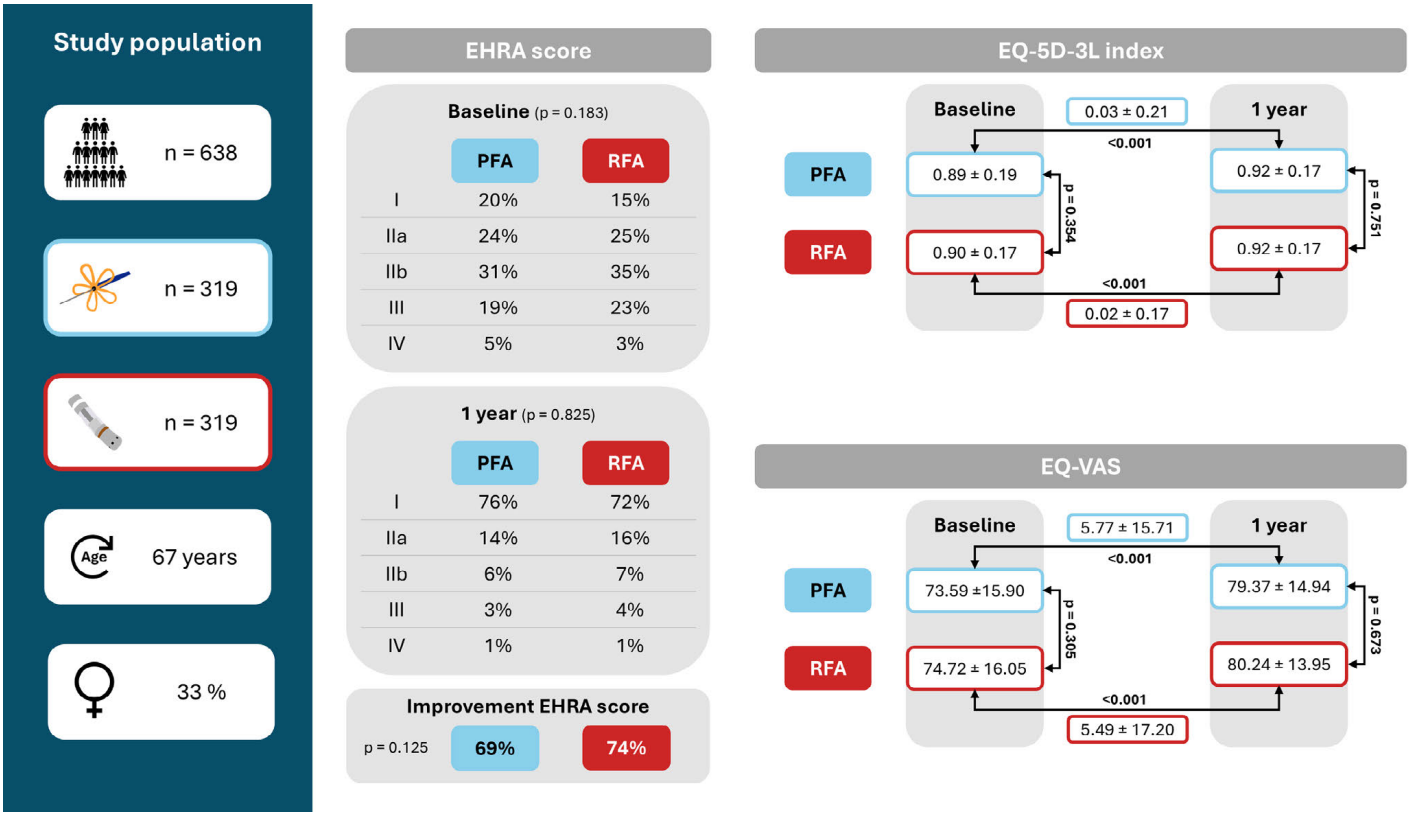
Introduction: Atrial fibrillation (AF) markedly impairs quality of life (QoL). Catheter ablation improves symptoms beyond rhythm control alone. Pulsed-field ablation (PFA) is rapidly emerging as a next-generation energy source, yet comparative real-world data on QoL versus radiofrequency ablation (RFA) are scarce. The aim of this study was to quantify QoL change one year after AF ablation and compare PFA with RFA.

Material & Methods: Consecutive AF patients undergoing first-time PVI using PFA or RFA at a tertiary center were prospectively enrolled. QoL was assessed at baseline and one year using EHRA score, EQ-5D-3L and EQ-VAS. Propensity score matching was performed for age, sex, AF type, and cardiovascular comorbidities.

Results: A total of 638 patients were included (median age 67 years, 33% female, 57% paroxysmal AF), with 319 matched pairs. Procedural time was markedly shorter with PFA (50 vs. 79 minutes, p <0.001). Both energy sources produced significant and clinically meaningful QoL improvements at one year (Figure). After one year, the proportion of patients in EHRA class I increased from 20% to 76% in the PFA group and from 15% to 72% in the RFA group, with overall EHRA improvement observed in 69% and 74% of patients, respectively (p = 0.125). PFA improved EQ-5D-3L index (0.89±0.19 to 0.92±0.17, p <0.001) and EQ-VAS (73.6±15.9 to 79.4±14.9, p <0.001). RFA improved EQ-5D-3L (0.90±0.17 to 0.92±0.17, p <0.001) and EQ-VAS (74.7±16.1 to 80.2±14.0, p <0.001). Between-group comparisons showed no significant differences at baseline or one year. Recurrence-free survival at one year was similar between PFA and RFA (70% vs 72%, $p_{log\text{-}rank}$ = 0.579).

Conclusion: AF ablation led to a meaningful improvement in patient-reported QoL, independent of whether the procedure was performed with PFA or RFA. PFA, however, achieved comparable outcomes with substantially shorter procedure times.

COI: No



O024

High Sensitivity Troponin-T after Pulsed Field Ablation: Associations with Procedural Characteristics and Outcome

Johannes Bruss[1], Thomas Kueffer[1], Hildegard Tanner[1], Fabian Noti[1], Andreas Haeberlin[1], Gregor Thalmann[1], Nikola Kozhuharov[1], Boldizsar Kovacs[1], Valon Spahiu[1], Claudia Herrera Siklody[1], Tobias Reichlin[1], Laurent Roten[1]

[1]*Inselspital Bern, Bern, Switzerland*

Introduction: Pulmonary vein isolation (PVI) using pulsed field ablation (PFA) leads to a markedly greater increase in high-sensitivity Troponin-T (hs-TnT) compared with radiofrequency or cryoballoon ablation. This study investigated associations between hs-TnT increase after PFA, procedural characteristics, and clinical outcomes.

Material & Methods: This retrospective registry analysis included patients undergoing primary PVI without posterior wall ablation using a pentaspline PFA catheter between September 2022 and June 2024. Laboratory, echocardiographic, and procedural data were obtained from chart review. Hs-TnT was measured before and the day after PVI. Follow-up comprised 7-day Holter ECGs at 3, 6, and 12 months and review of clinical records for arrhythmia recurrence. Associations were assessed using Spearman correlation, Wilcoxon rank-sum testing, ANCOVA, and the Mantel-Haenszel log-rank test.

Results: A total of 457 patients were analyzed (median age 67 years; 69% men). Median hs-TnT increased from 10 (7–15) ng/L pre-procedure to 1581 (1100–2216) ng/L post-procedure, with a median increase (Δhs-TnT) of 1547 (1078–2210) ng/L. Patients in the lowest quartile of hs-TnT increase exhibited significantly higher 1-year arrhythmia recurrence compared with others (p = 0.003, Figure 1). Hs-TnT increase was not associated with age, renal function, number of PFA applications, or operator experience (Figure 2a – d). Lower hs-TnT release was observed in patients with persistent atrial fibrillation (AF) and correlated inversely with left atrial volume and baseline NT-proBNP levels (Figure 2e -g)

Conclusion: A lower hs-TnT increase after PFA is associated with worse clinical outcomes. Hs-TnT release appears independent of procedural intensity or operator experience but is reduced in patients with persistent AF and advanced atrial remodeling. A robust hs-TnT response may reflect a healthier atrial substrate, whereas attenuated release may indicate advanced atrial cardiomyopathy and higher recurrence risk.

COI: No

Figure 1

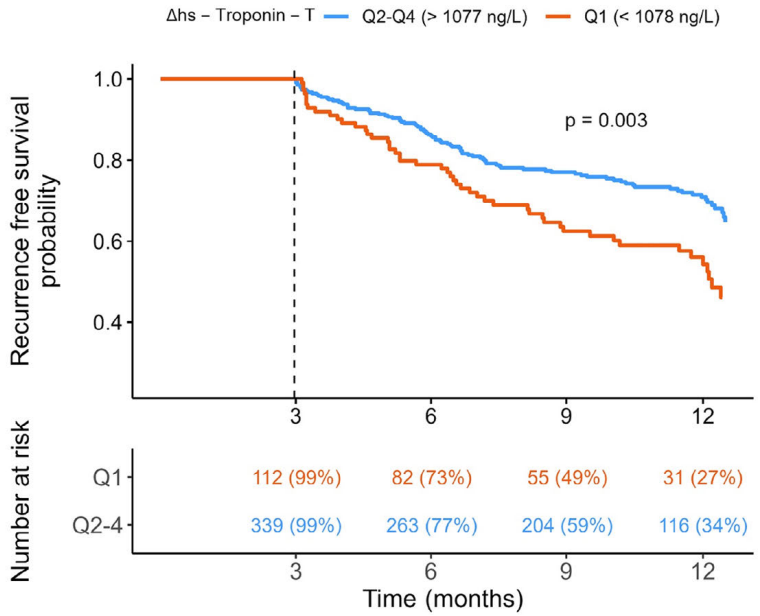
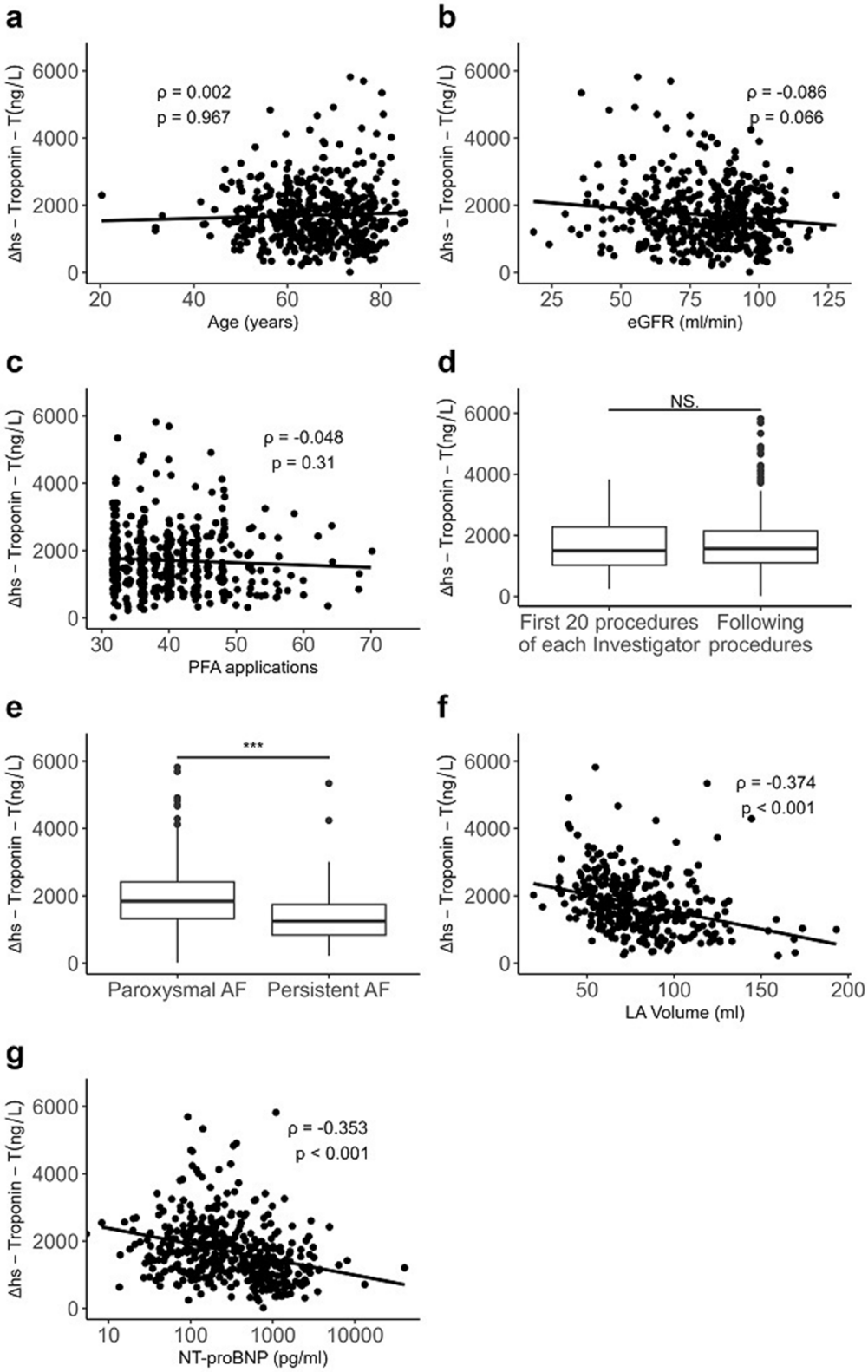


Figure 2



RAPID FIRE ABSTRACTS: CAD & ACC

O025

Perioperative Discontinuation of SGLT2-Inhibitors and Cardiac Complications After Non-Cardiac Surgery

Fisnik Haziri[1], Koray Durak[1], Noemi Glarner[1], Emre Ergin[1], Katrin Burri[1], Emel Kaplan[1], Vanessa Thommen[1], Mirjam Pargger[1], Gabrielle Huré[1], Daniel Bolliger[2], Luzius Steiner[2], Michael Kunz[1], Thorald Stolte[1], Felix Mahfoud[3], Danielle Menosi Gualandro[1], Christian Müller[1]

[1]Cardiovascular Research Institute Basel, Basel, Switzerland, [2]Department of Anaesthesia, University Hospital Basel, University of Basel, Switzerland, Basel, Switzerland, [3]1Department of Cardiology, University Hospital Basel, University of Basel, Switzerland, Basel, Switzerland

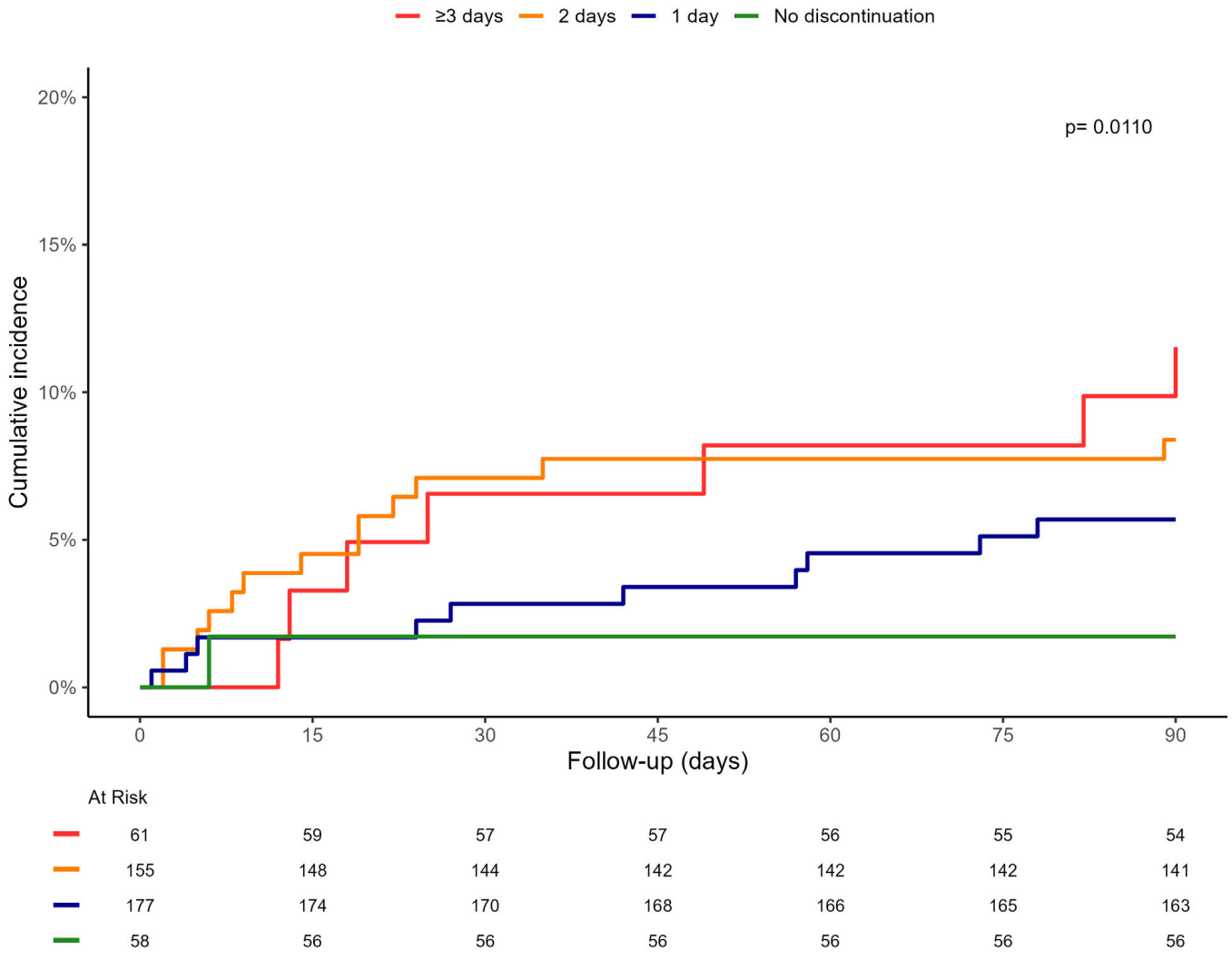
Introduction: Health authorities and clinical practice guidelines recommend discontinuing sodium–glucose cotransporter-2 inhibitors (SGLT2i) 3-4 days before surgery to prevent euglycemic diabetic ketoacidosis (EDKA), largely based on case reports. However, major uncertainties remain regarding the risk-benefit-ratio of SGLT2i discontinuation, due to potential increases in postoperative cardiac complications.

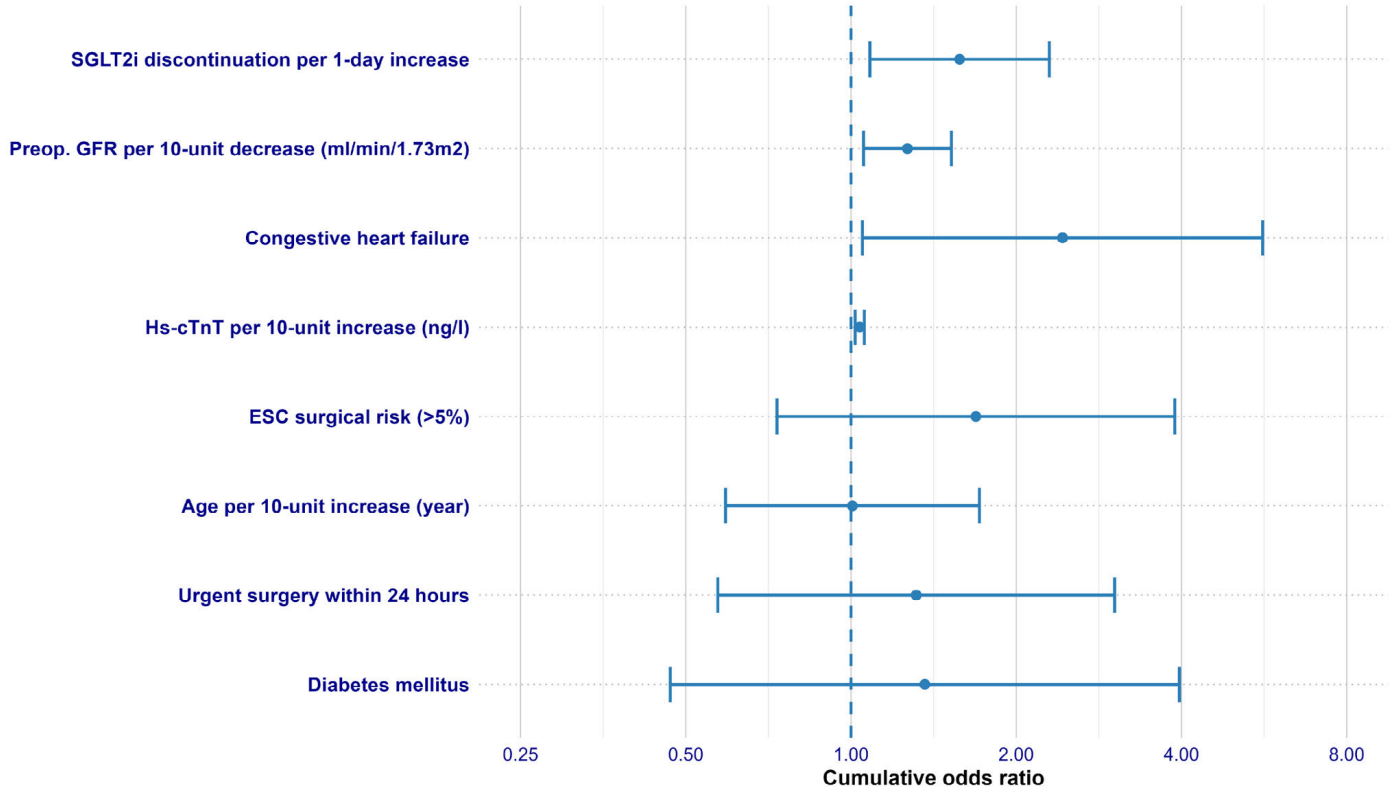
Material & Methods: Discontinuation of SGLT2i was assessed and quantified in consecutive high-risk patients on chronic SGLT2i therapy undergoing major non-cardiac surgery enrolled in two prospective cohort studies. The primary outcome was an ordinal composite of acute heart failure hospitalisation and cardiovascular death within 90 days, analysed using a partial proportional-odds ordinal logistic regression with adjustment for prespecified confounders. The secondary outcome was EDKA within 7 days.

Results: Among 451 patients (22% women; mean age 72 years), 95.1% had diabetes mellitus, 78.9% had chronic kidney disease and 36.9% had chronic heart . SGLT2i were preoperatively discontinued in 87.1% of patients (39.2% for 1 day, 34.4% for 2 days, 13.5% for ≥3 days). The primary outcome occurred in 1.7% of patients without discontinuation, 5.7% with discontinuation for 1 day, 8.4% with discontinuation for 2 days, and 11.5% with discontinuation for ≥3 days. This association persisted after multivariable adjustment: cumulative odds ratio 1.58 (95% CI, 1.08–2.30) per discontinued day. One mild case (0.22%) of EDKA was observed in a patient without SGLT2i discontinuation.

Conclusion: Perioperative SGLT2i discontinuation was associated with a substantially increased risk of 90-day cardiac complications, clearly outweighing the very low rate of EDKA. This suggests that the current recommendation for discontinuation may need reconsideration.

COI: No





O026

Intracoronary ECG ST-Segment Shift Remission Time (τ-icECG) for Assessing Hemodynamic Coronary Stenosis Severity: A Prospective Validation Study

Marius Bigler[1], Andrea Kieninger-Gräfitsch[1], Christian Seiler[1], Christine Tschannen[1], Miklos Rohla[1], Noé Corpataux[1], Frédéric Waldmann[2], Reto Wildhaber[2], Jonas Häner[1]

[1]Department of Cardiology, University Hospital Bern (Inselspital), University of Bern, Bern, Switzerland, [2]Institute for Medical Engineering and Medical Informatics, University of Applied Sciences and Arts Northwestern Switzerland, Muttenz, Switzerland

Introduction: Intracoronary electrocardiogram (icECG) ST-segment shift remission time (τ-icECG, seconds, s) following a 1-minute proximal coronary artery balloon occlusion is a novel pathophysiology-based approach for the detection of hemodynamically relevant coronary lesions. Longer τ-icECG indicates delayed myocardial ischemia recovery due to impaired postischemic hyperemic blood flow (figure1). This study aimed to validate τ-icECG for coronary artery stenosis assessment against the reference of fractional flow reserve (FFR).

Material & Methods: This prospective observational study enrolled patients with chronic coronary syndrome. All patients underwent hemodynamic coronary stenosis assessment following a standardized coronary artery balloon occlusion with continuous icECG recording. Primary study endpoint was τ-icECG, defined as the time from coronary balloon deflation until the ST-segment shift reached 37% (= 1/e = mean lifetime) of its maximum level during coronary occlusion. τ-icECG was tested against the reactive hyperemia FFR, and quantitative percent diameter stenosis (%DS).

Results: 100 patients (29% female, age 66±10years) with a total of 124 vessels were enrolled. Mean τ-icECG was longer in vessels with FFR≤0.80 (n = 45) as compared to vessels with FFR >0.80 (n = 79): 19.2±21.3s vs 11.3±10.2s, p = 0.006. τ-icECG at a threshold of 9.3s detected a hemodynamically relevant stenosis (FFR≤0.80) with a sensitivity of 78% and a specificity of 56% (AUC 0.651, p = 0.005), and a structurally relevant stenosis (≥50%DS) by a threshold of 9.1s (sensitivity 53%, specificity 81%; AUC 0.680; p = 0.001).

Conclusion: IcECG ST-segment shift remission time, τ-icECG accurately detects FFR-positive coronary artery stenoses at a threshold of ≥9.3s with high sensitivity but modest specificity. While the pressure-based FFR-technique is a surrogate for epicardial coronary obstruction only, τ-icECG allows the detection of myocardial perfusion impairment at any point between the coronary ostium and the cardiomyocytes. Therefore, the modest specificity may arise from microcirculatory impairment not detectable by FFR. These findings suggest a potential role for τ-icECG in the evaluation of ischemia with non-obstructive coronary arteries, INOCA (figure2).

COI: No

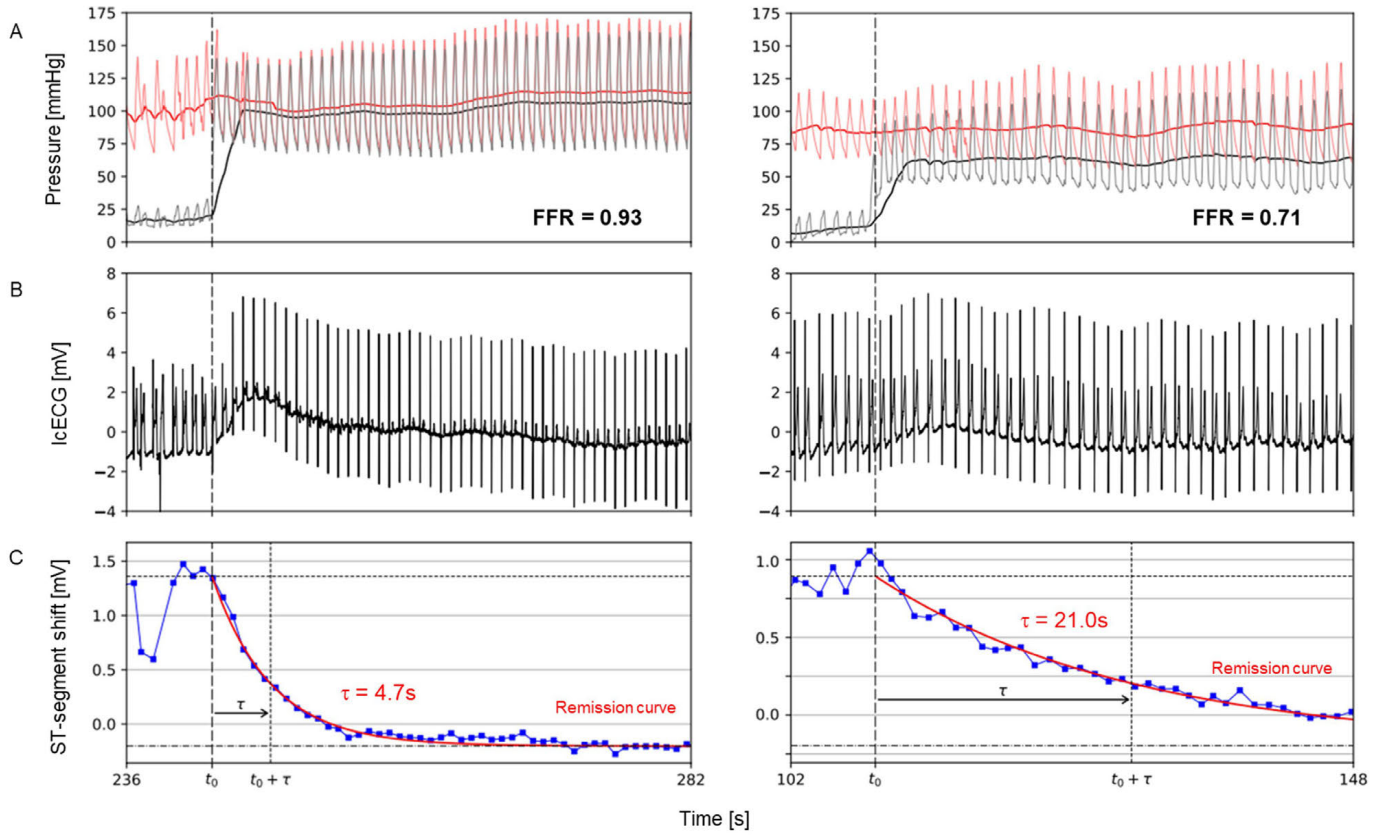


Figure 1) Algorithm analysis in hemodynamically relevant and non-relevant coronary artery stenoses A) Simultaneous recordings of mean and phasic aortic (red signals) and coronary occlusive (black signals) pressure at the end of a coronary artery occlusion and after the balloon deflation. FFR = fractional flow reserve **B)** icECG tracking **C)** Autonomous ST-segment tracking with the remission curve U(t) (red; plotted exponentially decaying curve for the calculation of τ-icECG). Vertical dashed line t0 = end of the occlusion, horizontal dashed line U0 = icECG ST-segment shift at the end of the occlusion, horizontal dashed and dotted linie U1 = initial ST-segment shift before the balloon occlusion.

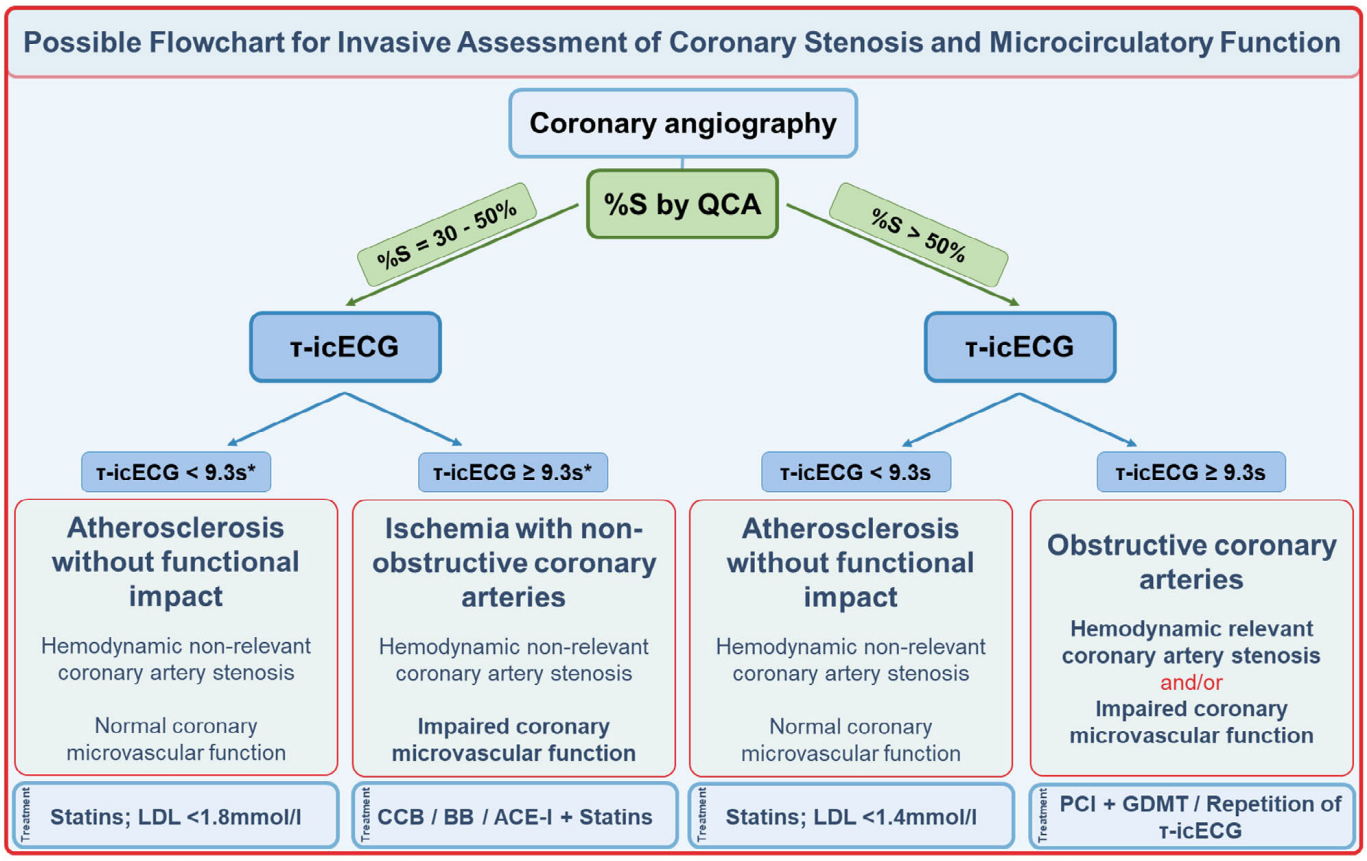


Figure 2 Flowchart of a new diagnostic approach for the physiologic assessment of intermediate coronary stenosis. *Exact cut-off to be evaluated in a dedicated clinical trial. %S = percent diameter stenosis; QCA = quantitative coronary angiography; T-icECG = intracoronary electrocardiogram ST-segment shift remission time; CCB = calcium channel blockers; BB = beta blockers; PCI = percutaneous coronary intervention; GDMT = guideline-direct medical therapy

O028

Management and Outcomes of Post-Infarction Ventricular Septal Defect: A Single-Center Experience

Matteo Chal[1], Georgios Giannakopoulos[1], Sarah Mauler-Wittwer[1], Raphaël Giraud[1], Mustafa Cikirikcioglu[1], Christoph Huber[1], Hajo Müller[1], Stéphane Noble[1]

[1]*Geneva University Hospitals (HUG), Geneva, Switzerland*

Introduction: Post–infarction ventricular septal defect (PIVSD) is a rare but life-threatening mechanical complication of acute myocardial infarction, associated with rapid hemodynamic deterioration and high mortality. Despite advances in reperfusion, PIVSD remains a major therapeutic challenge. Surgical repair is considered the standard of care, while transcatheter closure is an alternative for selected patients. Local real-world comparative data remain limited.

Material & Methods: We conducted a retrospective single-center study including adult patients with PIVSD between 2009 and 2025. Patients were classified according to initial management strategy: surgical repair, transcatheter closure, or best medical therapy. Clinical status and echocardiographic findings were assessed before and after intervention. The primary outcome was 30-day all-cause mortality.

Results: A total of 24 consecutive patients with PIVSD were included. Management strategies consisted of surgical repair (n = 14), transcatheter closure (n = 4), and conservative therapy (n = 6). Median age was 79 years [IQR 72–83]; 12 (50%) were male, with diabetes mellitus in 12 (50%), hypertension in 16 (66%), and prior smoking in 8 (33%). Despite multivessel coronary artery disease 12 (55%), PIVSD was related to a single culprit lesion in 17 (90%) cases, most often involving the anterior territory 16 (70%). Percutaneous coronary intervention was performed in 7 (29%) patients, while the remaining did not undergo revascularization. The median time from onset of acute myocardial infarction symptoms to PIVSD diagnosis was 30 hours [IQR 22–103]. At presentation, 16 (67%) were in cardiogenic shock. Mechanical circulatory support was used in 11 patients (46%), including 8 surgical (61%) and all transcatheter patients (100%). 30-day mortality was 58% overall (surgical repair 35.7%, transcatheter closure 100%, conservative 83%).

Conclusion: PIVSD remains associated with high early mortality despite contemporary management. Surgical repair was the predominant management strategy and was associated with the highest 30-day survival. Transcatheter patients died from infectious diseases and therapeutic withdrawal.

COI: No

O030

Different Temperature Strategies in Acute Type A Aortic Dissection Repair: A Multicenter Retrospective Analysis in Octogenerians

Laura Rings[1,2], Stephanie McKeown[2], Rasha Boulos[1], Luca Kilgus[1], Petar Risteski[1], Milan Milojevic[1], Héctor Rodríguez Cetina Biefer[1,2], Omer Dzemali[1,2]

[1]*Department of Cardiac Surgery, University Hospital Zurich, Zurich, Switzerland,* [2]*Department of Cardiac Surgery, City Hospital of Zurich – Site Triemli, Zurich, Switzerland*

Introduction: Acute type A aortic dissection (AADA) remains a lethal surgical emergency. In octogenarians, perioperative management is particularly challenging, as neurological injury,

coagulopathy, and renal dysfunction may strongly influence recovery. Optimal temperature management during circulatory arrest, which may significantly affect outcomes, remains debated. We evaluated outcomes in octogenarians undergoing AADA repair, focusing on the association between temperature strategy and early clinical results.

Material & Methods: We conducted a multicenter retrospective analysis of consecutive patients aged >80 years who underwent surgical repair for AADA between 2008 and 2024 at two centers. Patients were stratified by temperature strategy: deep hypothermic circulatory arrest (DHCA, <28°C), mild hypothermic circulatory arrest (MHCA, 28–34°C), and normothermic circulatory arrest (NCA, >34°C). Primary endpoint was in-hospital mortality. Secondary endpoints included operative times, neurological deficits, postoperative renal function, and transfusion/coagulation product requirements.

Results: 75 octogenarians, mean age 82.4 ± 2.2, were evaluated and stratified by temperature strategy: DHCA (n = 36), MHCA (n = 25), and NCA (n = 14). In-hospital mortality was 19% overall and did not differ significantly between strategies, a numerical trend favoured MHCA (DHCA 31%, MHCA 8%, NCA 14%; p = 0.093). Operative duration decreased with warmer strategies, and cumulative cardiopulmonary bypass, cross-clamp, circulatory arrest, and reperfusion times were shorter in the NCA group (all p <0.001). New neurological symptoms and postoperative creatinine levels were comparable across groups. Transfusion requirements were lower with warmer strategies: intraoperative red blood cell transfusion (DHCA 2.7±2.9 vs NCA 0.8±1.7, p = 0.009), platelet transfusion (DHCA 1.7±1.7 vs NCA 0.6±0.6, p = 0.025) and fibrinogen supplementation (DHCA 4.6±3.0 vs NCA 2.6±2.4, p = 0.034).

Conclusion: Warmer temperature strategies, particularly NCA, were associated with shorter procedures and substantially reduced allogeneic transfusion and coagulation requirements, without apparent adverse effects on early neurological or renal outcomes. These findings support NCA as a feasible and potentially resource-sparing strategy in this vulnerable population, warranting confirmation in larger cohorts

COI: No

O031

Underuse of long-term antithrombotic and anti-inflammatory therapies: a cross-national comparison between Switzerland and Germany

Michael Kunz[1], Marita Kieble[2], Andrea Espinosa Daudí[2], Lucas Lauder[1], Ulrich Laufs[3], Felix Götzinger[1], Jasper Boeddinghaus[1], Gregor Leibundgut[1], Christoph Meier[4], Christian Müller[1], Martin Schulz[2], Felix Mahfoud[1]

[1]Clinic of Cardiology, University Heart Center Basel, University Hospital Basel, Basel, Switzerland, [2]German Institute for Drug Use Evaluation (DAPI), Berlin, Germany, [3]Klinik und Poliklinik für Kardiologie, Universitätsklinikum Leipzig, 04103 Leipzig, Germany, Leipzig, Germany, [4]Clinical Pharmacy & Epidemiology, Department of Pharmaceutical Sciences, University of Basel, Basel, Switzerland

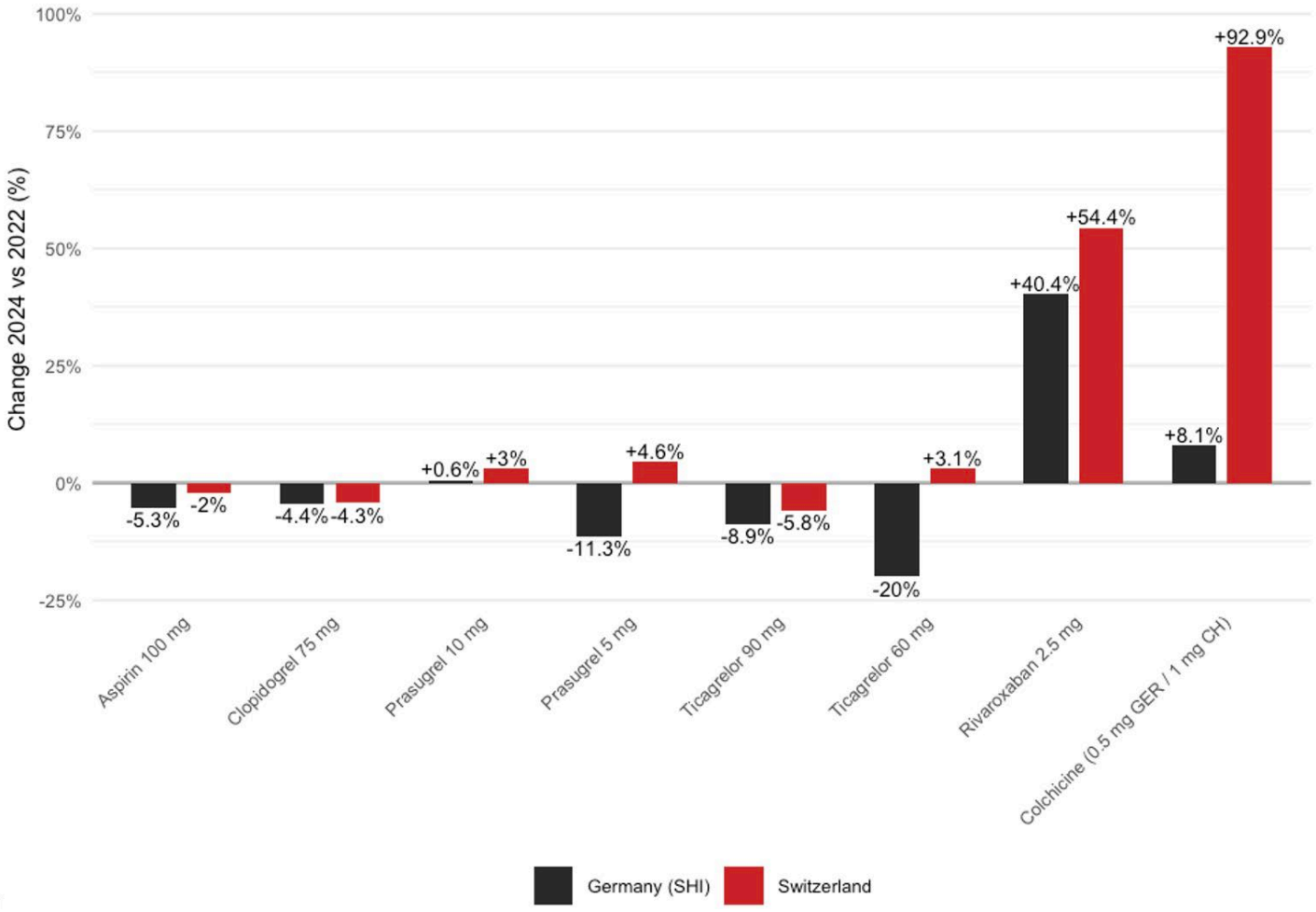
Introduction: Prolonged low-dose antithrombotic and anti-inflammatory therapies have shown to reduce cardiovascular events and are guideline-recommended in patients with coronary artery disease at high ischemic risk. However, their real-world uptake remains uncertain. This study aimed to quantify and compare the use of these therapies in Switzerland and Germany from 2022 to 2024.

Material & Methods: We analyzed national pharmaceutical delivery data from Switzerland and outpatient prescription data from Germany for low-dose monotherapy products containing aspirin (100 mg), clopidogrel (75 mg), prasugrel (5 mg and 10 mg), ticagrelor (60 mg and 90 mg), rivaroxaban (2.5 mg), and colchicine (0.5 mg in Germany, 1 mg in Switzerland). Drug utilization was standardized as the number of tablets per 1,000 persons per year. Cross-country comparisons and time trends were assessed.

Results: Aspirin 100 mg was the most frequently dispensed drug in both Switzerland and Germany (both >8,000 tablets/ 1,000 persons/ year). Clopidogrel ranked second in both countries. Ticagrelor (60 mg and 90 mg) was more frequently dispensed in Switzerland, whereas prasugrel 10 mg was more frequently dispensed in Germany. However, absolute use of both agents remained low (all <500 tablets/ 1,000 persons/ year). Rivaroxaban 2.5 mg was the only agent that showed a consistent increase over time (+54.4% Switzerland; +40.4% Germany). Colchicine use was low in both countries.

Conclusion: Despite minor country-specific differences in cardiovascular disease prevalence, the use of low-dose antithrombotic and anti-inflammatory therapies remains low in Switzerland and Germany, suggesting gaps in implementing guideline-recommended treatments in clinical practice.

COI: MKunz has received consulting fees from Merck (Merck KGaA).



Graphical abstract: **Underuse** of long-term **antithrombotic and anti-inflammatory therapies**: a cross-national **comparison** between **Switzerland and Germany**

Background

Prolonged low dose antithrombotic/ anti-inflammatory medical therapy is recommended by current ESC guidelines for secondary prevention in patients with CAD at high ischemic risk. Real-world utilization in Germany seems to be low and is unknown in Switzerland.

Methods

DAPI
National outpatient prescription **claims data**
+
IQVIA
National pharmaceutical **delivery data**

SHI
Individuals insured under **SHI in Germany**

Individuals living in **Switzerland (inhabitants)**

Drug utilization of aspirin, clopidogrel, prasugrel 5/10 mg, ticagrelor 60/90 mg, rivaroxaban 2.5 mg, colchicine 1 mg (CH)/ 0.5 mg (GER)

Tablets / 1,000 Inhabitants / 1 Year

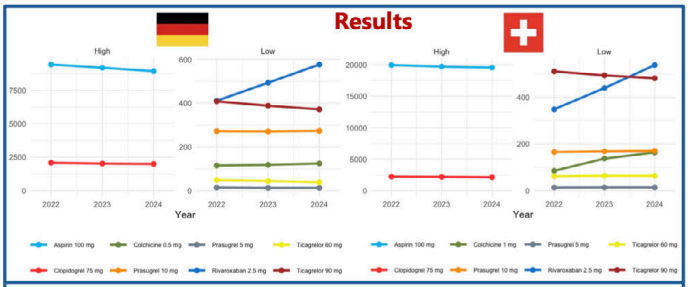
Standardized drug utilization:

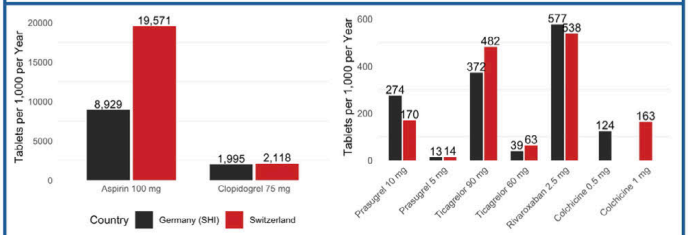
Switzerland: Number of tablets per 1,000 inhabitants per year

Germany: Number of tablets per 1,000 SH-insured persons per year

created with ChatGPT

Results





Rivaroxaban 2.5 mg: +40% and +54% from 2022 to 2024

Conclusion

Despite **modest country-specific differences**, overall use of low-dose antithrombotic and anti-inflammatory therapies **remains low in both countries**, suggesting **gaps in the implementation of guideline-recommended** secondary prevention.

RAPID FIRE ABSTRACTS: INTERVENTIONAL CARDIOLOGY & CARDIAC SURGERY

O032

Recurrent visits to the emergency department for suspected acute myocardial infarction: patient profiles, resource utilization, and clinical outcomes

Iryna Vyshnevska[1], Julia Reinhardt[1], Tzu-Ying Tsao[1], Ivo Strebel[1], Jasper Boeddinghaus[1], Ksenija Slankamenac[2], Stephan Steuer[3], Philipp Schuetz[4], Michael Christ[5], Christian Müller[1]

[1]Cardiovascular Research Institute Basel (CRIB) and Department of Cardiology, University Hospital Basel, University of Basel, Basel, Switzerland, [2]Emergency Department, University Hospital Zurich, Zurich, Switzerland, [3]Emergency Department, St Clara Hospital, Basel, Switzerland, [4]Medical University Department, Division of General Internal and Emergency Medicine, Aarau, Switzerland, [5]Emergency Department, Kantonsspital Luzern, Luzern, Switzerland

Introduction: The frequency of recurrent emergency department (ED) presentations for chest pain remains insufficiently investigated. These patients undergo repeated diagnostic tests and use more healthcare resources, highlighting the importance of understanding this pattern. The clinical characteristics and predictors of recurrent presentations remain unclear. This study aimed to define the clinical profile of patients with recurrent ED visits for suspected acute myocardial infarction (AMI).

Material & Methods: We included 34,414 patients presenting with chest pain from the international multicenter PRESC1SE-MI cohort across 19 sites, including Zurich, Basel, Aarau, and Lucerne. All had at least one high-sensitivity cardiac troponin test within 6 hours of arrival. Recurrent presentation was at least one ED revisit within 30 days of the index visit. We used χ^2 or Mann–Whitney U tests for group comparisons and multivariable logistic regression to identify predictors of 30-day ED return.

Results: Overall, 3.4% (n = 1,165) of patients re-presented to the ED within 30 days. Compared to those with a single ED visit, patients with recurrent presentations were older (median 62 vs 58 years, p <0.001), more often transported by ambulance (31.5% vs 27.7%, p <0.001), and more frequently hospitalized during the index visit (25.8% vs 24.3%, p = 0.010). Median ED length of stay was longer (5.7 [IQR 3.9–9.0] vs 5.3 [IQR 3.5–7.9] hours, p <0.001), and peak hs-cTn concentrations were higher (median 9.0 [IQR 4.0–23.2] vs 6.0 [IQR 3.0–17.5], p <0.001). In multivariable analysis, 30-day ED re-presentation was independently associated with known coronary artery disease (adjusted OR 3.03, 95% CI 2.46–3.73), prior myocardial infarction (adjusted OR 1.43, 95% CI 1.18–1.74), and prior coronary revascularization (adjusted OR 1.59, 95% CI 1.27–1.99; all p <0.001).

Conclusion: Recurrent ED visits for suspected AMI are linked to repeated evaluations and longer ED stays, underscoring the need for better continuity of integration between emergency and outpatient services.

COI: No

O033

Age-related delays and outcomes in elderly patients with ST-elevation myocardial infarction

Wesley Bennar[1], Dorian Garin[1], Puricel Serban[1], Thaïs Pittet[1], Selma Cook[1], Sonja Lehmann[1], Pascal Meier[1], Mario Togni[1], Stéphane Cook[1]

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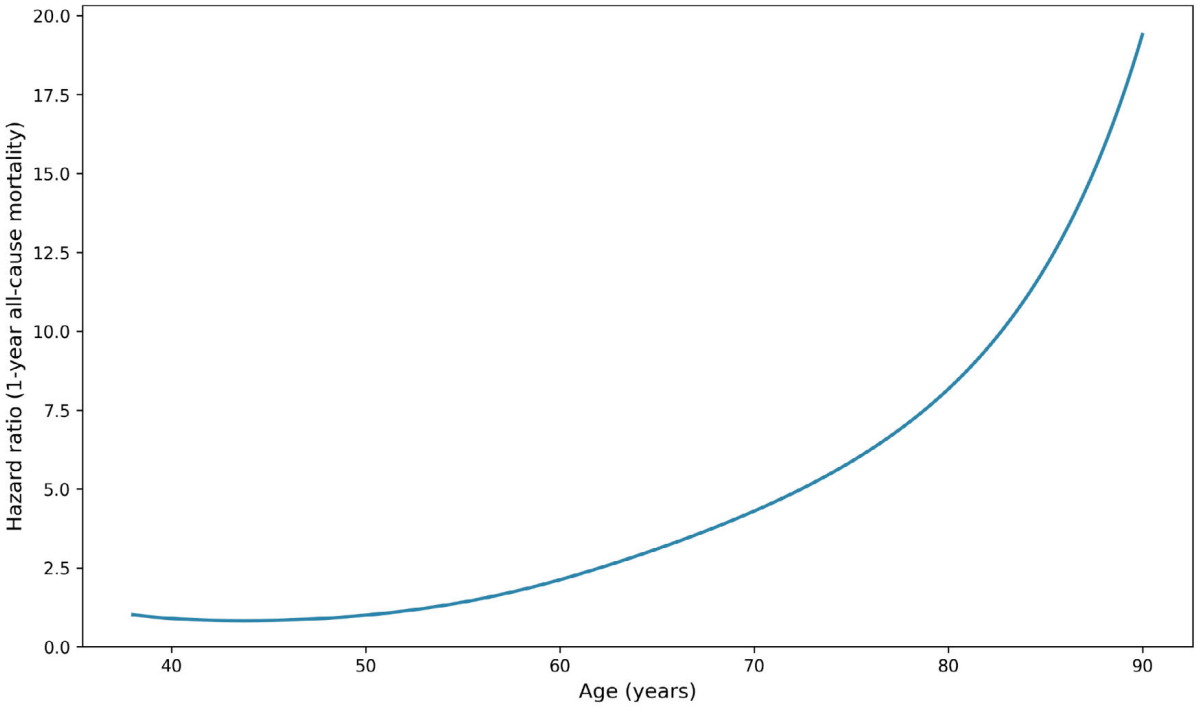
Introduction: Older patients with ST-elevation myocardial infarction (STEMI) experience higher mortality and longer treatment delays. Whether these disparities persist within structured fast-track primary percutaneous coronary intervention (PCI) networks, and how frailty modifies outcomes, remains insufficiently characterized. We aimed to assess the impact of advanced age and frailty on treatment delays, left ventricular function, and survival in a contemporary STEMI network.

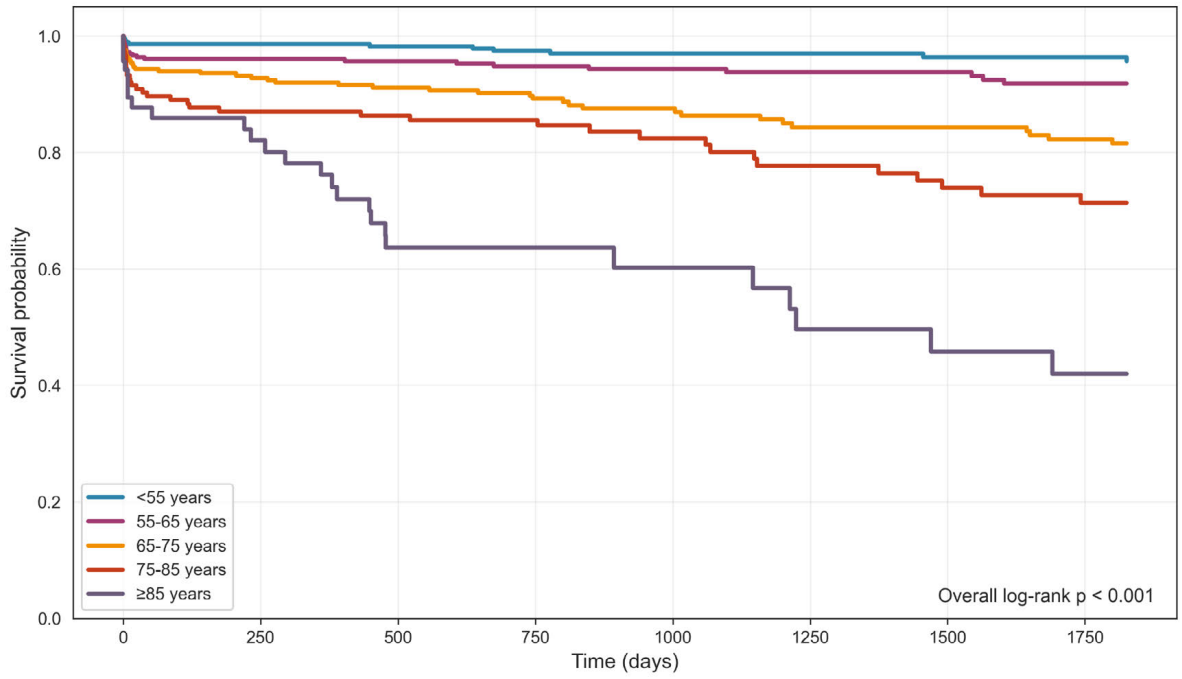
Material & Methods: We analysed consecutive STEMI patients enrolled in the prospective EVALFAST registry at a tertiary PCI centre between 2008 and 2024. Patients aged ≥85 years formed the geriatric cohort. Primary endpoints were time intervals from first medical contact (FMC) to diagnosis, diagnosis-to-balloon, and FMC-to-balloon. Secondary endpoints included left ventricular ejection fraction (LVEF), infarct size (peak creatine kinase-MB), and all-cause mortality at 30 days, 1 year, and 5 years. Frailty was assessed using the Clinical Frailty Scale. Outcomes were analysed using adjusted regression models, Cox proportional hazards, and standardized mortality ratios.

Results: Among 1,330 STEMI patients, 71 (5.3%) were aged ≥85 years. Compared with younger patients, the geriatric cohort had significantly longer FMC-to-balloon times (+36.7 minutes, p <0.001), driven mainly by prolonged diagnosis-to-balloon intervals (+9.1 minutes, p = 0.031). LVEF was lower in elderly patients (39.4% vs 48.8%, p <0.001), while infarct size did not differ significantly. Thirty-day mortality was markedly higher in patients aged ≥85 years (hazard ratio 10.0, 95% CI 3.1–32.7), with excess mortality concentrated in the early phase and declining at longer follow-up. Frailty independently predicted mortality (hazard ratio 1.85 per Clinical Frailty Scale point, p <0.001).

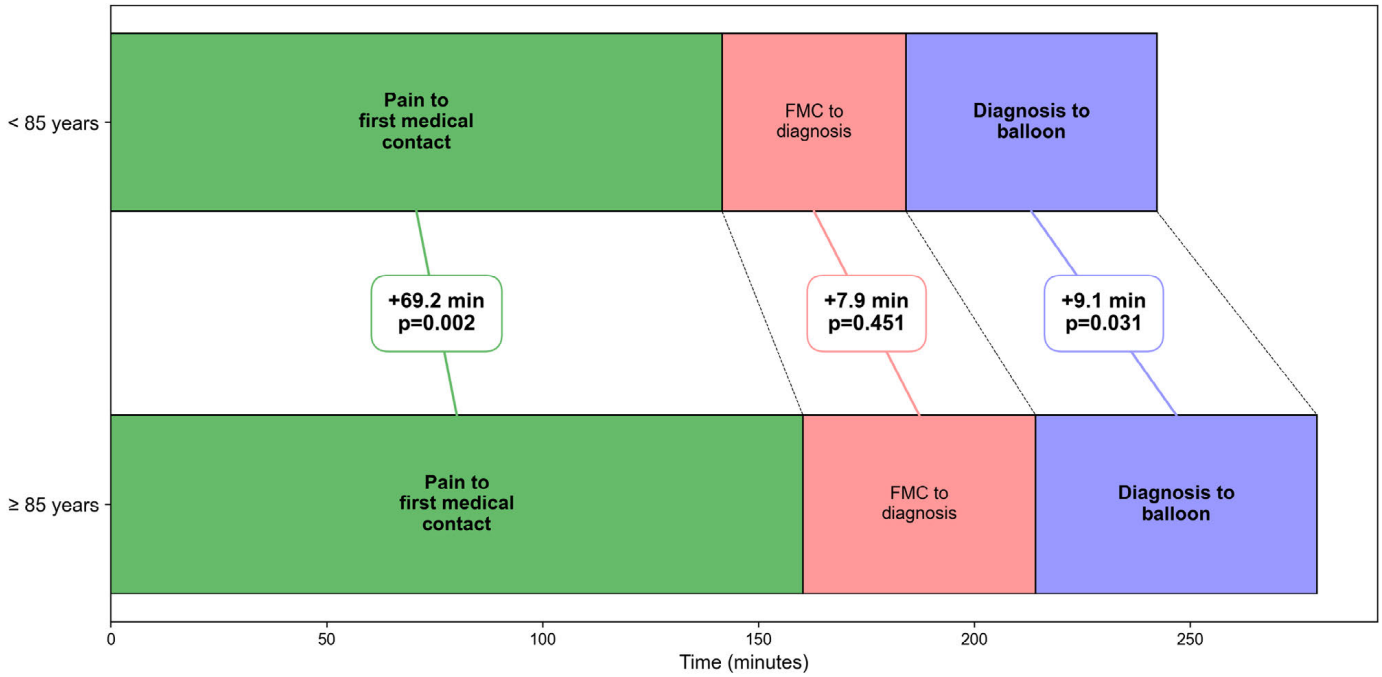
Conclusion: Despite a dedicated fast-track system, very elderly STEMI patients experience delayed reperfusion, reduced left ventricular function, and excess early mortality. Delays occur predominantly after diagnosis, suggesting system-level contributors. Frailty identifies patients at highest risk and should be integrated into STEMI care pathways to improve equity and outcomes.

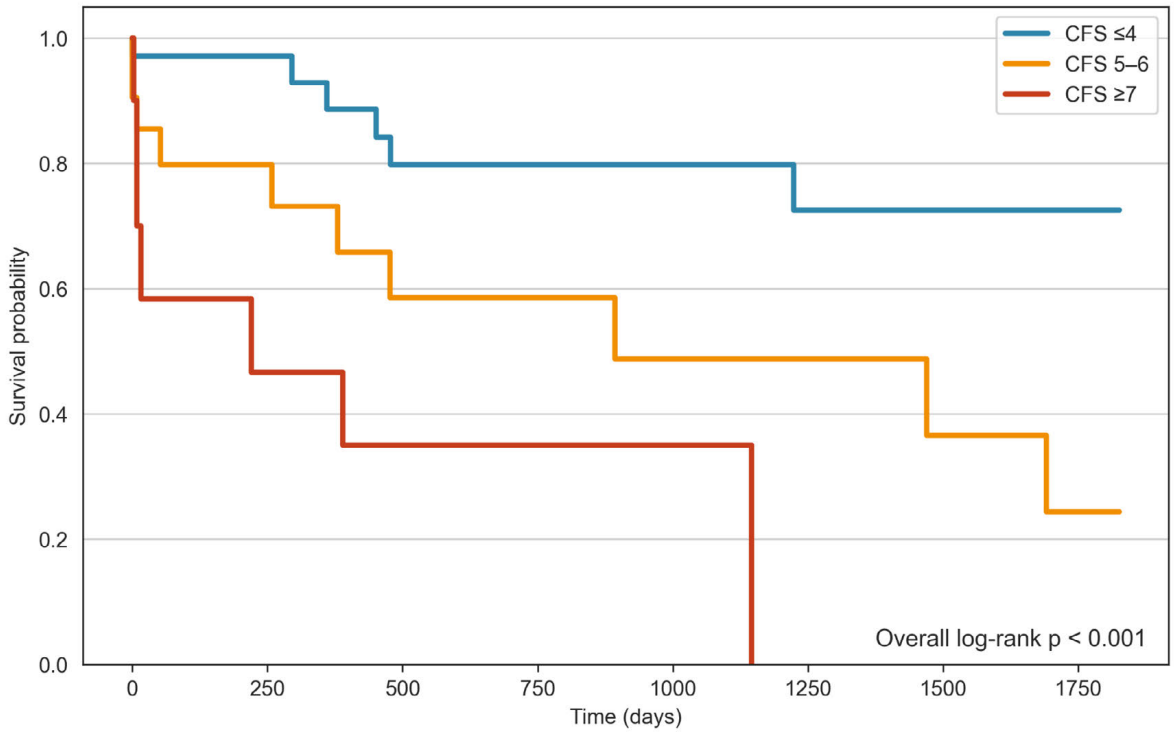
COI: No



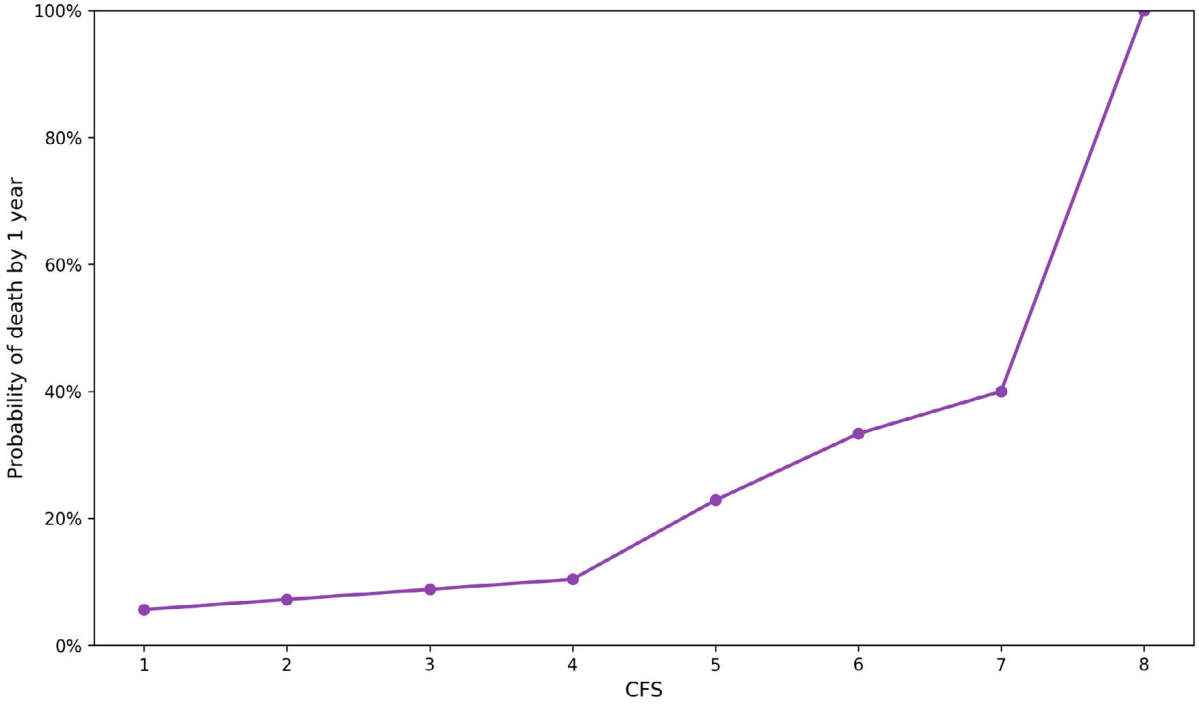


At risk								
<55 years	361	276	256	228	173	159	150	139
55-65 years	367	281	242	213	170	155	148	138
65-75 years	306	233	206	177	138	130	123	117
75-85 years	180	122	115	98	70	63	59	54
≥85 years	69	42	30	21	17	14	12	10





At risk								
CFS ≤4	34	23	18	12	11	10	9	8
CFS 5–6	21	12	8	6	4	4	3	2
CFS ≥7	10	4	2	2	1	0	0	0



O034

Impact of Access Management Strategies on Outcomes After Percutaneous Left Ventricular Assist Device Removal: Insights From a Real-World Registry

Lena Künzli[1], Firat Erdogan[1], Chiara Schaffner[1], Alessandro Spirito[1], Tobias Göldi[1], Yuan Zhi[1], Adrian Attinger-Toller[1], Florim Cuculi[1], Matthias Bossard[1]

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Introduction: The Impella pLVAD is increasingly used in high-risk procedures and cardiogenic shock but carries substantial access-site risks. As the optimal post-explant closure strategy remains uncertain, this study compared clinical outcomes across different vascular closure techniques.

Material & Methods: From an ongoing registry (NCT04117230), we analyzed consecutive patients treated with at least one fully percutaneously implanted Impella device. Between January 2014 and January 2026, 661 patients were enrolled, of whom 465 received an Impella as their first pLVAD. Clinical and management characteristics, including vascular closure techniques, were assessed. Patients with upfront surgical removal or death with the device in situ (n = 60) were excluded. Outcomes included bleeding, vascular complications, and access-site infections, each evaluated independently.

Results: Among 465 patients (mean age 67.3 ± 11.9 years; 76.6% male), CS was the leading indication for pLVAD implantation (57.7%). Access-site management used plug-based closure in 29.9%, suture-based closure in 18.1%, and manual compression in 16.1%. Major bleeding (BARC 3a) occurred in 1.7% with suture-based closure, 4.3% with plug-based closure, and 4.7% with manual compression ($p = 0.07$). Any access-site bleeding was most frequent with manual compression (61.3%), followed by plug-based (48.1%) and suture-based closure (25.0%) ($p < 0.0001$). Major vascular complications were rare; pseudoaneurysm occurred most often after manual compression (4.0%). Acute vascular complications were slightly more frequent with plug-based closure (4.5%) than with suture-based (3.8%) or manual compression (3.0%). Access-site infections occurred in 2.9% of plug-based, 5.3% of suture-based, and 4.8% of manual-compression cases. The 180-day mortality rate was similar across groups (19.4%).

Conclusion: Among Impella-treated patients, access-site complications – especially bleeding – are frequent, occurring in roughly one in four cases. Closure strategy appears to influence risk, with plug-based devices and manual compression performing less favorably than suture-based closure.

COI: I have received consulting and speaker fees from Abbott Vascular, Abiomed, Amarin, Amgen, Astra Zeneca, Bayer, Boehringer Ingelheim Boston Scientific, MedAlliance, Novartis, OM Pharma, SIS Medical and Shockwave by J&J Medtech.

O035

Circulating S1P as a novel target to address adverse outcomes following acute myocardial infarction

Carolina Balbi[1 2], Yifan Wang[1], Evanthia Pavlidou[1 2], Meret Allemann[1 2], Aurelia Seeberger[1 2], Simon Kraler[3], Giovanni G. Camici[1], Thomas F. Lüscher[1 4], Jürg Beer[1 2]

[1]Center for Molecular Cardiology, University of Zürich, Schlieren, Switzerland, [2]Department of Internal Medicine,Cantonal Hospital Baden, Baden, Switzerland, [3]Bern University Hospital, Inselspital, Switzerland, [4]Royal Brompton and Harefield Hospitals, Imperial and Kings College, London, United Kingdom

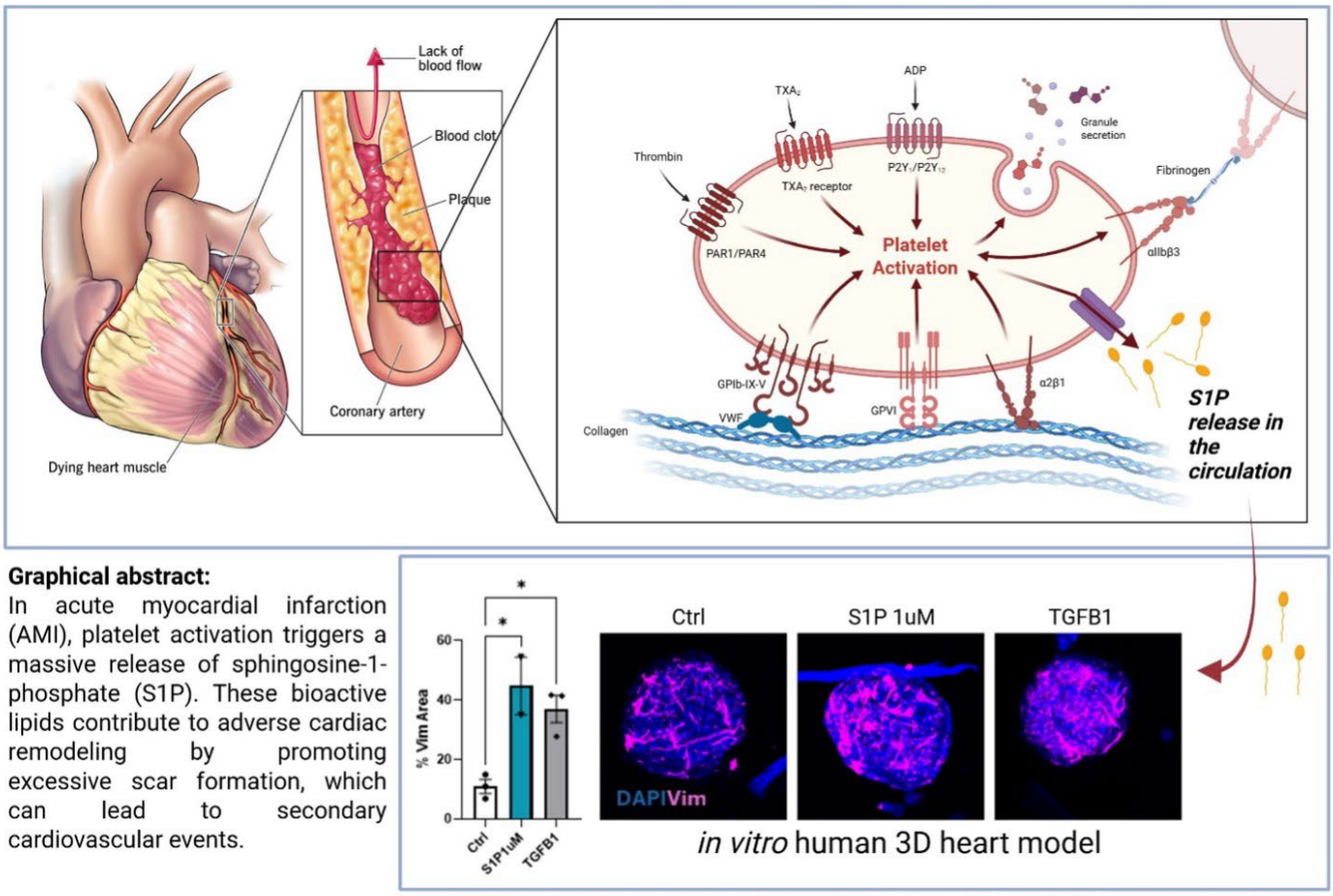
Introduction: Sphingosine-1-phosphate (S1P) is a bioactive lipid released by platelets during activation, particularly in the setting of acute myocardial infarction (AMI). Data on the role of S1P in AMI pathophysiology remains limited. This project aims to elucidate the mechanistic role of S1P in cardiac remodelling following AMI, and to evaluate its potential as a predictive biomarker of cardiovascular risk.

Material & Methods: Quantitative lipidomics on 440 human plasma samples obtained from AMI patients was performed. By harnessing human iPS-derived cardiomyocytes, human cardiac fibroblasts and 3D cardiac microtissue models, comprehensive *in vitro* analyses of the role of S1P in cardiac cells was performed.

Results: Lipidomic analysis of human patient samples revealed significantly elevated circulating S1P levels in individuals who later developed ischemic complications ($p < 0.05$), supporting its potential pathogenic role in post-MI outcomes. In vitro, S1P promoted fibroblast activation, evidenced by an increase in αSMA^+ cells and enhanced Collagen-1 secretion ($p < 0.05$ and $p < 0.001$, respectively), findings that were preliminarily confirmed in a 3D culture model. Additionally, S1P induced cardiomyocyte hypertrophy, as demonstrated by upregulation of NPPB and NPPA gene expression ($p < 0.05$ and $p < 0.001$, respectively) and increased cell area ($p < 0.001$), while concurrently reducing cell viability ($p < 0.001$). Finally, clinical data analysis indicated that elevated S1P levels were associated with increased susceptibility to secondary cardiovascular events ($p < 0.001$) and correlated with myocardial damage, as measured by CK-MB ($p < 0.01$).

Conclusion: By integrating molecular insights with clinical outcome data, we established S1P as a mechanistic driver and potential biomarker of post-MI remodelling and cardiovascular risk. These findings may support the development of novel diagnostic tools and therapeutic approaches aimed at mitigating post-infarction damage and improving both short- and long-term outcomes.

COI: No



Graphical abstract:
In acute myocardial infarction (AMI), platelet activation triggers a massive release of sphingosine-1-phosphate (S1P). These bioactive lipids contribute to adverse cardiac remodeling by promoting excessive scar formation, which can lead to secondary cardiovascular events.

O036

Impact of Aspirin Use on Mortality and Cardiac Allograft Vasculopathy After Heart Transplantation: A Systematic Review and Meta-analysis

Noémie Fournier[1], Panagiotis Antiochos[1], Anna Nowacka[1], Tamila Abdurashidova[1], Patrick Yerly[1], Philippe Meyer[2], Valentina Rancatti[1], François Verdy[1], Zied Ltaief[1], Matthias Kirsch[1], Roger Hullin[1], Henri Lu[1]

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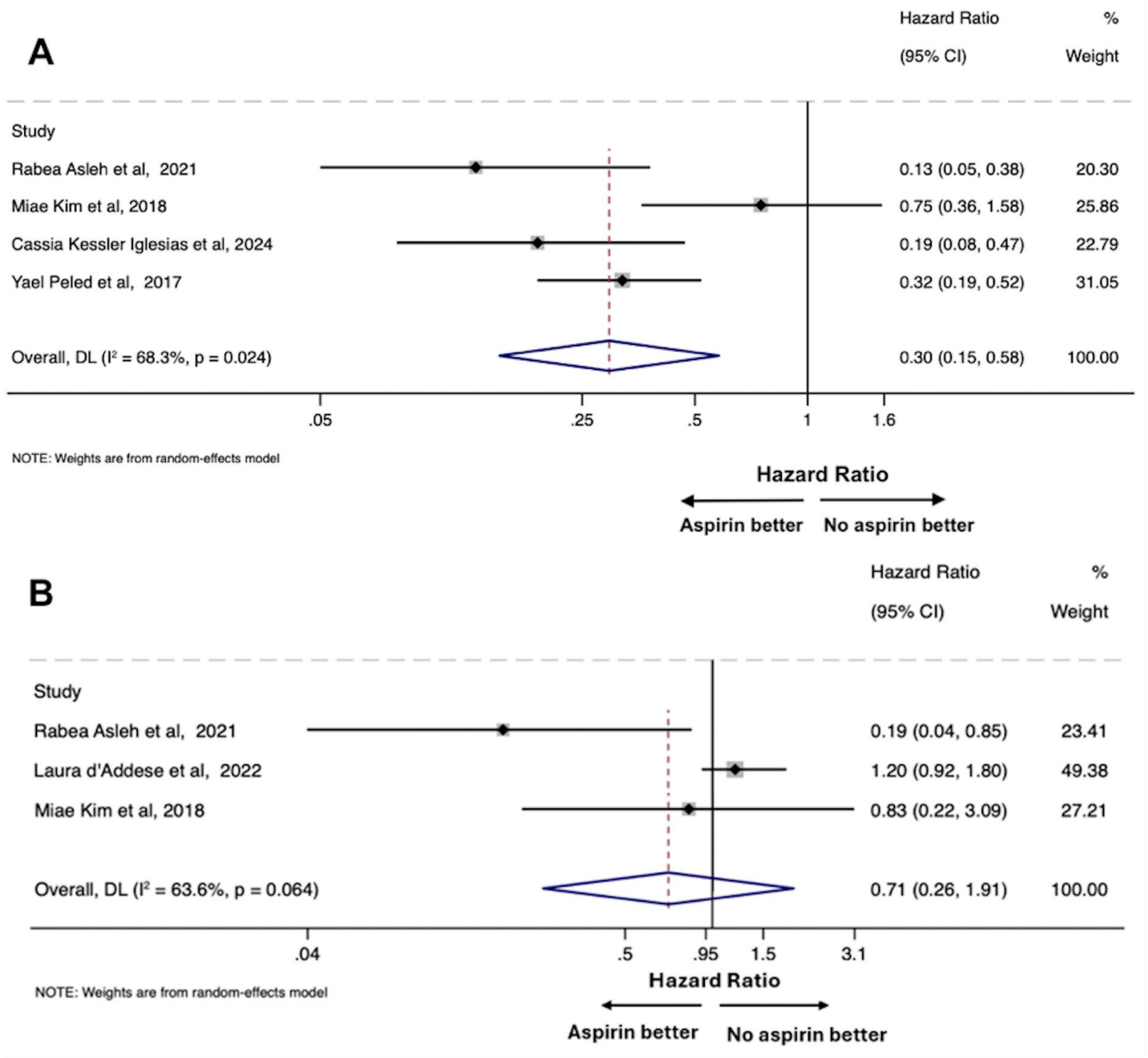
Introduction: Heart transplant (HTx) remains the treatment of choice for eligible patients with end-stage heart failure, with 5-year survival reaching ≈75% in contemporary cohorts. Cardiac allograft vasculopathy (CAV) is a major cause of long-term graft failure and post-transplant morbidity. Management of CAV is primarily preventive, focusing on cardiovascular risk factor control, rejection prevention, and mitigation of cytomegalovirus-related risk. Although aspirin is commonly recommended for the prevention of CAV, the evidence supporting a clinical benefit remains limited. We conducted a systematic review and meta-analysis to assess the association between aspirin use and clinical outcomes in HTx recipients

Material & Methods: The primary outcome was all-cause mortality. The secondary outcome was a composite endpoint of CAV related graft dysfunction and CAV related death. A systematic literature search of PubMed and EMBASE was performed, covering the period from inception to December 2025. Studies comparing the outcomes of interest in HTx patients treated with aspirin versus no aspirin were included. Hazard ratios (HRs) and their corresponding 95% confidence intervals (CIs) were meta-analyzed across studies.

Results: A total of five observational studies were included, comprising 1,983 HTx recipients (mean age ≈51 years, 793 men [40.0%]), of whom 730 received aspirin and 1,253 did not. Aspirin use was associated with a significant reduction in all-cause mortality (HR 0.30 [95% CI: 0.15–0.58]; Panel A). No significant association was observed between aspirin use and the secondary outcome (HR 0.71 [95% CI: 0.26–1.91]; Panel B).

Conclusion: In HTx recipients, aspirin use was associated with a significant reduction in all-cause mortality, with no clear effect on graft dysfunction or CAV progression, possibly reflecting limited power due to the small number of studies reporting this outcome. These findings underscore the need for well-designed prospective trials to better define the role of aspirin in CAV and post-transplant outcomes.

COI: No



O037

Performance of the novel high-sensitivity cardiac troponin T- Generation 6 assay in the early diagnosis of myocardial infarction in patients with prior coronary artery bypass grafting

Luca Koechlin[1,2], Jasper Boeddinghaus[2], Pedro Lopez Ayala[2], Emel Kaplan[2], Oscar Miro[3], Michael Christ[4], Dagmar Keller[5], Ivo Strebel[2], Felix Mahfoud[2], Héctor Rodríguez Cetina Biefer[1], Omer Dzemali[1], Christian Müller[2]

[1]Allianz Herzchirurgie Zurich, Zurich, Switzerland, [2]Cardiovascular Research Institute Basel (CRIB), Basel, Switzerland, [3]Emergency Department, Hospital Clínic, IDIBAPS, University of Barcelona, Barcelona, Catalonia, Spain., Barcelona, Spain, [4]LUKS, Luzern, Switzerland, [5]KLINIK GUT AG, St. Moritz, Switzerland

Introduction: The new high-sensitivity cardiac troponin (hs-cTn) T-Generation (gen) 6 assay has higher analytical sensitivity than the current hs-cTnT-gen5 assay. However, its performance in the vulnerable cohort of patients with prior coronary artery bypass grafting (CABG) presenting to the Emergency Department (ED) with chest pain is unclear. Therefore, we aimed to directly compare the performance of the hs-cTnT-gen6 assay with that of the established hs-cTnT-gen5 assay in patients who underwent CABG.

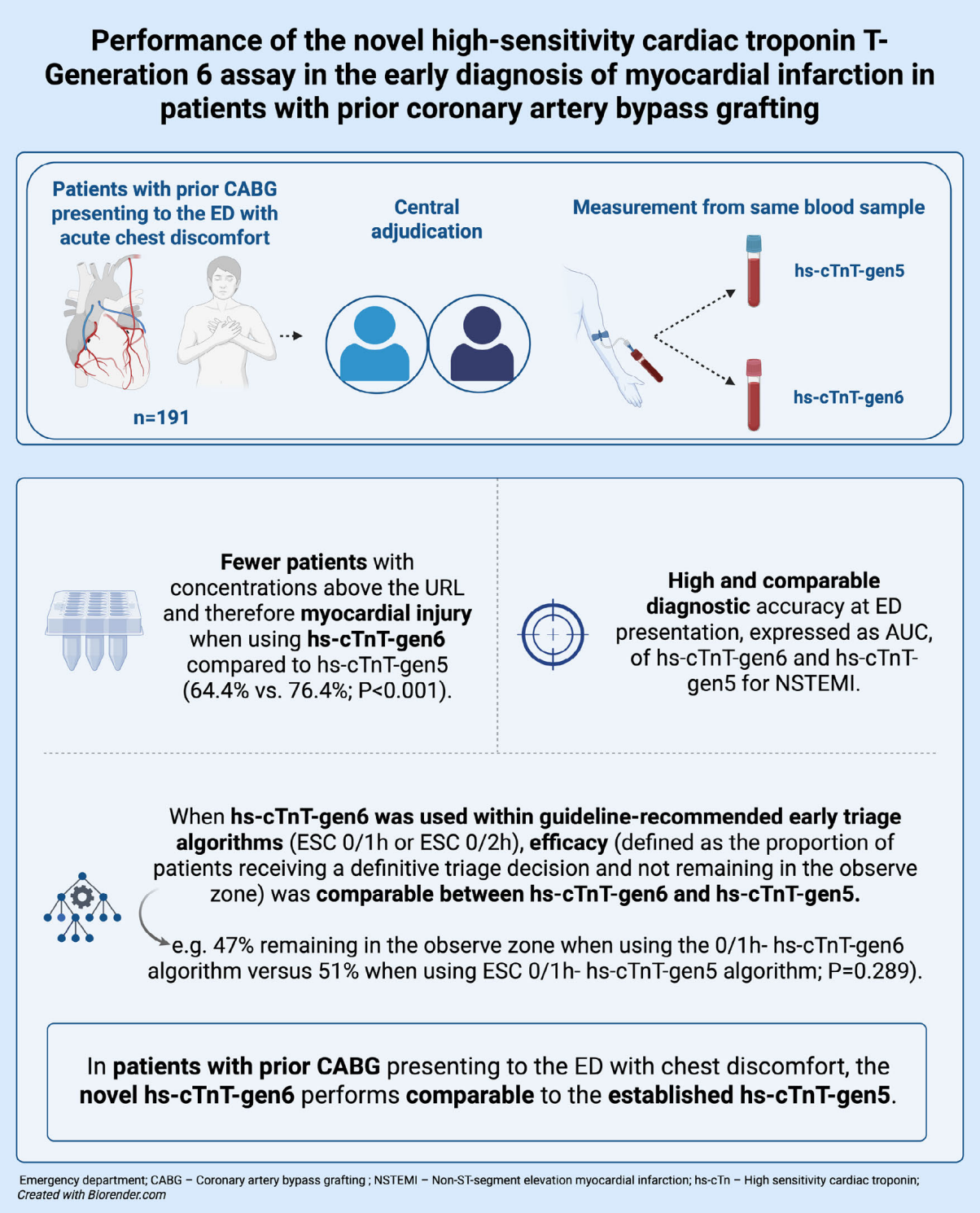
Material & Methods: In this secondary analysis of an international, prospective, diagnostic study with central adjudication, patients with prior CABG who presented with suspected myocardial infarction (MI) to the emergency department (ED) were enrolled. hs-cTnT-gen6 and gen5 were measured at the same timepoints. The diagnostic performance of hs-cTnT-gen6 was compared with that of hs-cTnT-gen5.

Results: Among 191 patients, MI was the final diagnosis in 74 (38.8%) patients. The proportion of patients with concentrations above the upper reference limit and therefore myocardial injury was lower for hs-cTnT-gen6 (64.4%) than for hs-cTnT-

gen5 (76.4%; P <0.001). Diagnostic accuracy at ED presentation, expressed as AUC, was 0.864 (95%CI: 0.812–0.916) for hs-cTnT-gen6 and 0.867 (95% CI: 0.816–0.919) for hs-cTnT-gen5 (P = 0.542). Applying the previously derived cutoffs for the ESC 0/1h- and 0/2h-hs-cTnT-gen6-algorithms triaged patients with 100% negative predictive value and sensitivity to rule-out MI. When hs-cTnT-gen6 was used within guideline-recommended early triage algorithms (ESC 0/1h or ESC 0/2h), the efficacy (defined as the proportion of patients receiving a definitive triage decision and not remaining in the observe zone) was comparable between hs-cTnT-gen6 and hs-cTnT-gen5.

Conclusion: In patients with prior CABG surgery presenting with chest discomfort in the ED, the novel hs-cTnT-gen6 assay demonstrated excellent and comparable diagnostic performance to the established hs-cTnT-gen5 assay with fewer patients identified as having myocardial injury. Use-optimized, assay-specific cut-offs for the ESC 0/1h- and 0/2h-algorithms provided very high sensitivity.

COI: No



O038

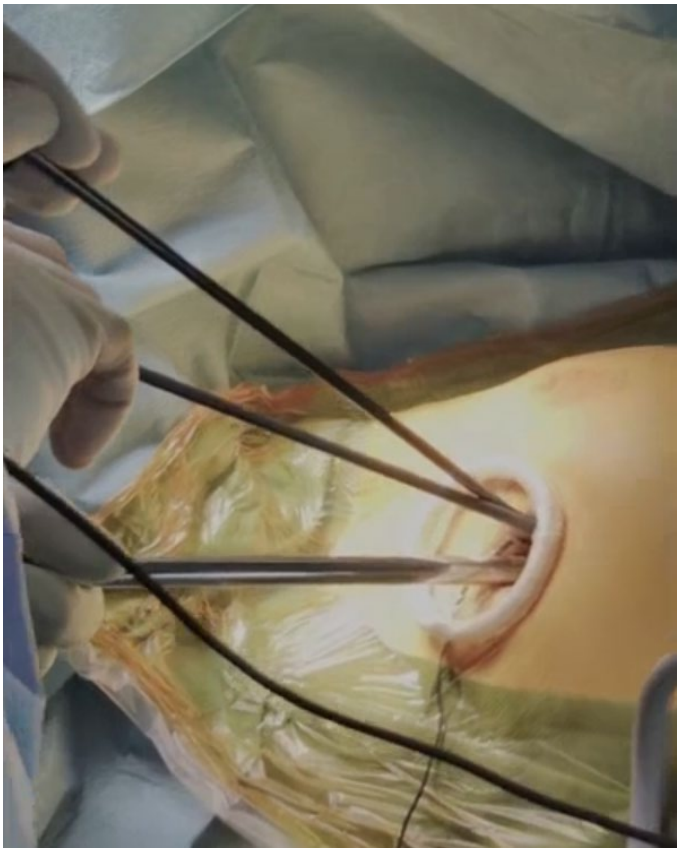
Single-port Thoracoscopic Pericardo-pleural Window For Pericardial Effusion: Early Outcomes From A Single-center Experience.:

Ziyad Gunga[1], Mathias Kirsch[1]

[1]*CHUV, Lausanne University Hospital, Lausanne, Switzerland*

Introduction: Pericardial effusion drainage is traditionally performed through subxiphoid or thoracotomic access. With advances in minimally invasive surgery, thoracoscopic drainage has emerged as a less invasive alternative offering superior visualization and reduced trauma. We report our single-center experience with a 2.5 cm single-port thoracoscopic pericardial drainage and creation of a pericardio-pleural window, highlighting feasibility, safety, postoperative comfort, and early outcomes.

Material & Methods: Between January 2023 and December 2025, 12 patients (mean age 66 ± 12 years, 60% male) underwent single-port thoracoscopic pericardial drainage via a right axillary mini-thoracoscopic approach. Indications included one traumatic effusion, two late postoperative effusion at 2 and 3 months respectively after mitral valve surgery related to excessive anticoagulation, one pericardial cyst associated with effusion, and eight idiopathic or inflammatory effusions. Procedures were performed under general anesthesia with single-lung ventilation. A 2.5 cm incision in the fifth intercostal space accommodated a 30° thoracoscope and standard instruments. In one complex case, concomitant pericardial drainage and inferior-lobe decortication for empyema were successfully performed through the same incision.



Results: Procedural success was 100%. Mean operative time was 45 ± 14 minutes, and mean drainage output 460 ± 190 mL. No intraoperative conversions occurred. No patient required re-exploration for postoperative bleeding; there were no in-hospital deaths. Median chest-drain duration was 2 days, and median hospital stay 3 days-two days shorter than in comparable thoracotomy cases. Postoperative pain scores were significantly lower compared with multiport thoracoscopic approaches. At three-month follow-up, all patients were asymptomatic with no recurrence.

Conclusion: Single-port thoracoscopic pericardial drainage is a safe, efficient, and minimally invasive alternative to conventional techniques. It provides excellent exposure, reduced pain, and a shorter recovery compared with multiport or open approaches. Its ability to treat concomitant thoracic pathology through the same incision further underscores its versatility and value in modern cardiac surgery.

COI: No

O039

First series of BIMA use during CABG in Women in Europe: Outcomes of over 500 Female Patients

Dritan Useini[1], Brigitta Gahl[1], Oliver Reuthebuch[1], Ingo Kutschka[1], Hassina Baraki[1]

[1]*Department of Cardiac Surgery, University Hospital Basel, University of Basel, Basel, Switzerland, Basel, Switzerland*

Introduction: Women are underrepresented in clinical trials regarding coronary artery bypass grafting (CABG). Furthermore, the utilization of multiple arterial grafts worldwide is still underused, especially in women. We provide short and mid-term clinical and angiographic outcomes after CABG of pioneering efforts in Germany of using bilateral mammary artery (BIMA) in women.

Material & Methods: Between 1989-1998, 599 women underwent CABG with BIMA. The primary end-point was all-cause mortality. Second end-points were: a composite of all-cause mortality, myocardial infarction and repeat revascularization, cardiac mortality and BIMA patency. 22 patients were lost to follow-up. 577 patients were included in the study. The mean follow-up time was 3.1 ± 1.4 years. At follow-up 112 patients underwent coronary angiography.

Results: The mean age was 63 ± 8.5 years. The mean body mass index was 26.9 ± 6.1 kg/m². Insulin dependent diabetes were 52 patients (8.7%). There were performed 1325 arterial anastomosis with BITA (average: 2.2) and 555 anastomosis with saphenous vein (average: 0.9). The 30-day mortality was 3%. Nineteen patients (3.2%) suffered from sternal wound healing disorders. The Re-thoracotomy rate was 2.8%. The overall 1-, and 3-year survival rates were 95, and 90%, respectively. The overall 3-year rate of freedom from a cardiac mortality was 97%. The overall 3-year rate of freedom from a composite of all-cause mortality, myocardial infarction and repeat revascularization was 83%. Four patients (0.7%) underwent Redo-CABG. From 112 angiography assessments there were: 8.1% left mammary, 21.8% right mammary and 48.7% venous grafts no patent, p <0.01.

Conclusion: Multiple arterial bypass grafting using BIMA in this historical collective of women patients with very good early and mid-term results are encouraging. The fact that BIMA grafts shows significantly better patency as saphenous vein grafts, arterial revascularization using BIMA should be preferred over venous grafts in women.

COI: No

RAPID FIRE ABSTRACTS: CARDIOVASCULAR BASIC SCIENCE

O049

Plasma adiponectin predicts 1-year MACEs in older acute coronary syndrome patients and is linked to increased endothelial tissue factor activity

Amedeo Tirandi[1], Luca Liberale[2], Francesco Bruno[3], Yifan Wang[4], Federico Carbone[2], Davide Ramoni[5], Margherita Moriero[5], Davide Di Vece[6], Fabrizio Montecucco[2], Thomas F. Lüscher[7], Giovanni G. Camici[8], Simon Kraler[9]

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Introduction: Adiponectin is an adipokine with documented cardiovascular protective effects in individuals without cardiovascular disease however, its role in high-risk patients after acute coronary syndrome (ACS) is unclear. This study assessed its association with major cardiovascular events (MACE) in ACS patients and examined its potential prothrombotic effects in primary human aortic endothelial cells (HAECs).

Material & Methods: Adiponectin was assessed by ELISA in 4'787 ACS patients (SPUM-ACS) whose plasma samples were collected prior to coronary angiography. The primary endpoint was 1-year MACEs, and the secondary was 1-year mortality. Uni- and multivariable Cox models were fit to assess independent associations of adiponectin levels with outcomes. Young (p6) and senescent (p16) HAECs were pre-incubated with different doses of adiponectin and then treated with tumor-necrosis factor-α to simulate pro-inflammatory effect. Cell lysates were assessed for tissue factor (TF), plasminogen activator-1 (PAI-1), and TF pathway inhibitor (TFPI) expression. TF activity was measured with a colorimetric assay.

Results: In patients with a recent ACS, high plasma adiponectin was linked to an increased risk of 1-year MACE (HR, tertile (T)3, 1.75, 95% CI, 1.30-2.34) and 1-year death (HR, 2.59, 1.71-3.93). For both primary and secondary endpoints, similar observations were made in multivariable-adjusted models accounting for established risk factors (adiponectin: HR, 1.59, 1.13-2.20 and 2.12, 1.30-3.45, respectively. *In vitro*, no differences in TF, PAI-1, or TFPI expression were found. However, TF activity was significatively higher in senescent cells for the highest adiponectin dose (Figure 1).

Conclusion: In patients with recent ACS, higher plasma adiponectin is associated with an increased risk of ischemic events. Adiponectin enhances TF activity in senescent HAECs, suggesting it may promote a persistent pro-coagulant state and elevate MACE risk after ACS. Further studies are required to confirm this mechanism.

COI: No

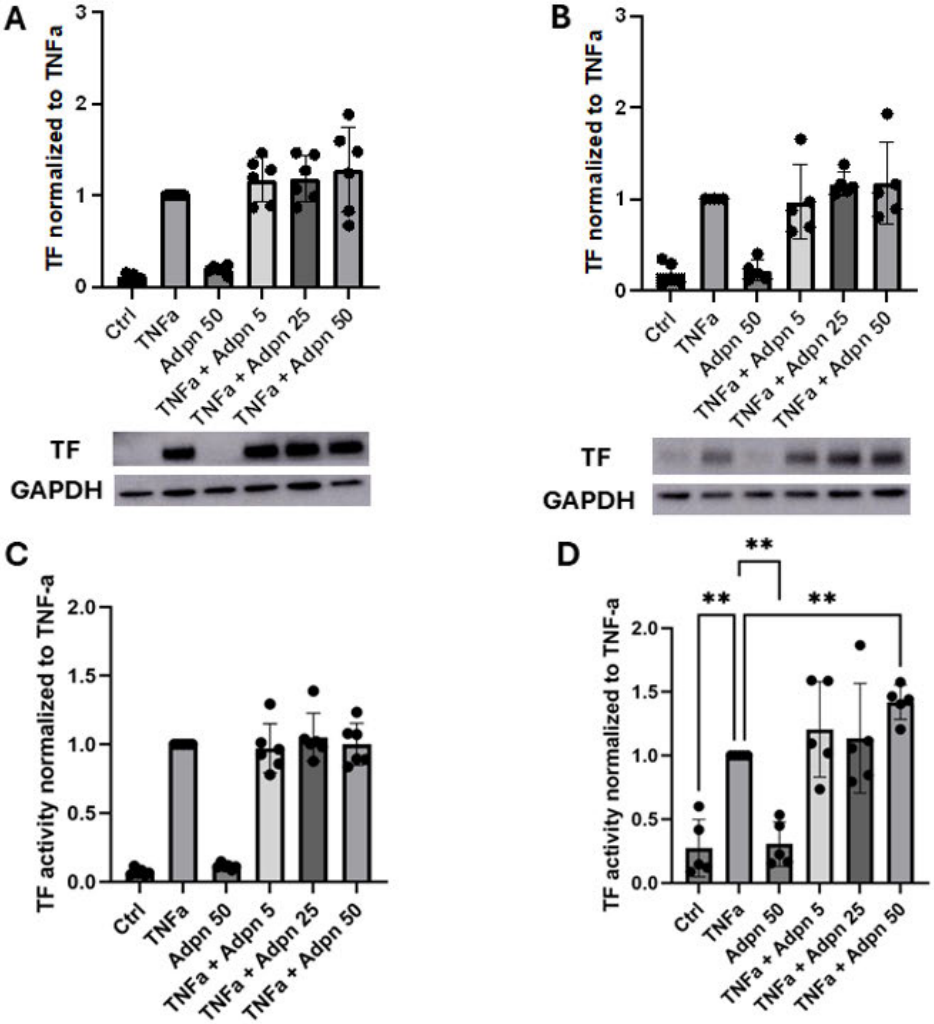


Figure 1. Tissue factor expression and activity in young and senescent human aortic endothelial cells.

Human aortic endothelial cells were pre-incubated with different doses of adiponectin for 18 h and then treated with tumor necrosis factor-α for another 5 h to simulate pro-inflammatory effect. Panel A: Tissue factor (TF) expression in young cells (passage 6; n = 6). Panel B: Tissue factor expression in senescent cells (passage 16; n = 5). Panel C: Tissue factor activity in young cells (passage 6; n = 6). Panel D: Tissue factor activity in senescent cells (passage 16; n = 5). Abbreviations: Adpn, adiponectin; Adpn 5, adiponectin 5 µg/mL; Adpn 25, adiponectin 25 µg/mL; Adpn 50, adiponectin 50 µg/mL; Ctrl, control; GAPDH, glyceraldehyde-3-phosphate dehydrogenase; TF, tissue factor; TNF-a, tumor necrosis factor-α 5 ng/mL.

O050

Vascular Rejuvenation by Fecal Microbiota Transplantation from Young to Old Mice

Meret Allemann[1], Carolina Balbi[1], Stefano Ministrini[1], Evanthia Pavlidou[1], Aurelia Seeberger[1], Pratintip Lee[1], Thomas Lüscher[1], Giovanni G. Camici[1], Jürg Beer[1]

[1]*Center for Molecular Cardiology, Schlieren, Switzerland*

Introduction: Vascular stiffness is one of the hallmarks of aging. In recent years, the ageing gut microbiome has been shown to be significantly altered towards a more dysbiotic state and associate with several risk factors for cardiovascular disease (CVD). Our objective was to investigate the therapeutic potential of the young microbiome in aged mice and its effects on vascular function.

Material & Methods: Fecal microbiota transplantation (FMT) from young male and female (2-months) C57BL/6 mice to aged (18-months) mice was done via oral gavage, followed by an engraftment period of four weeks. Pre and post FMT, echocardiography measurements were carried out on the heart and the carotid artery. Aortas were collected and snap frozen for mRNA and western blot or embedded in formalin for microscopy. Feces were analyzed using 16S rRNA sequencing of the V3-V4 region.

Results: Sequencing showed a shift in the species richness between the aged control and aged treatment groups, suggesting a partial gut microbial rejuvenation (Figure 1A). There was a clear and significant increase in PWV in the aged mice pre-FMT ($p < 0.01$) and a significant reduction after FMT ($p < 0.05$) (Figure 1B). Intima media thickness and lumen area increased in the aged, whereas elastin breaks showed a trend towards young levels in the transplanted aged (Figure 1C). Adventitial collagen deposition in the aorta (Figure 1C) and its protein expression was reduced after FMT. a-SMA protein levels decreased during aging in the male, and increased in the female. Senescence marker p16 was upregulated in the aged female mice ($p < 0.05$) and levels returned similar to the young after FMT. This data was also confirmed on the mRNA level (Figure 1D).

Conclusion: Our data show that gut microbial rejuvenation via FMT improves vascular function, possibly via endothelium and EM-dependent pathways, including senescence pathways.

COI: No

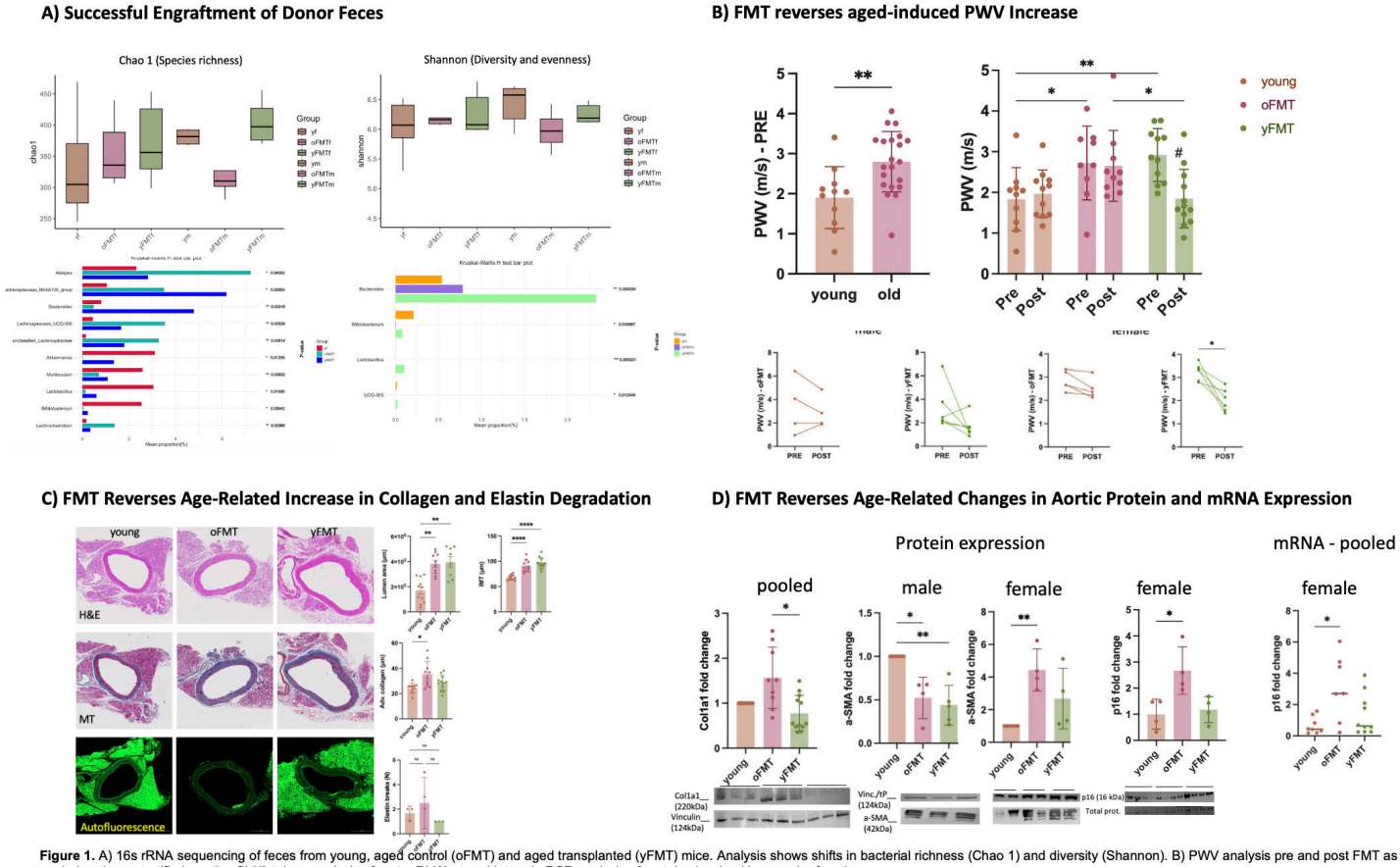


Figure 1. A) 16s rRNA sequencing of feces from young, aged control (oFMT) and aged transplanted (yFMT) mice. Analysis shows shifts in bacterial richness (Chao 1) and diversity (Shannon). B) PWV analysis pre and post FMT as pooled and sex-stratified results. C) Histology analysis of aorta. D) Western blot and qPCR analysis of proteins involved in vascular function.

O051

Systems Biology Identifies TARS2 as a Cardiomyocyte Regulator of Mitochondrial Oxidative Stress in Dilated Cardiomyopathy

Liming Chen[1], Yunzeng Zou[1]

[1]*Shanghai Institute of Cardiovascular Diseases, Zhongshan Hospital and Institute of Biomedical Sciences, Fudan University, Shanghai, China*

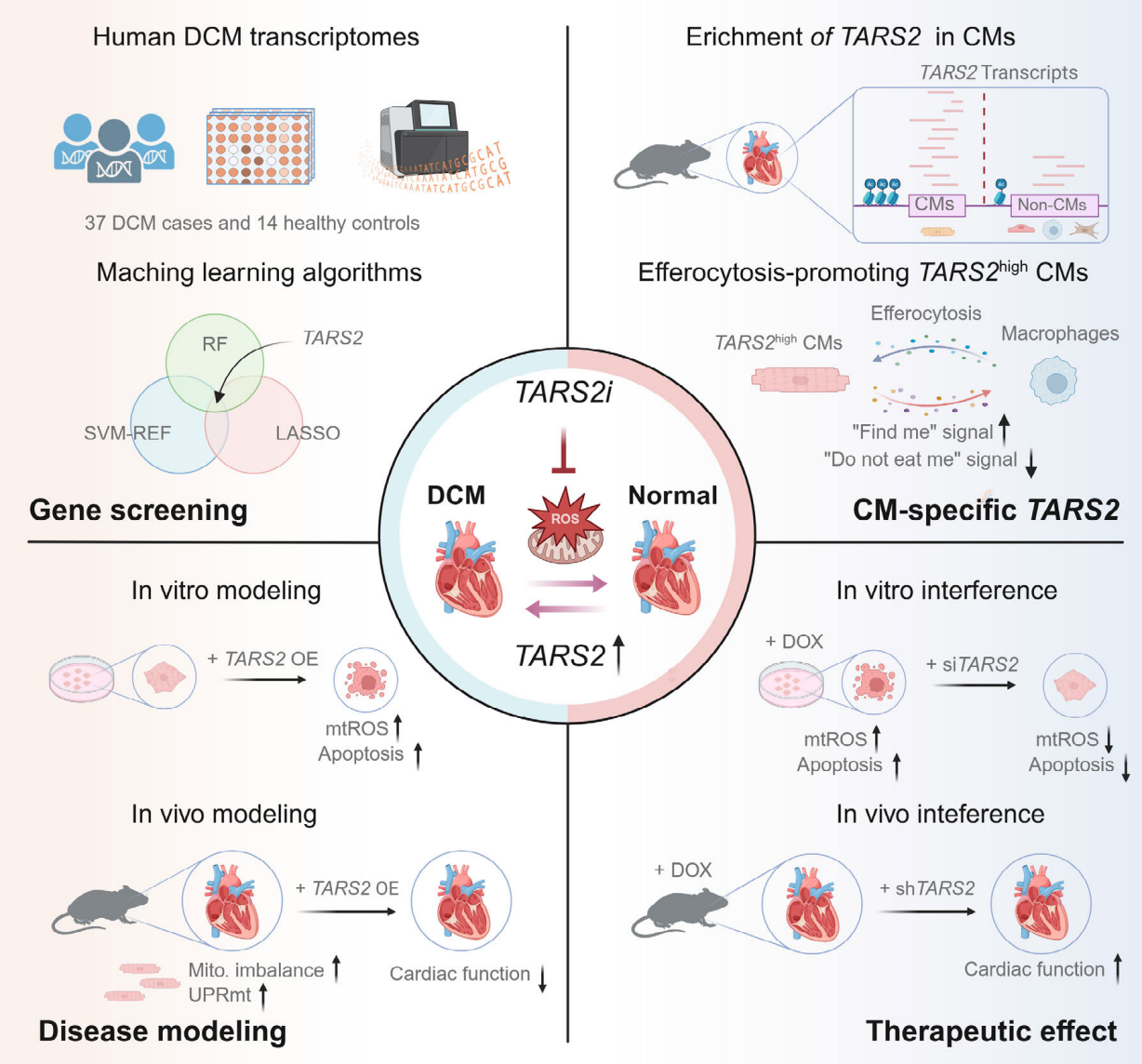
Introduction: Dilated cardiomyopathy (DCM) is a predominant cause of heart failure, characterized by progressive cardiomyocyte (CM) loss due to impaired oxygen and energy metabolism. Mitochondria, the primary cellular source of reactive oxygen species (ROS), play a critical role in response to oxidative stress in DCM. However, whether mitochondrial ROS (mtROS) dysregulation drives DCM progression or arises secondarily remains unclear, and the underlying mediators remain largely unknown.

Material & Methods: We applied bulk, single-cell RNA, and spatial transcriptomics to identify key regulators of mtROS in DCM. Candidate genes were prioritized using multiple machine learning models, and causality was evaluated by Mendelian randomization. Functional characterization of TARS2 was performed in human CMs and a mouse DCM model using overexpression (OE) and knockdown (KD) strategies.

Results: *TARS2*, *NOX4*, and *SNCA* were identified as top mitochondrial oxidative stress–associated genes with high diagnostic accuracy for DCM across machine learning models. *TARS2* was predominantly expressed in CMs and consistently upregulated in human DCM hearts. CMs with elevated TARS2 exhibited activation of apoptotic and efferocytic pathways, indicating increased cell death and enhanced pathological communication with macrophages. Conditioned medium from mouse CMs with *Tars2* overexpression or knockdown was applied to bone marrow–derived macrophages (BMDMs), confirming these effects. TARS2 OE disrupted mitonuclear balance, and induced CM apoptosis, which was mitigated by MitoTEMPO treatment. Drug-target Mendelian randomization demonstrated causal associations between *TARS2* expression and DCM risk, as well as adverse cardiac remodeling. In vivo, *Tars2* OE in mouse hearts induced DCM-like features accompanied by mitochondrial dysfunction, whereas genetic inhibition of *TARS2* alleviated mitochondrial abnormalities and CM apoptosis in vitro. Therapeutic benefits of *Tars2* KD were also observed in doxorubicin-induced cardiomyopathy, as evidenced by improved cardiac function.

Conclusion: This multi-modal study identifies TARS2 as a key regulator of mitochondrial oxidative stress in DCM and supports its potential as a diagnostic biomarker and therapeutic target.

COI: No



O052

When PANDA Attacks: A Novel lncRNA Driving Oxidative Stress in Diabetic Vessels

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Introduction: Long non-coding RNAs (lncRNAs) are emerging as pivotal players in the pathogenesis of cardiovascular disease. Recent work has shown that PANDA, a newly identified lncRNA, is a key regulator of cellular senescence, apoptosis and oxidative stress acting as decoy lncRNA.

Material & Methods: RNAseq was performed to human aorta specimen (diabetic vs no diabetic) and HAECs exposed to normal (NG) and high glucose (HG). PANDA modulations were performed by siRNA transfection and by the lipid transfection of PANDA carrying plasmid. Network perturbation amplitude (NPA) of treated HAECs unveiled transcriptional changes upon PANDA depletion. Expression of PANDA was assessed by real time PCR. RNA immuno-precipitation (RIP) was performed to check its binding to NRF2 and ChIP analysis was performed to check the NRF2-oxidative gene promoters binding.NRF2 was investigated by Immunofluorescence. Beta-galactosidase, superoxide staining and lipid peroxidation assay were used to detect ROS formation and senescence. Human vessels were treated with siRNA or plasmid and the activity was checked at the myograph.

Results: In diabetic human aorta as well in HAECs exposed to HG, PANDA levels were significantly increased. Upon PANDA removal, the upregulation of antioxidant genes (HMOX1, TFRC1) was observed. PANDA was involved mainly in NRF2 signaling, which is the main transcription factor of antioxidant genes. In HG, NFR2 is downregulated while PANDA silencing restores its level. Under HG condition PANDA binds the transcription factor NRF2 and blocks its nuclear translocation thus impeding its binding on gene promoters. Silencing of PANDA in HG-treated HAECs prevents cellular senescence, restores the expression of anti-apoptotic genes, and reduces ROS formation and lipid peroxidation, a marker of ferroptosis. After the depletion of PANDA vascular parameters were improved

Conclusion: In hyperglycemia PANDA upregulation drives oxidative stress. PANDA depletion rescues maladaptive transcriptional changes through NRF2 pathway improving vascular relaxation.

COI: No

Figure 1

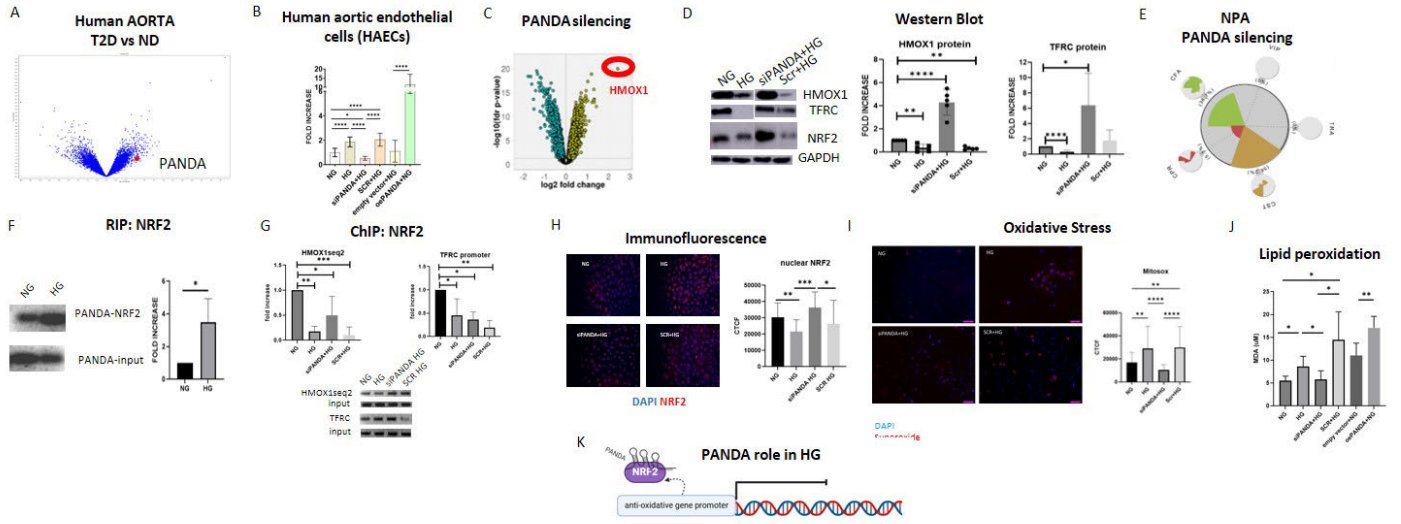

Figure1: in T2D aorta specimens the up-regulation of PANDA was observed **(A)**. HG treatment in HAECs reveals the up-regulation of PANDA **(B)**. After PANDA depletion, the RNAseq shows the increase of heme oxigenase1 (HMOX1)**(C)**. Protein analysis by western blot, demontrastes the restored level of HMOX1, TFRC and NFR2 after PANDA silencing **(D)**. Network protein assay (NPA) reveals that after PANDA silencing the cell fate (CFA); cell proliferation (CPR) and cell stress through NRF2 pathway (CST) are modulated **(E)**. RNA immunoprecipitation (RIP) analysis shows the binding of PANDA to NRF2 in HG condition **(F)**. ChIP analysis reveals that the depletion of PANDA increases the binding of NRF2 to HMOX1 and TFRC1 promoters **(G)**. Immunofluorescence staining shows the citosolic localization of NRF2 in HG, while in NG and PANDA silenced cell in HG NRF2 is mostly nuclear **(H)**. Oxidative stress is higher in HG condition and the depletion of the lncRNA reduces the production of superoxide**(H)**. Lipid peroxidation assay demonstrates that in HG as well in cells overexpressing PANDA in NG, the production of MDA is increased **(J)** Panda binds NRF2 from anti-oxidative gene promoters triggering oxidative stress **(k)**

O053

Zinc-alpha-2-glycoprotein is associated with poorer neurological outcomes in acute ischemic stroke and compromises blood–brain barrier endothelial integrity

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Introduction: Acute ischemic stroke (AIS) remains a major public health challenge, highlighting the need to identify new prognostic markers and therapeutic targets. Zinc-α-2-glycoprotein (ZAG), an adipokine involved in lipid and glucose regulation and known to influence inflammatory processes, has emerged as a molecule of interest in cardiovascular research. Despite the established role of inflammation in stroke pathophysiology, the potential contribution of ZAG in patients with stroke has not yet been investigated.

Material & Methods: Blood samples from 283 AIS patients were collected within 2 hours of symptom onset (baseline) and analyzed using enzyme-linked immunosorbent assay (ELISA) to measure levels of ZAG. Results were correlated with 90-day outcomes following the ischemic event. Given the critical role of the blood-brain barrier (BBB) in stroke-related inflammation, the effect of ZAG on endothelial integrity was assessed using primary human brain microvascular endothelial cells (HBMVECs), with barrier function monitored via transendothelial electrical resistance (TEER) measurements. Cells were treated with different doses of recombinant ZAG or PBS (control) and either underwent 4 hours of hypoxia followed by 72 hours of reoxygenation (to mimic AIS) or remained in normoxia (controls).

Results: Baseline ZAG levels predict unfavorable functional outcome in AIS patients at 90 days, assessed by Modified Rankin Scale (MRS 3-6 points) (adjusted OR: 1.07 with 95% CI: 1.02 – 1.13), independently of potential confounders, such as age, sex, NIH Stroke Scale/Score at baseline evaluation, and C-reactive protein circulating levels. On HBMVECs, recombinant ZAG caused a dose-dependent impairment of endothelial integrity, especially in hypoxic conditions.

Conclusion: Elevated baseline ZAG concentrations are associated with poorer functional outcomes at 90 days following AIS. Mechanistically, ZAG compromises endothelial integrity within the blood–brain barrier. Additional mechanistic studies are planned to further confirm the role of ZAG in AIS pathophysiology.

COI: No

O054

Smooth muscle cell–derived S100A4 regulates plaque composition and stability through metabolic control of phenotypic switching

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Introduction: S100A4, a calcium-binding protein, is crucial for smooth muscle cell (SMC) phenotypic switching, yet its specific role in atherosclerotic plaque development and SMC plasticity remains poorly understood. Our previous work using S100A4 neutralizing antibodies in ApoE$^{-/-}$ mice demonstrated reduced atherosclerotic burden but did not clarify which S100A4-expressing cell populations were responsible. Here, we investigated the direct contribution of SMC-derived S100A4 to plaque development and composition.

Material & Methods: We employed a carotid artery ligation model and a lineage-tracing ApoE$^{-/-}$ mouse model with SMC-specific S100A4 deletion (SMC-S100A4$^{\Delta/\Delta}$). Atherosclerosis and neointimal formation were assessed using en face Oil Red O staining, immunohistochemistry, and single-cell RNA sequencing (scRNA-seq) across multiple vascular beds. *In vitro* studies on S100A4 knockout (KO) SMCs evaluated proliferation, migration, mitochondrial function, and ultrastructure using metabolic assays and transmission electron microscopy (TEM).

Results: Neointimal hyperplasia was unchanged in S100A4KO mice compared with wild-type controls. However, in SMC-specific S100A4 deleted mice fed ahigh cholesterol diet, necrotic core area was reduced without altering total plaque size, indicating improved plaque composition. scRNA-seq revealed a shift in SMCs toward mesenchymal-like phenotypes and away from inflammatory states. Similarly, the myeloid compartment exhibited increased TREM2+ anti-inflammatory macrophages and fewer inflammatory subsets. Immunofluorescence of aortic roots showed increased YFP+ and YFP+Acta2+ SMCs in plaques of SMC-S100A4$^{\Delta/\Delta}$ mice suggesting more stable plaque. Transcriptomic analyses demonstrated downregulation of mitochondrial metabolism and oxidative phosphorylation genes across SMC clusters. Functionally, S100A4$^{-/-}$ SMCs displayed increased basal oxygen consumption and proton leak, altered mitochondrial number and morphology, and reduced migration, while proliferation remained unchanged.

Conclusion: Our findings identify SMC-derived S100A4 as a key modulator of plaque composition and stability rather than plaque size. S100A4 deletion promotes anti-inflammatory cellular states and alters SMC metabolism and plasticity, suggesting that S100A4 influences atherosclerotic plaque evolution through metabolic regulation of SMC phenotypic switching.

COI: No

O055

Investigating amyloid kinetics using a 3D in vitro model of wild-type transthyretin amyloidosis cardiomyopathy

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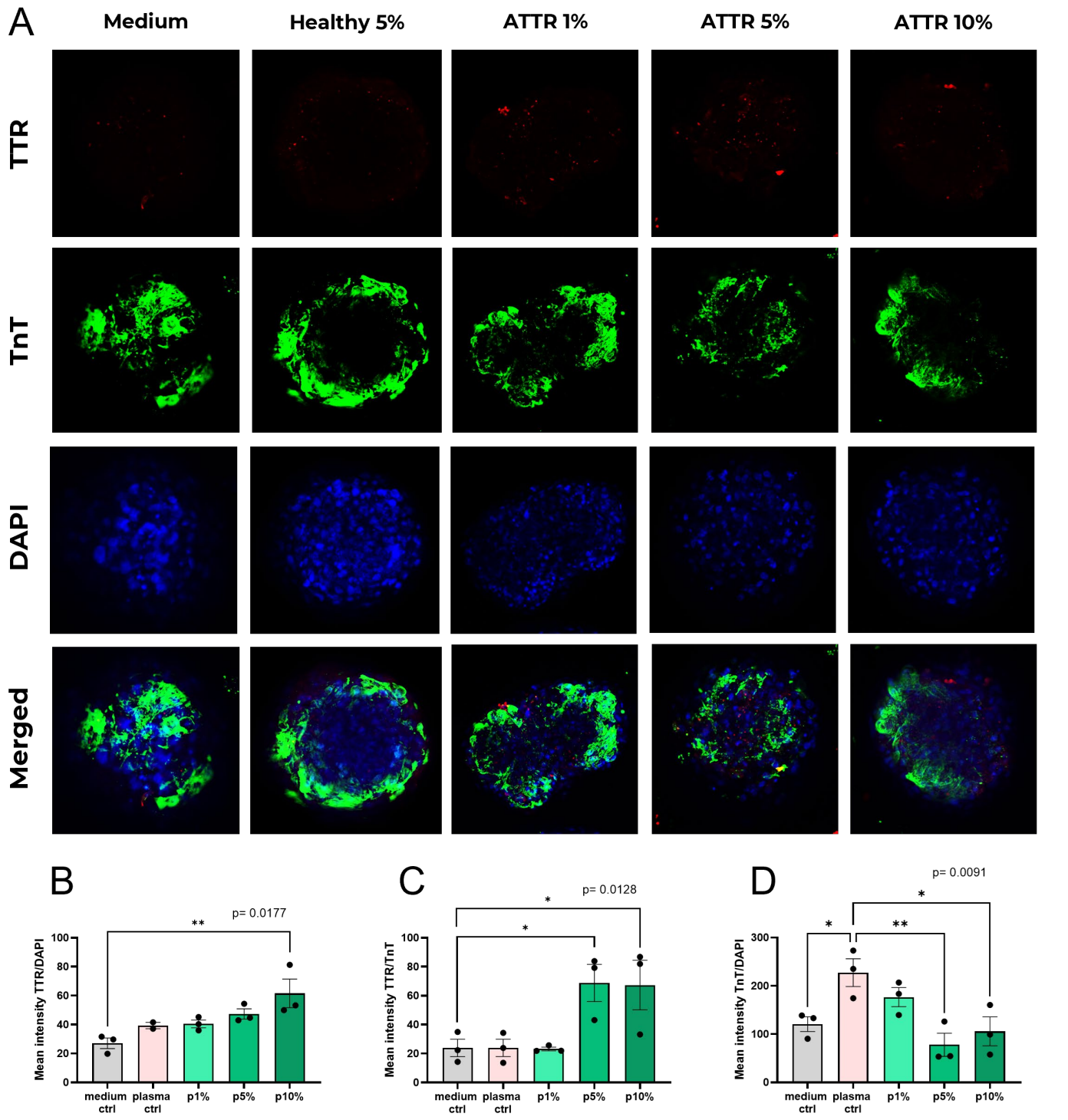
Introduction: Wild-type transthyretin amyloidosis (ATTR) is the most common form of cardiac ATTR [1]. The accumulation of misfolded transthyretin is the result of a complex protein kinetics, involving destabilization, misfolding, deposition and clearance of ATTR [2]; [3]. However, the molecular mechanisms underlying these processes are largely unknown [4]. Aim of this study is to investigate the kinetics of ATTR amyloid in a 3D in vitro model of human heart tissue.

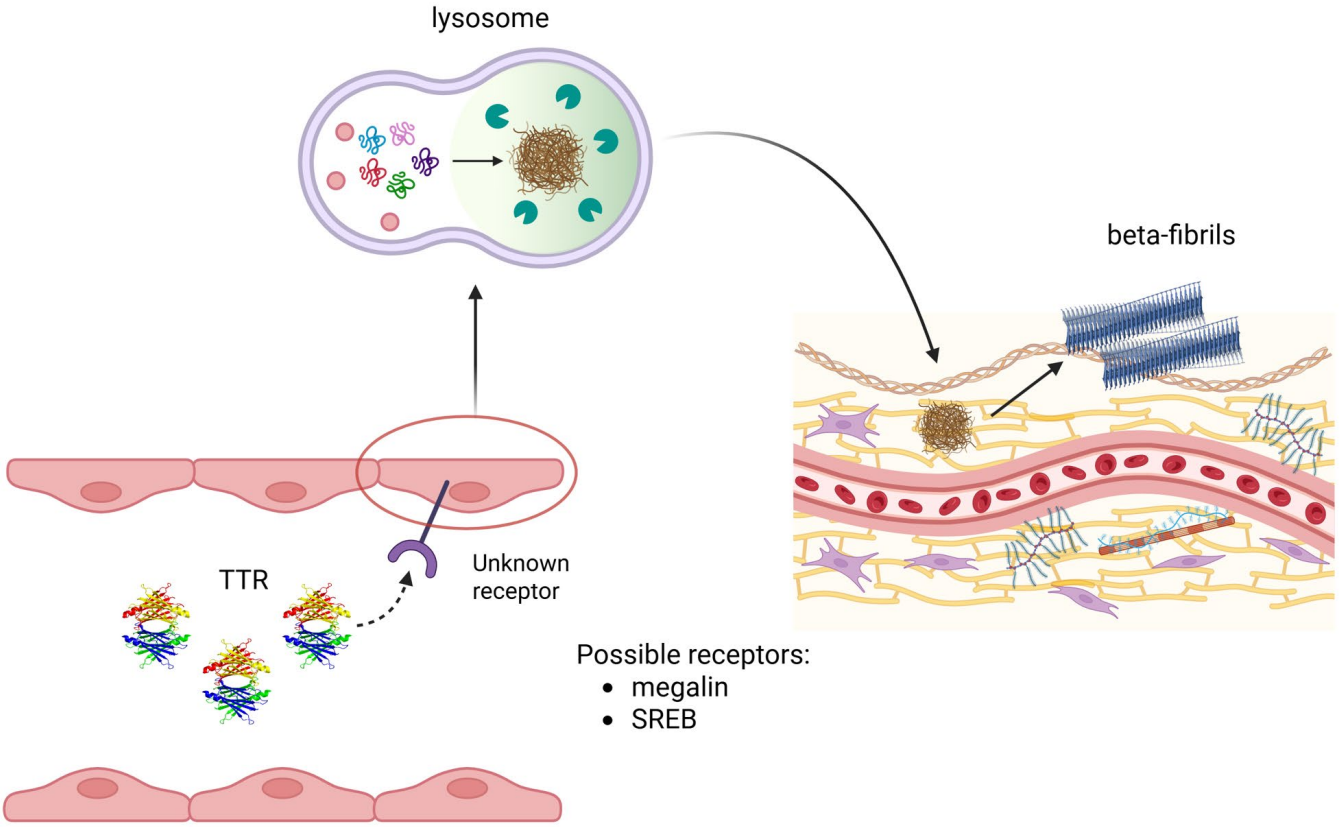
Material & Methods: Myocardial human microtissues (hMTs) were formed by combining defined ratios (4.5:1:1) of human induced pluripotent stem cells derived-cardiomyocytes (hCM), human primary Cardiac Fibroblast (hCF) and human aortic endothelial cells (HAECs) [5]. hMTs were incubated in medium with 0.5% FBS, 5% plasma from healthy subjects, or increasing concentrations of plasma from patients with wtATTR cardiomyopathy (1%, 5% and 10%). Each cell type was individually cultured in appropriate conditions and treated as described above. The accumulation of transthyretin was assessed by immunofluorescence, while cell damage was assessed by morphological analysis, and lactic dehydrogenase (LDH) and Troponin T (TnT) release in the medium.

Results: hMTs treated with plasma from patients with wild-type ATTR displayed unspecific signs of cellular injury, associated with a concentration-dependent increase of TTR content showing an extracellular pattern of accumulation (Figure 1). When analyzing each cell type separately, no significant accumulation of TTR, either intracellular or extracellular, is observed in fibroblasts or cardiomyocytes. A significant increase in intracellular content of transthyretin is observed instead in endothelial cells, without any sign of cell injury.

Conclusion: These results show for the first time that transthyretin is actively up-taken by endothelial cells before the deposition of ATTR in the extracellular space (Figure 2). Therefore, endothelial internalization could be a limiting step in the process of amyloidogenesis, representing a potential source of inter-individual variability the disease phenotype and a novel potential pharmacological target.

COI: No





RAPID FIRE ABSTRACTS: VALVES

O056

Effects of transcatheter tricuspid edge-to-edge repair on right heart dimension and function.

Stephanie Brunner[1], Lukas Stolz[2], Jörg Hausleiter[2], Stefan Toggweiler[1]

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Introduction: Transcatheter tricuspid edge-to-edge repair (T-TEER) has developed as treatment option for patients suffering from severe tricuspid regurgitation (TR). T-TEER consistently reduces TR, but data on subsequent right heart remodeling remain limited. Therefore, we aimed to evaluate changes in right heart dimension and function after T-TEER with extended echocardiographic follow-up.

Material & Methods: The study was based on the international EuroTR registry, including consequtive patients undergoing T-TEER for treatment of severe TR between 2016 and 2024 at 27 European heart valve centers. All patients with available baseline and follow-up echocardiographic assessments at ≥90 days post-procedure were eligible for inclusion.

Results: A total of 699 patients (mean age 78 ± 8 years, 56% female) were included. Echocardiographic follow-up was performed at a median of 445 days (interquartile range (IQR) 232, 893). Right heart dimensions significantly decreased: right ventricular (RV) mid-diameter from 40.2 ± 8.9 mm to 37.1 ± 8.4 mm ($p < 0.001$), end-diastolic area (EDA) from 28.7 ± 9.0 cm^2 to 26.2 ± 9.6 cm^2 ($p < 0.001$), and right atrial (RA) area from 36.6 ± 12.2 cm^2 to 35.7 ± 12.5 cm^2 ($p = 0.048$). Procedural success was significantly associated with reduction in RV dimension. Regarding RV function, a decline of fractional area change (FAC) from 37.8 ± 10.2% to 33.3 ± 10.1% ($p < 0.001$), and a decline of tricuspid annular plane systolic excursion (TAPSE) from 17.4 ± 4.7 mm to 16.6 ± 4.7 mm ($p < 0.001$) was observed.

Conclusion: Treatment of TR with T-TEER led to favorable long-term changes at echocardiographic follow-up assessment, reflected by reduced RV and RA dimensions. RV size reduction was significantly associated with procedural success. RV function showed a decrease after T-TEER, likely reflecting normalization of RV function after resolution of the prior hyperdynamic volume-overloaded state.

COI: No

Figure 1. Changes of right heart parameters.

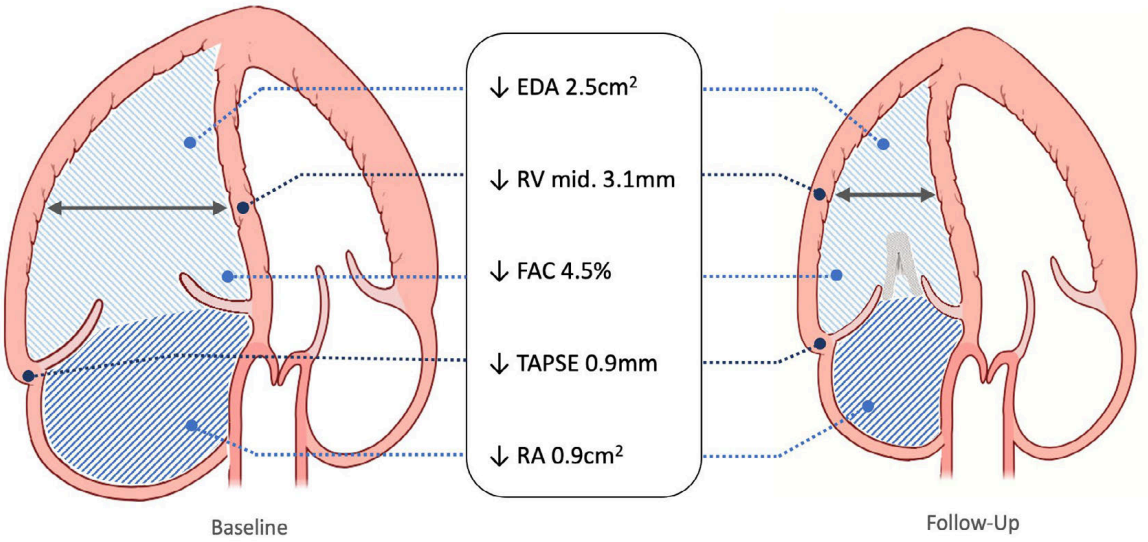
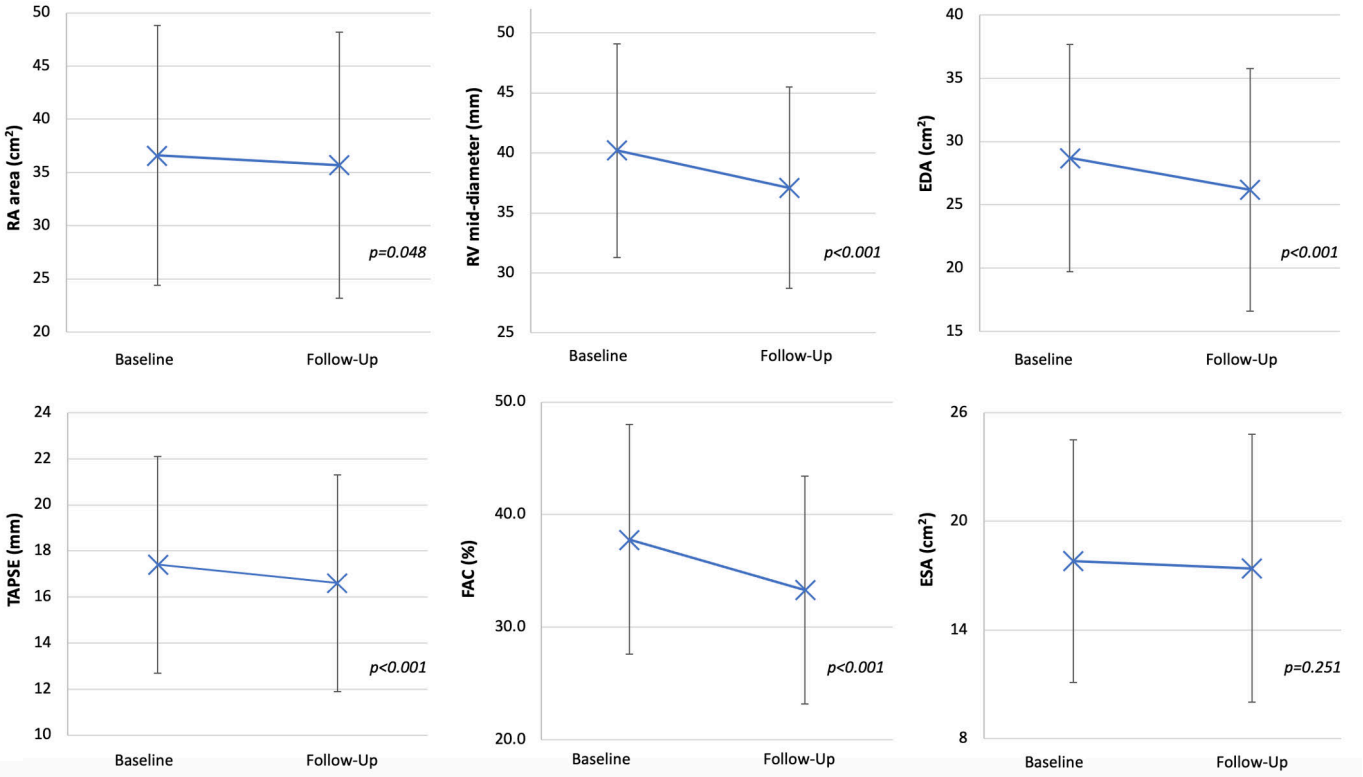


Figure 2. Detailed numeric changes of right heart parameters.



O057

Gut microbiota tryptophan and cholesterol pathways alterations in calcific aortic stenosis

Caroline Nguyen[1 2 3], Sarah Atighetchi[1], David Reineke[4], Bahtiyar Yilmaz[5], Harry Sokol[6], Cyril Ferro[1], Camille Dupuy[7], Lydie Nadal-Desbarats[7], Yara Banz[8], Daijiro Tomii[1], Masaaki Nakase[1], Matthias Siepe[4], Yvonne Döring[2 9], Thomas Pilgrim[1]

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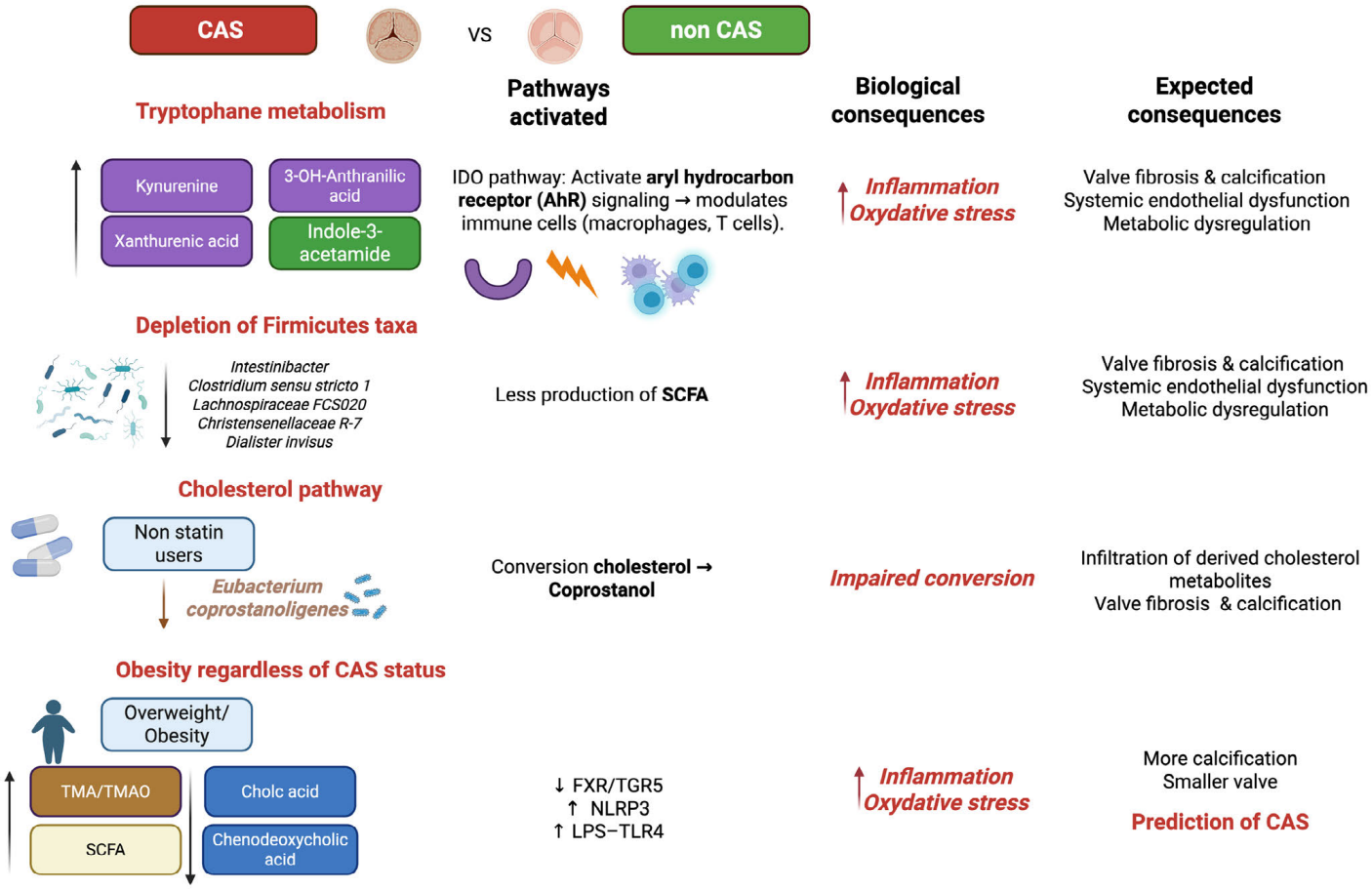
Introduction: Calcific aortic stenosis (CAS) is a progressive valvular disease characterized by lipid accumulation, inflammation, and osteogenic remodeling. The contribution of gut microbiota dysbiosis and microbial-derived metabolites – particularly tryptophan (TRP) metabolic pathways – remains incompletely understood, and cholesterol-related mechanisms remain unclear. The aim is to characterize gut microbiota composition and circulating microbial-derived metabolites associated with CAS.

Material & Methods: In a prospective cohort of 55 CAS patients and 55 non-CAS controls, gut microbiota composition was assessed by 16S rRNA sequencing and targeted serum metabolomics quantified TRP derivatives, short-chain fatty acids, bile acids, and TMA/TMAO-related metabolites. Comparisons were performed in the full cohort and after BMI matching. Effect sizes were estimated using Cliff's delta for metabolites and Cohen's f^2 for microbial taxa, with Benjamini–Hochberg correction for multiple testing.

Results: Baseline characteristics (age, sex, cardiovascular risk factors, medications, and coronary artery disease) were comparable between groups ($p > 0.05$). CAS patients displayed a distinct TRP metabolic profile with upregulation of inflammatory kynurenine-pathway metabolites. After BMI matching, CAS patients showed higher concentrations of xanthurenic acid ($p = 0.039$), indole-3-acetamide ($p = 0.045$), and 3-hydroxyanthranilic acid ($p = 0.028$), with consistent effect sizes (Cliff's $\delta \approx 0.23$–0.28). Microbiota analysis revealed depletion of Firmicutes-related taxa, including *Eubacterium coprostanoligenes*, a cholesterol-reducing bacterium converting cholesterol to coprostanol, suggesting impaired gut-mediated cholesterol metabolism. In multivariable logistic regression, BMI category remained independently associated with CAS ($\beta = 1.21$, $p = 0.024$). Male CAS patients showed higher concentrations of inflammatory kynurenine pathway metabolites, whereas female CAS patients exhibited relatively higher TMAO levels and indole metabolites, indicating sex differences in gut-derived metabolic activation.

Conclusion: CAS is associated with a coherent gut microbiota–metabolite signature marked by activation of inflammatory TRP catabolism and depletion of cholesterol-modulating bacteria. These alterations persist after BMI matching, supporting a gut–cholesterol–TRP–inflammation axis that may contribute to valvular calcification.

COI: No



O059

M-TEER and Anatomical Challenges: Analysis of Barlow and MAC Populations

Arslan Dilara[1], Gaspard Suc[2], Jules Mesnier[3], Fabien Praz[4], Rainer Zbinden[5], Caroline Nguyen[5]

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Introduction: Transcatheter mitral valve edge-to-edge repair (M-TEER) has emerged as a safe and effective therapeutic alternative to surgery for severe organic mitral regurgitation. However, outcomes of M-TEER in complex anatomies remain poorly defined. This study evaluate immediate and long-term procedural outcomes of M-TEER in patients with Barlow disease or MAC, compared to patients without these anatomical characteristics.

Material & Methods: Multicenter observational study of 192 consecutive patients undergoing M-TEER across five international centers. Barlow (n = 39) and MAC patients (n = 64) were compared with propensity score–matched controls. Multivariate Cox regression and Kaplan–Meier analyses were used to assess outcomes. The primary endpoint was long-term all-cause mortality. The secondary endpoints included residual mitral regurgitation, technical success, heart failure hospitalizations, and a composite of death, heart failure or reintervention.

Results: Technical success was high in the Barlow population. Despite a favorable comorbidity profile, Barlow patients exhibited significantly higher mortality (41.0% vs 17.9%, p = 0.047) with more frequent grade 1 residual mitral regurgitation (61.5% vs 33.3%, p = 0.023). MAC patients showed suboptimal valvular correction (significant residual mitral regurgitation (grade ≥2) persisted in 72% of MAC patients versus 36% of non-MAC patients (p = 0.001)) with a trend toward increased mortality (40% vs 22%, p = 0.084). Multivariate analysis identified Barlow disease (OR = 3.48, p = 0.0019) and MAC (OR = 4.04, p = 0.046) as independent predictors of mortality. Kaplan-Meier curves revealed distinct evolutionary patterns: delayed decompensation in Barlow (2-year survival: 57.4% vs 89.2%) and early excess mortality in MAC.

Conclusion: M-TEER in patients with Barlow disease or MAC is associated with poorer outcomes. In Barlow disease, this is not driven by technical failure but likely by associated cardiomyopathies, while in MAC systemic frailty appears to be a key determinant of prognosis. Isolated valvular correction alone does not normalize outcomes, highlighting the potential benefit of optimized medical therapy and earlier intervention.

COI: No

O060

Minimally Invasive Mitral Valve Repair vs. MitraClip in Octogenarians with Mitral Regurgitation

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Introduction: In octogenarians with severe mitral regurgitation, transcatheter edge-to-edge repair (MitraClip) and minimally invasive mitral valve repair (mini-MVR) are therapeutic options. Comparative outcome data in this high-risk population are limited.

Material & Methods: We conducted a retrospective cohort study of patients aged 80–89 years undergoing isolated mitral intervention at two Zurich cardiac surgery centers between 2009 and 2023. Baseline characteristics, operative metrics, perioperative complications, and echocardiographic outcomes were compared between MitraClip and mini-MVR. Categorical variables were analyzed using Chi-squared or Fisher's exact tests, and continuous variables with Wilcoxon rank-sum tests.

Results: A total of 105 patients were included (59 MitraClip, 46 mini-MVR; median age 82.8 years), no in-hospital deaths occurred in either group. Procedure duration was shorter in the MitraClip group (median 73 minutes vs. 167 minutes; p <0.001). Length of hospital stay was reduced after MitraClip (5 vs. 11 days; p <0.001), as was ICU stay (0 vs. 1 day; p <0.001) and intubation duration (3 vs. 7 hours; p <0.001). Mini-MVR was associated with higher complication rates, including neurological events such as delirium or neurological deficit without imaging correlate suggestive of stroke (28% vs. 8.5%; p = 0.008), new-onset atrial fibrillation (24% vs. 1.7%; p <0.001), pacemaker implantation (15% vs. 0%; p = 0.002), and temporary renal replacement therapy (13% vs. 1.7%; p = 0.042). Conversely, moderate and severe residual mitral regurgitation was more common after MitraClip (44.6% vs. 0%; p <0.001).

Conclusion: In octogenarians, MitraClip is associated with shorter procedure times, reduced hospital stay and fewer perioperative complications compared with minimally invasive MVR. However, this comes at the expense of inferior valve-specific outcomes, including higher residual regurgitation. Importantly, the in-hospital survival was 100% across both groups. Individual treatment decisions in elderly high-risk patients must therefore find a balance between procedural safety and long-term valve function.

COI: No

O061

Myocardial Protection Using a Novel Combined Mitral Valve Water-Probe Test: A Retrospective Two-Center Analysis

Stak Dushaj[1], Lukasz Jezierski[1], Rasha Boulos[1], Vasileios Ntinopoulos[1], Milan Milojevic[1], Héctor Rodríguez Cetina Biefer[1], Omer Dzemali[1]

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Introduction: Mitral valve surgery is among the most frequently performed cardiac surgical procedures worldwide. The optimization of postoperative recovery is partially contingent upon minimizing intraoperative myocardial injury, which can be achieved by reducing the ischemic burden imposed on the myocardium during the surgical intervention. This study investigates whether intraoperative mitral valve function testing, combined with antegrade and intraventricular cardioplegia, can mitigate myocardial damage.

Material & Methods: This retrospective, two-center study included patients who underwent isolated mitral valve surgery between 2016 and 2024. The primary endpoint was postoperative myocardial enzyme levels, while the secondary endpoint was the quality of mitral valve repair outcomes. Statistical analyses were performed using R, including grid-search and subset analyses.

Results: A total of 405 patients were included: 163 in the cardioplegia group (Group A) and 242 in the water group (Group B), with similar preoperative characteristics (Table 1). On day 1 post-op, CK (712.0 [543.0-981.0] vs. 887.5 [514.0-1487.0];p = 0.001) and troponin (374.3 [292.0-560.0] vs. 625.6 [401.0-1103.0]; p <0.001) were lower in Group A, while myoglobin (189.0 [149.0-302.0] vs. 228.5 [153.0-350.0]; p = 0.086)

showed a trend toward reduction (Table 2). CPB and aortic cross-clamp times were longer in Group B (p <0.001), indicating potential confounders. To control for this, procedures by the most experienced surgeons (n = 107; 38 in Group A, 69 in Group B) were analyzed, showing similar enzyme trends but persistent differences in CPB and ischemic times (Table 2). A sensitivity analysis of patients with comparable bypass and ischemic times (p = 0.14; n = 17 in Group A, 129 in Group B) confirmed lower troponin in Group A (660.5 [425.0-1057.0] vs. 631.0 [454.0-1056.5];p = 0.9). CK was higher (1210.0 [795.0-1591.0] vs. 957.0 [613.0-1315.0];p = 0.2), and myoglobin was significantly higher in Group A (303.0 [229.0-595.0] vs. 223.0 [153.0-297.0];p = 0.002; Table 2).

Conclusion: Cardioplegia-testing was associated with lower enzyme levels indicating better myocardial protection. However, confounding factors necessitate further investigations.

COI: No

Characteristic	Overall N = 405	Cardioplegia N = 163	Water N = 242
Gender - male	305 (75%)	124 (76%)	181 (181%)
Age	61 [53,68]	62 [54,69]	61 [53,68]
BMI	24.69 [22.1, 27.41]	24.62 [22.09, 27.16]	24.7 [22.34,27.63]
EuroSCORE	0.89 [0.62,1.33]	0.8 [0.59,1.14]	0.97 [0.67, 1.63]
Hypertension	184 (45%)	78 (48%)	106 (44%)
COPD	12 (3%)	1 (0.6%)	11 (4.5%)
EF at admission	55 [55, 62]	60 [55,65]	55 [50,60]

		Overall data			Best surgeons			Comparable CPB & ischemic times		
	Characteristics	Group A - Cardiolegia N = 163	Group B - Water N = 242	p-value	Group A - Cardiolegia N = 107	Group B - Water N = 38	p-value	Group A - Cardiolegia N = 17	Group B - Water N = 129	p-value
Intraoperative	Surgery duration	163.00 [142.00-191.00]	211.50 [180.00-245.00]	< 0.001	149.00 [131.00-170.00]	155.50 [139.00-182.00]	0.06	219.00 [205.00, 247.00]	216.00 [200.00, 238.00]	0.4
	CPB time	60.00 [51.00-84.00]	124.00 [98.00-154.00]	< 0.001	54.00 [46.00-60.00]	92.00 [79.00-108.00]	< 0.001	120.00 [108.00, 131.00]	129.00 [114.00, 147.00]	0.14
	Ischemic time	46.00 [36.00-66.00]	82.50 [64.00-102.00]	< 0.001	40.00 [35.00-47.00]	58.00 [46.00-66.00]	< 0.001	87.00 [83.00, 97.00]	88.00 [74.00, 98.00]	0.5
Postoperative	CRP day 1	34.8 [21.3-44.2]	50.5 [33.00-68.00],	< 0.001	34.40 [21.30, 43.00]	60.00 [43.00, 82.00]	<0.001	25.2 [13.7-35.9]	54.0 [33.00-68.00]	<0.001
	Creatinine day 1	72.0 [63.0-84.0]	77.0 [66.0-95.0]	0.008	69.00 [62.00, 78.00]	81.00 [67.00, 112.00]	0.002	93.0 [67.0-98.0]	75.0 [64.0-88.0]	0.2
	CK day1	712.0 [543.0-981.0]	887.5 [514.0-1487.0]	0.001	654.00 [479.00, 873.00]	612.50 [459.00, 850.00]	0.6	1210.0 [795.0-1591.0]	957.0 [613.0-1315.0	0.2
	CK-MB	28.4 [21.1-37.0]	31.0 [24.0-43.5]	0.051	28.00 [20.90, 36.00]	27.00 [23.00, 33.00]	>0.9	39.0 [30.0-55.4]	36.5 [28.0-56.0]	0.5
	Myoglobin day 1	189.0 [149.0-302.0]	228.5 [153.0-350.0]	0.086	168.00 [139.00, 229.00]	257.50 [163.00, 441.00]	<0.001	303.0 [229.0-595.0]	223.0 [153.0-297.0],	0.002
	LDH day 1	268.0 [229.0-312.0]	551.5 [370.0-688.5]	< 0.001	252.00 [223.00, 298.00]	286.00 [250.00, 326.50]	<0.001	352.0 [312.0-437.0]	552.0 [415.0-663.0]	<0.001
	Troponin day 1	374.3 [292.0-560.0]	625.6 [401.0-1103.0]	< 0.001	345.00 [275.00, 481.00]	227.50 [189.00, 369.00]	<0.001	660.5 [425.0-1057.0]	631.0 [454.0-1056.5]	> 0.9

RAPID FIRE ABSTRACTS: TAVI

O062

1-Year Outcomes after Conduction System Pacing after Transcatheter Aortic Valve Implantation

Teodor Serban[1], Stefan Stortecky[2], Philipp Krisai[1], Sven Knecht[1], Christoph Huber[3], Marco Roffi[3], Felix Mahfoud[1], Michael Kühne[1], Christian Sticherling[1], Patrick Badertscher[1]

[1]University Hospital of Basel, Basel, Switzerland, [2]Inselspital Bern, Bern, Switzerland, [3]University Hospital of Geneva, Geneva, Switzerland

Introduction: Studies comparing conduction system pacing (CSP) compared to conventional pacemaker implant after transcatheter aortic valve replacement (TAVI) are lacking.

Material & Methods: In this prospective, observational, multicentric cohort study, the outcomes of patients undergoing permanent PM implantation were investigated. Patients were enrolled from three centers across Switzerland between February 2011 and June 2022.

Results: Among 778 patients enrolled receiving PM implant within 30 days after TAVI (mean age 83±7 years, 40% female, self-expanding valves 54%), 85 (11%) underwent CSP.

Patients that received CSP had lower prevalence of coronary artery disease (36, 42% vs. 377, 54%, p = 0.04), received more often cerebral protection (20, 24% vs. 101, 15%, p = 0.04) and balloon-valvuloplasty (36, 42% vs 376, 54%, p = 0.04).

At one year after PM implant, a composite of all-cause death, a LVEF decline ≥ 10%, and worsening of NYHA class III/IV occurred less often in CSP (HR 0.61, 95% CI 0.43-0.88, p = 0.007) but was not significant after adjusting for age, sex and year of TAVI implant (aHR 0.72, 95% CI 0.49- 1.05, p = 0.09). A LVEF decline ≥10% occurred numerically less often, yet not statistically different in CSP compared to right ventricular pacing (3, 8% vs. 78, 18%, HR 0.45, 95% CI 0.15-1.35, p = 0.16).

Conclusion: In this prospective multicentric study, patients receiving CSP within 30 days after TAVI

had a lower occurrence of a combined endpoint of death, LVEF decline of ≥ 10% or a worsening in NYHA class III/IV at one year compared to conventional right ventricular pacing but not significantly after multivariable adjustment.

COI: No

CENTRAL ILLUSTRATION: 1-Year Outcomes after Conduction System Pacing after Transcatheter Aortic Valve Implantation

Patient Population

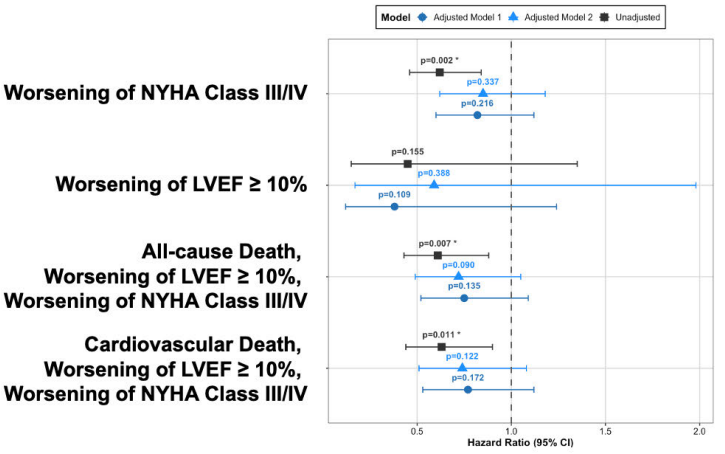
Three Swiss TAVI centers, between February 2011 and June 2022

Conventional RV Pacing (n = 693)	vs	Conduction-System Pacing (n = 85)

Patients with CSP had lower prevalence of coronary artery disease, received more often valvuloplasty

Patients with CSP were implanted on average within four days after TAVI versus only two days for patients with conventional right ventricular pacemakers

1-Year Outcomes



- In this multicentric, prospective, observational study, patients with CSP within 30-days after TAVI had better unadjusted all-cause mortality and cardiovascular mortality than patients with conventional right ventricular pacing at one year.
- The rates of left ventricular ejection fraction worsening were not significantly different between the groups but the worsening in NYHA class occurred less often in patients CSP.

O063

Early Rehospitalization after Transcatheter Aortic Valve Implantation

Polyxeni Alexiou[1], Federico Moccetti[1], Indra Bat-Ochir[1], Lorena Niederberger[1], Stephanie Brunner[1], Mathias Wolfrum[1], Stefan Toggweiler[1]

[1]*Heart Center, University Teaching and Research Hospital Lucerne, Lucerne, Switzerland, Luzern, Switzerland*

Introduction: Early rehospitalization after transcatheter aortic valve implantation (TAVI) is common. However, data regarding its prevalence, predictors and prognostic relevance are limited.

Material & Methods: We retrospectively analysed consecutive patients undergoing TAVI between 2012 and 2025. Early rehospitalization was defined as any admission within 30 days from the index hospitalization discharge. Predictors and outcomes were evaluated using uni- and multivariable regression models and Kaplan–Meier analyses.

Results: A total of 1,347 patients (44% women, mean age 81±6 years), were included. 131(9.7%) required early rehospitalization, most frequently due to infection/sepsis (22.9%), heart failure (HF, 19.8%) and delayed bradyarrhythmia requiring pacemaker implantation (9.9%). Independent predictors of rehospitalization for infection were chronic HF (OR 2.60, 95% CI 1.02–6.59; p = 0.045), chronic obstructive pulmonary disease (OR 3.18, 95% CI 1.27–7.96; p = 0.013), and procedural contrast volume (OR 1.25 per 25 ml increase, 95% CI 1.09–1.44; p = 0.002). Rehospitalization for HF was associated with chronic HF (OR 3.86, 95% CI 1.25–11.89; p = 0.019), ≥ mild paravalvular leak (OR 5.91, 95% CI 1.49–16.83; p = 0.009) and reduced post-procedural ejection fraction (OR 3.84, 95% CI 1.22–12.09; p = 0.022). Conduction abnormalities at discharge predicted rehospitalization for bradyarrhythmia requiring pacemaker implantation (OR 4.65, 95% CI 1.39–15.57; p = 0.013). Early (≤ 2 days) discharge was not associated with an increased risk of rehospitalization (336,27.7% vs 26,20.3%, p = 0.073). Early rehospitalization was associated with increased one-year all-cause mortality after multivariable adjustment for potential confounders (HR 3.03, 95% CI 1.90–4.86; p <0.001).

Conclusion: One in ten patients is readmitted within 30 days following TAVI. Modifiable predictors of rehospitalization were contrast volume, ≥mild paravalvular leaks and conduction disturbances at discharge. One-year mortality risk was three times higher in patients requiring rehospitalization, highlighting the need for closer post-discharge follow-up care.

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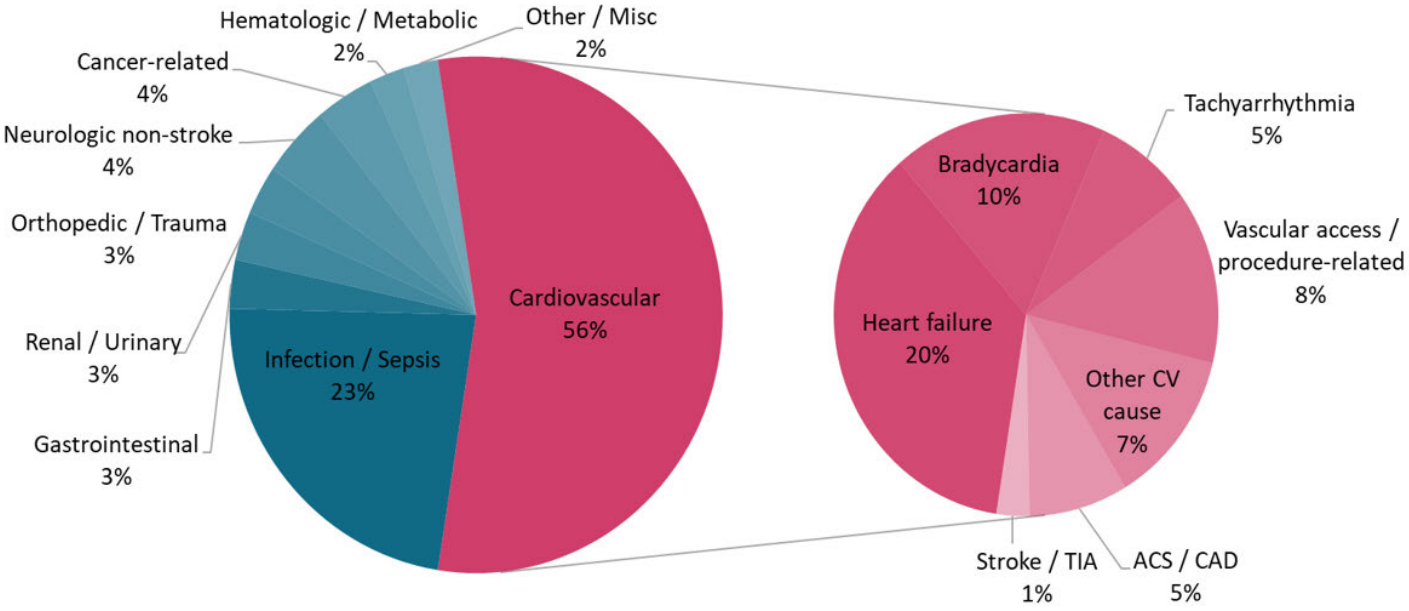

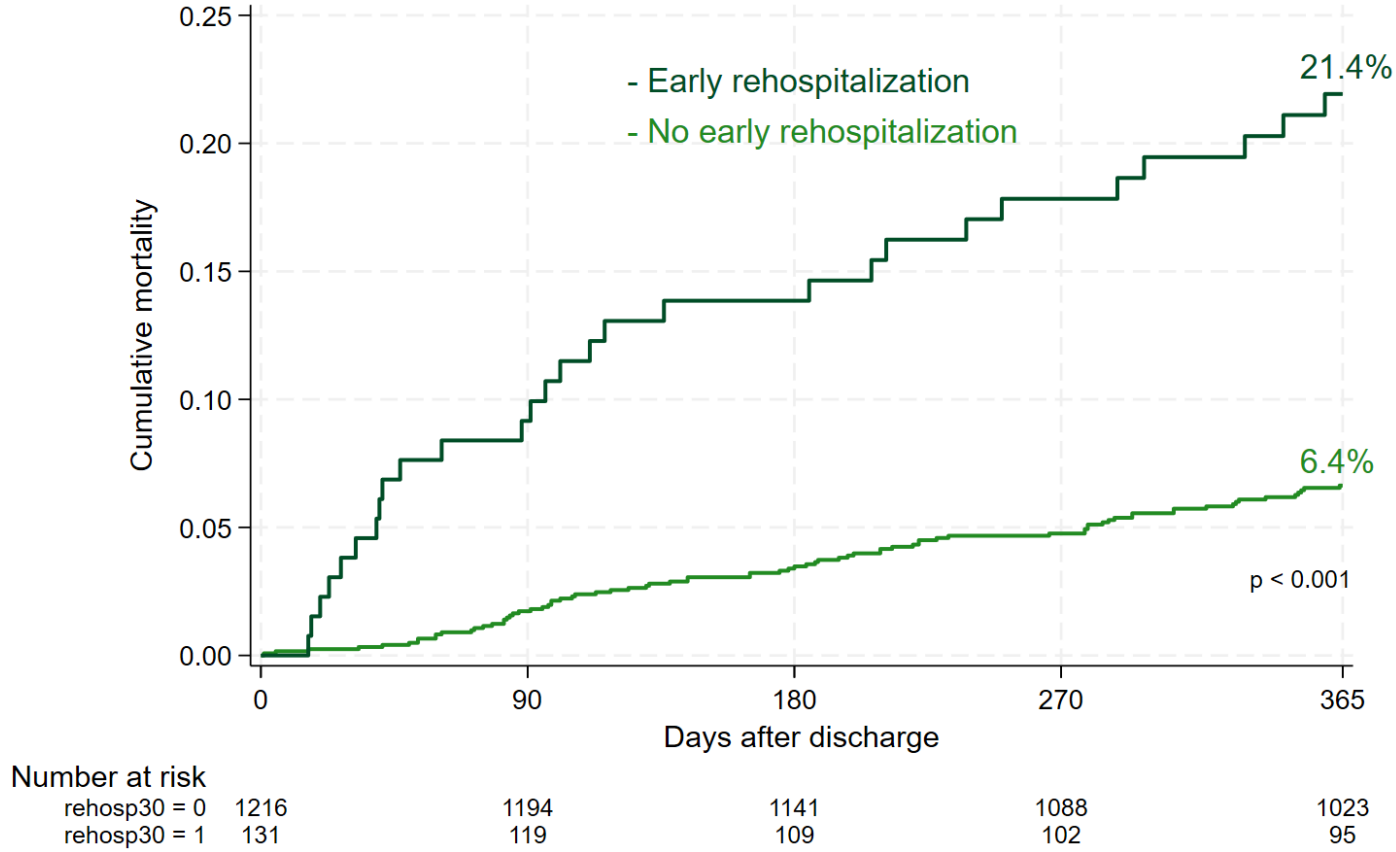

O064

Left bundle branch block after transcatheter aortic valve implantation: Comparison of two risk stratification strategies

Jonas Brügger[1,2], Teodor Serban[1,2], Thomas Nestelberger[1,2], Arianna Bettelini[1,2], Celine Moser[1,2], Thorald Stolte[1,2], Fabian Jordan[1,2], Sven Knecht[1,2], Reto Stump[1,2], Beat Schär[1,2], Nicolas Schärli[1,2], Michael Kühne[1,2], Christian Sticherling[1,2], Felix Mahfoud[1,2], Patrick Badertscher[1,2]

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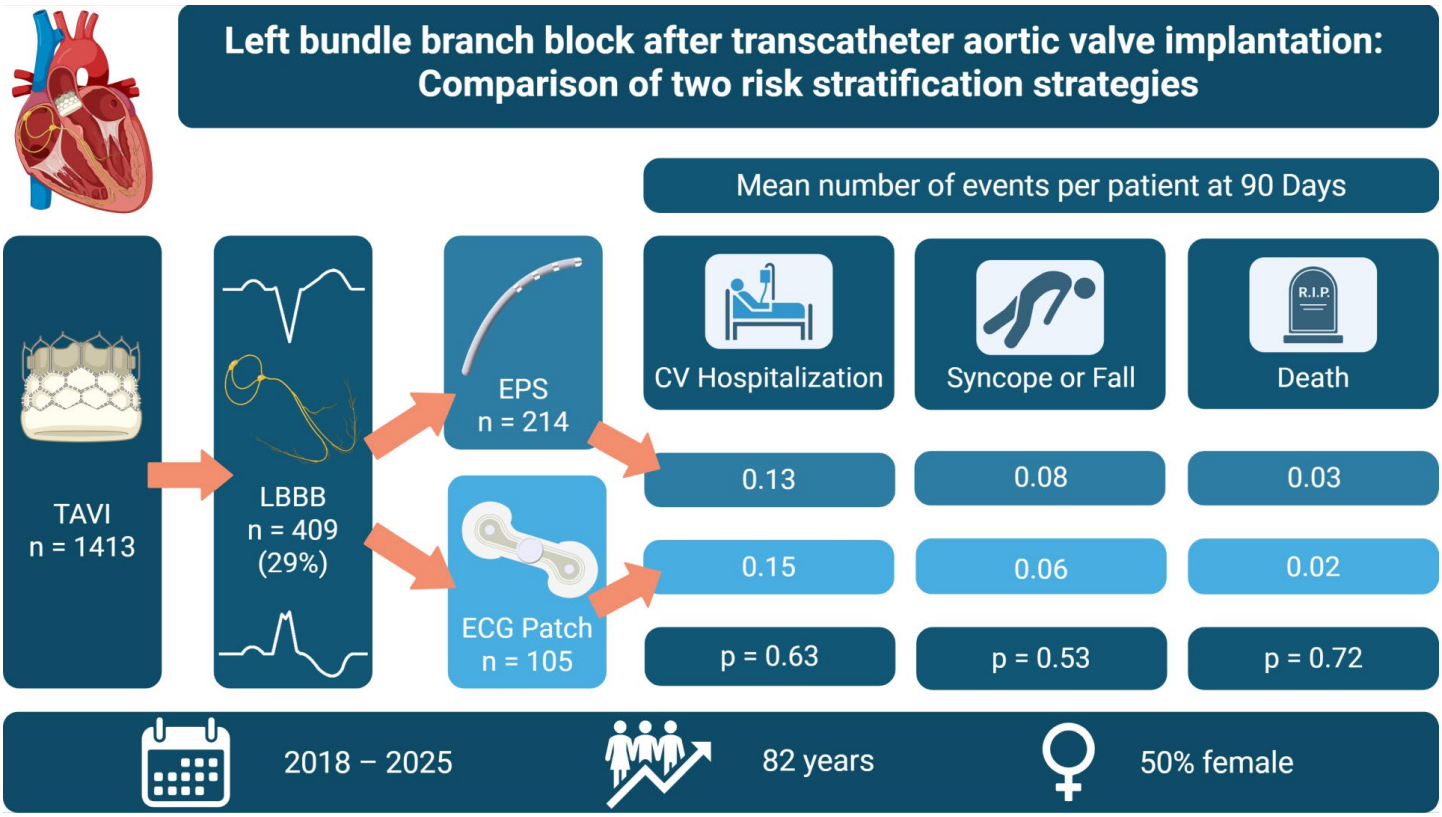
Introduction: Left bundle branch block (LBBB) remains the most frequent conduction disturbance after transcatheter aortic valve implantation (TAVI). Current guidelines recommend electrophysiological study (EPS) or prolonged ambulatory ECG monitoring for risk stratification, but these strategies have not been directly compared. The aim was to compare short-term clinical outcomes and permanent pacemaker (PM) implantation rates in patients with LBBB after TAVI managed with EPS-guided versus ambulatory ECG-patch-guided risk stratification.

Material & Methods: Adult patients undergoing TAVI at a tertiary care hospital in Switzerland with new-onset or pre-existing LBBB after TAVI were prospectively enrolled and underwent either EPS (phase 1) or 14-day ambulatory ECG-patch monitoring (phase 2). Patients with a pre-existing PM or post-TAVI PM indication were excluded. PM implantation was recommended for prolonged HV interval (>55 ms until 2022; ≥70 ms thereafter) in EPS or for documented HAVB or clinically relevant pauses in the ECG patch. The primary endpoint was a composite of syncope, fall, unplanned cardiovascular hospitalization, or all-cause death within 90 days after TAVI. The secondary endpoint was PM implantation attributable to the assigned risk stratification strategy.

Results: Among 409 patients with LBBB after TAVI (median age, 82 years; 50% women), 214 underwent EPS and 105 underwent ECG-patch monitoring. Within 90 days, 52 primary endpoint events occurred in the EPS group (mean, 0.24 events/patient) and 24 in the patch group (mean, 0.23 events/patient) (incidence rate ratio, 0.94 [95% CI, 0.53–1.67]; *P* = .83). PM implantation attributable to the risk stratification strategy occurred in 35 EPS patients (16%) versus 5 patch patients (5%) (*P* = .002).

Conclusion: In patients with LBBB after TAVI, ECG-patch-guided risk stratification was associated with similar short-term clinical outcomes compared with EPS-guided management but substantially fewer pacemaker implantations. Ambulatory ECG monitoring may therefore represent a safe and less invasive alternative for post-TAVI LBBB management.

COI: No



O065

Procedural and Long-Term Outcomes in Patients With Very High-Gradient Aortic Stenosis Undergoing TAVI

Federica Gramegna[1,2], Lukas Weber[1,3], Julien Ternacle[1], Johann Cattan[1], Pierre Jeantet[1], Melchior Jonveaux[1], Lionel Leroux[1], Thomas Modine[1], Guillaume Bonnet[1]

[1]Bordeaux University Hospital, Pessac, France, [2]San Raffaele Hospital, Milano, Italy, [3]HOCH – Kantonsspital St. Gallen, St. Gallen, Switzerland

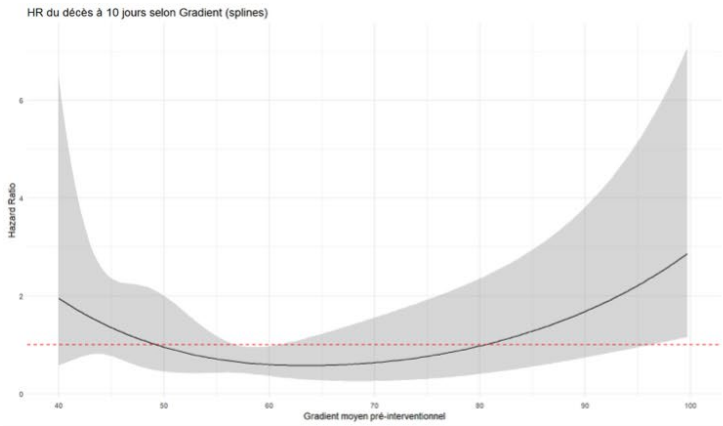
Introduction: High-gradient severe aortic stenosis (AS) is usually associated with preserved ventricular function and low comorbidity burden. However, the impact of extreme gradients (≥80 mmHg) on outcomes after transcatheter aortic valve implantation (TAVI) is poorly defined.

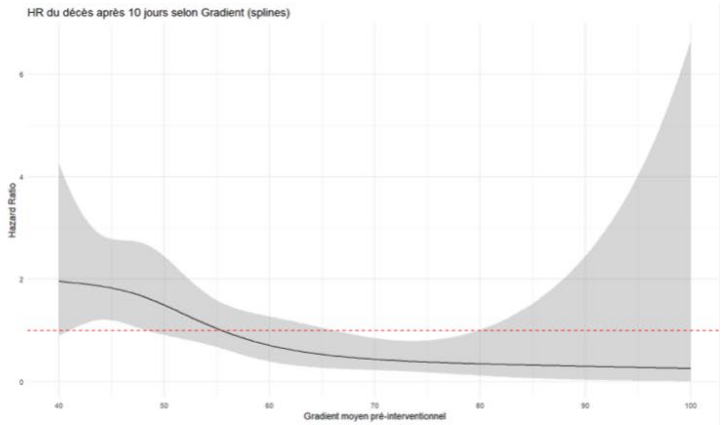
Material & Methods: We retrospectively analyzed 1'086 patients undergoing transfemoral TAVI in a single center between january 2023 and april 2025, all with a baseline mean transvalvular gradient ≥40 mmHg. Patients were stratified into three groups: 40–59 mmHg (n = 717), 60–79 mmHg (n = 278), and ≥80 mmHg (n = 91). Baseline features, imaging, procedural characteristics, and in-hospital outcomes were compared across strata. Survival was assessed with Kaplan–Meier analysis and a 10-day landmark.

Results: Patients with very high gradients were younger and less comorbid, with higher LVEF and smaller valve area. Procedures were longer, required more post-dilatation, and carried increased risks of tamponade, coronary obstruction, and major bleeding. In-hospital mortality was 4.4% in the ≥80 mmHg group, with most deaths occurring intraprocedural. 10 days landmark analysis confirmed clustering of early events in this subgroup (p = 0.036). Long-term survival differed significantly across gradient categories (p = 0.018) with the best survival in the 60–79 mmHg group and similar survival in the 40–59 mmHg and ≥80 mmHg group.

Conclusion: Extreme transvalvular gradients identify a distinct phenotype of AS patients undergoing TAVI. Despite preserved ventricular function and favourable baseline risk, these patients experienced greater procedural complexity and excess early mortality. Recognizing very high gradient as a marker of procedural vulnerability may inform risk stratification, procedural planning, and timing of intervention.

COI: No





O066

Short- and long-term outcomes after transcatheter implantation of large self-expanding valves

Polyxeni Alexiou[1], Federico Moccetti[1], Stephanie Brunner[1], Lorena Niederberger[1], Mathias Wolfrum[1], Stefan Toggweiler[1]

[1]Heart Center, University Teaching and Research Hospital Lucerne, Lucerne, Switzerland, Luzern, Switzerland

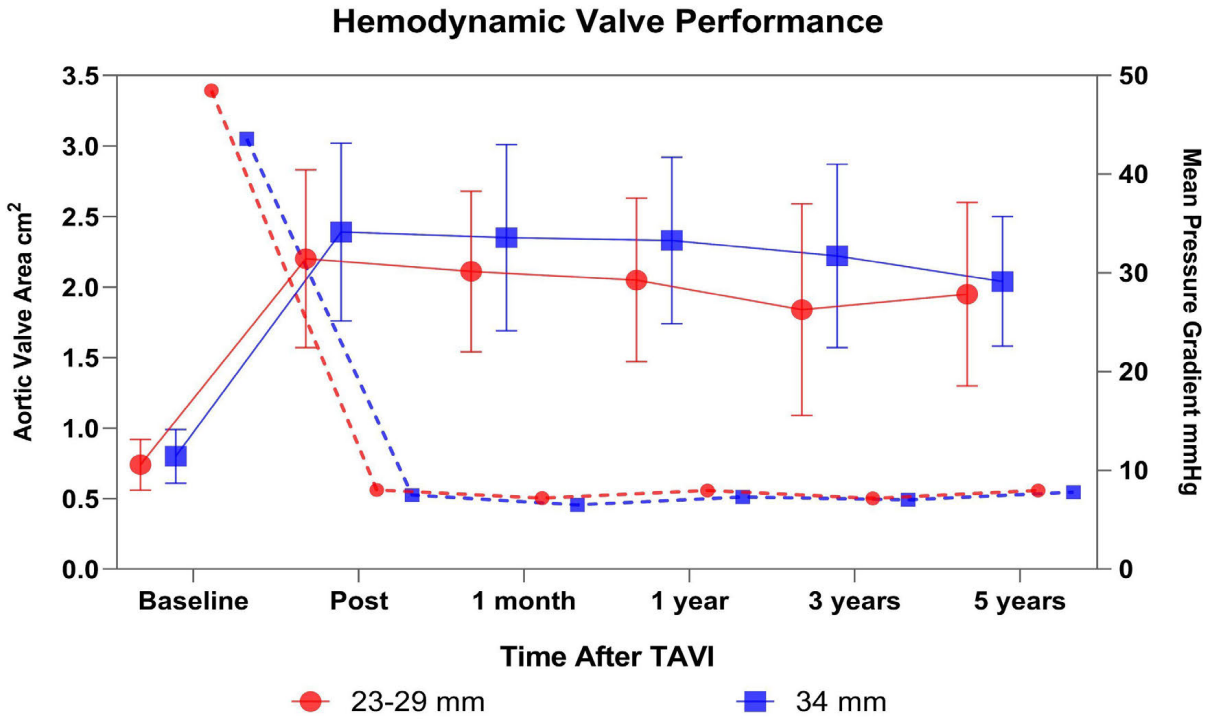
Introduction: Patients with large anatomies are more challenging to treat with transcatheter aortic valve implantation (TAVI). Evidence on long-term outcomes comparing different valve sizes is limited.

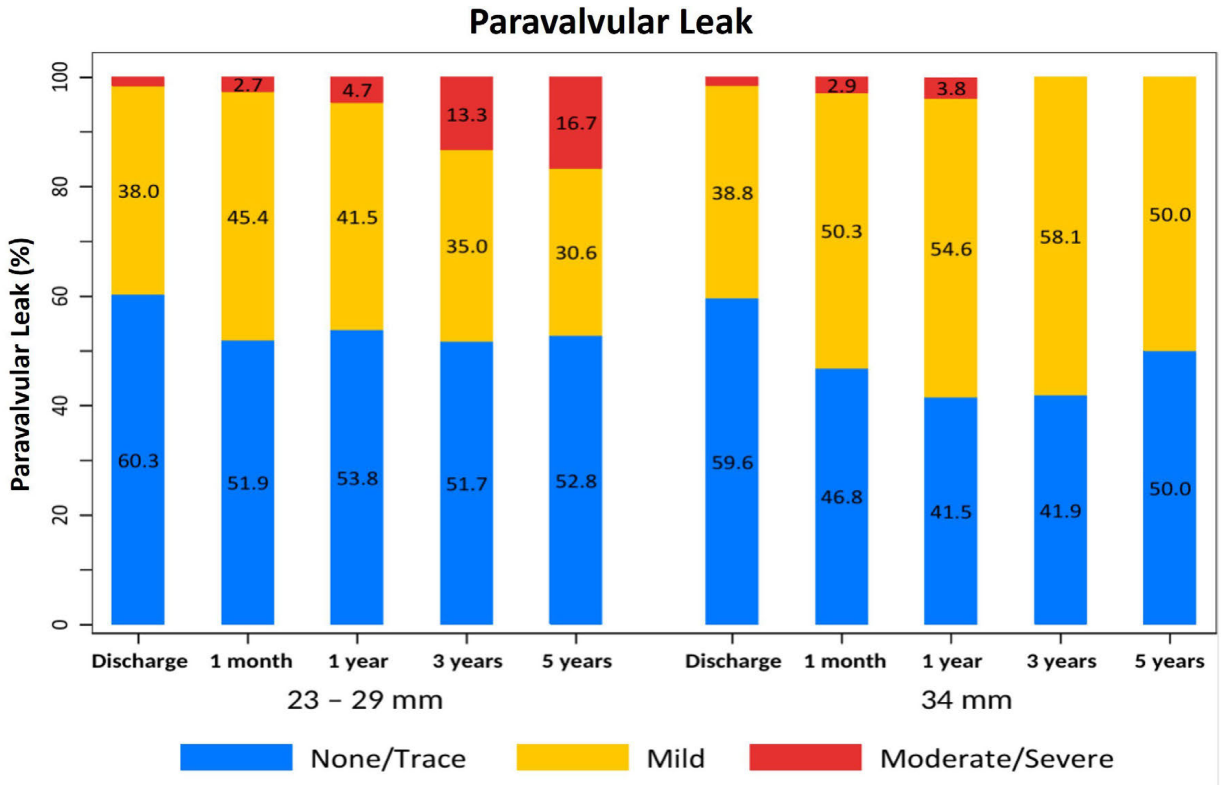
Material & Methods: Between 2015 and November 2025, 488 patients underwent transfemoral TAVI with different generations of Evolut valves for the treatment of native aortic stenosis. Patients treated with 34-mm valves were compared with those receiving 23–29-mm valves and follow-up was performed for up to six years.

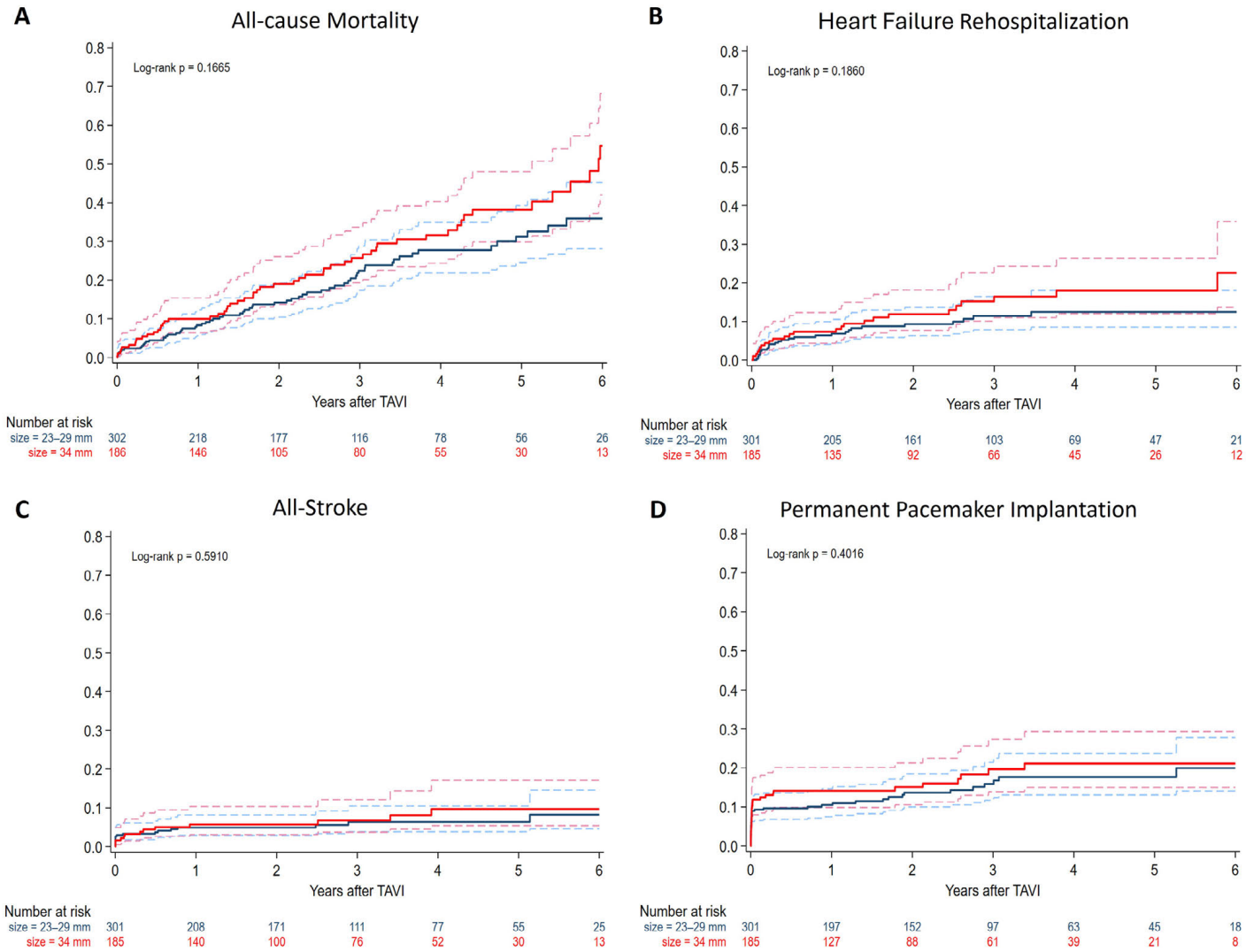
Results: Among 488 patients (30% females, mean age 81 ± 7 years), 182 (39%) received a 34-mm valve and 285 (61%) regular size 23-29-mm valves. Technical success was similar in both groups (p = 0.3) while device success at 30 days was achieved in 214 (71%) in the 23-29-mm, compared to 154 (83%) in the 34-mm group (p = 0.003). At 1 year, patients receiving 34-mm valves had a significantly larger aortic valve area compared with 23–29-mm valves (2.33 vs 2.05cm², p = 0.009). 1-year mean pressure gradient (p = 0.22) and paravalvular leak severity (p = 0.20) were comparable between groups. Pacemaker implantation within 30 days occurred in 11.8% vs 9.3% of patients in the 23-29- and in the 34-mm valve group, respectively (p = 0.40). All-cause mortality at 1 year (8.0% vs 9.5%) and 5 years (31.0% vs 37.9%) in the 23–29- and 34-mm-valve group, respectively, did not differ significantly (p = 0.18). Heart failure hospitalization occurred in 6.8% vs 7.4% at 1 year and 12.5% vs 18.0% at 5 years (p = 0.19). Stroke rates at 1 year were 4.9% vs 5.7%, increasing to 6.4% vs 9.7% at 5 years (p = 0.59).

Conclusion: Outcomes after TAVI with large self-expanding valves were comparable to regular sizes, demonstrating favourable procedural success, safety, and short- and long-term clinical endpoints.

COI: No







O067

Prevalence and influence of cardiovascular kidney metabolic syndrome on outcomes after transcatheter aortic valve implantation

Felix Götzinger[1], Thorald Stolte[1], Jakob Reichl[1], Anke von Peter[1], Michael Kunz[1], Mirjam Wild[2], Max Wagener[1], Christoph Kaiser[1], Christian Müller[1], Dirk Westermann[2], Thomas Nestelberger[1], Felix Mahfoud[1]

[1]Department of Cardiology, University Heart Center Basel, University Hospital Basel, 4031 Basel, Switzerland, Cardiovascular Research Institute Basel, Basel, Switzerland, [2]University Heart Center Freiburg/Bad Krozingen, University of Freiburg, Germany, Bad Krozingen, Germany

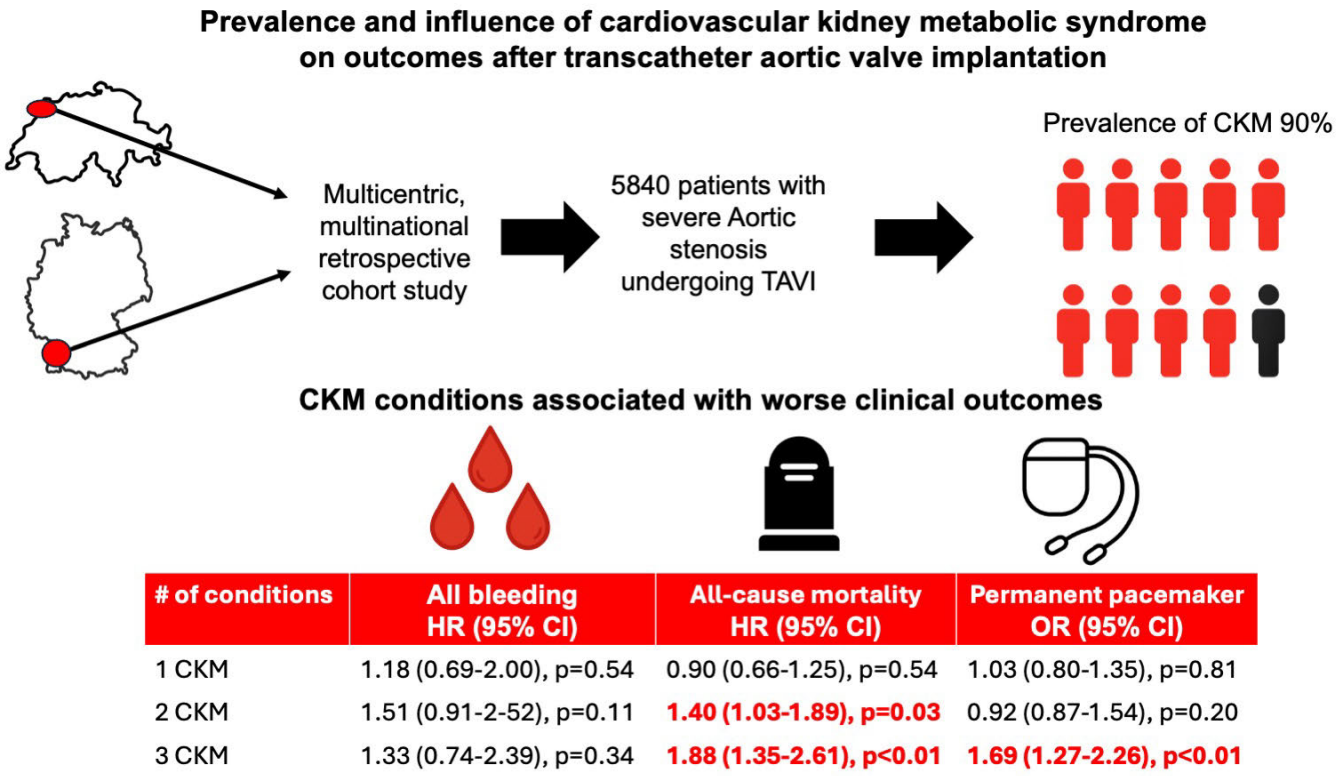
Introduction: The cardiovascular-kidney-metabolic syndrome (CKM) is a multisystem disorder increasingly recognized for its association with adverse outcomes. The incidence and prognostic relevance of CKM in patients undergoing transcatheter aortic valve implantation (TAVI) has not been investigated.

Material & Methods: A multicentric, multinational, retrospective analysis of patients undergoing TAVI from 2011 to 2024 was performed. CKM syndrome was diagnosed and quantified using a simplified definition based on the presence of atherosclerotic cardiovascular diseases (ASCVD), chronic kidney disease (CKD), and diabetes mellitus (DM). The prevalence of CKM and its association with procedural success, bleeding events, permanent pacemaker implantation and 1-year mortality was evaluated.

Results: Among 5,834 TAVI patients, 5,280 (90%) fulfilled criteria for CKM. The overall prevalence remained stable. Procedural success was highest in patients with no CKM conditions, followed by patients with 1 and 2 CKM conditions. Patients with 3 CKM conditions had the worst procedural success (p = 0.002). Patients with 2 and 3 CKM conditions had higher probability of death within 1 year as compared to patients with no CKM conditions and 1 CKM condition (HR for 2 CKM conditions 1.40, p = 0.030, HR for 3 CKM conditions 1.88, p <0.001). Patients with 3 CKM conditions had higher rates of permanent pacemaker implantation (OR 1.69, p <0.001), but not of major bleeding (HR 1.24, p = 0.328).

Conclusion: CKM is highly prevalent in patients undergoing TAVI. Having more CKM conditions appears to be associated with increased rates of adverse events.

COI: I have received speaker honoraria from Astra Zeneca, have been Supported by the German Foundation for Heart Research and am currently supported by the University of Basel Junior Excellent Research Grant.



# of conditions	All bleeding HR (95% CI)	All-cause mortality HR (95% CI)	Permanent pacemaker OR (95% CI)
1 CKM	1.18 (0.69-2.00), p=0.54	0.90 (0.66-1.25), p=0.54	1.03 (0.80-1.35), p=0.81
2 CKM	1.51 (0.91-2-52), p=0.11	**1.40 (1.03-1.89), p=0.03**	0.92 (0.87-1.54), p=0.20
3 CKM	1.33 (0.74-2.39), p=0.34	**1.88 (1.35-2.61), p<0.01**	**1.69 (1.27-2.26), p<0.01**

RAPID FIRE CASE REPORTS

O068

Electromagnetic Interference during Epicardial Ablation in a Patient with an Extravascular Implantable Cardioverter Defibrillator and Arrhythmogenic Right Ventricular Cardiomyopathy: a Case Report

Matthieu Proust[1 2], Feifan Ouyang[3], Alexander Breitenstein[1], Firat Duru[1 4], Ardan Saguner[4 5]

[1]Department of Cardiology, University Hospital Zurich, Zurich, Switzerland, [2]Sorbonne Université, Assistance Publique – Hôpitaux de Paris (AP-HP), Hôpital La Pitié-Salpêtrière, Département de Cardiologie, Paris, France, [3]Department of Cardiology, Inselspital Bern, Bern, Switzerland, [4]Center for Translational and Experimental Cardiology (CTEC), Schlieren, Switzerland, Schlieren, Switzerland, [5]SwissDNALysis, Dübendorf, Switzerland, Dübendorf, Switzerland

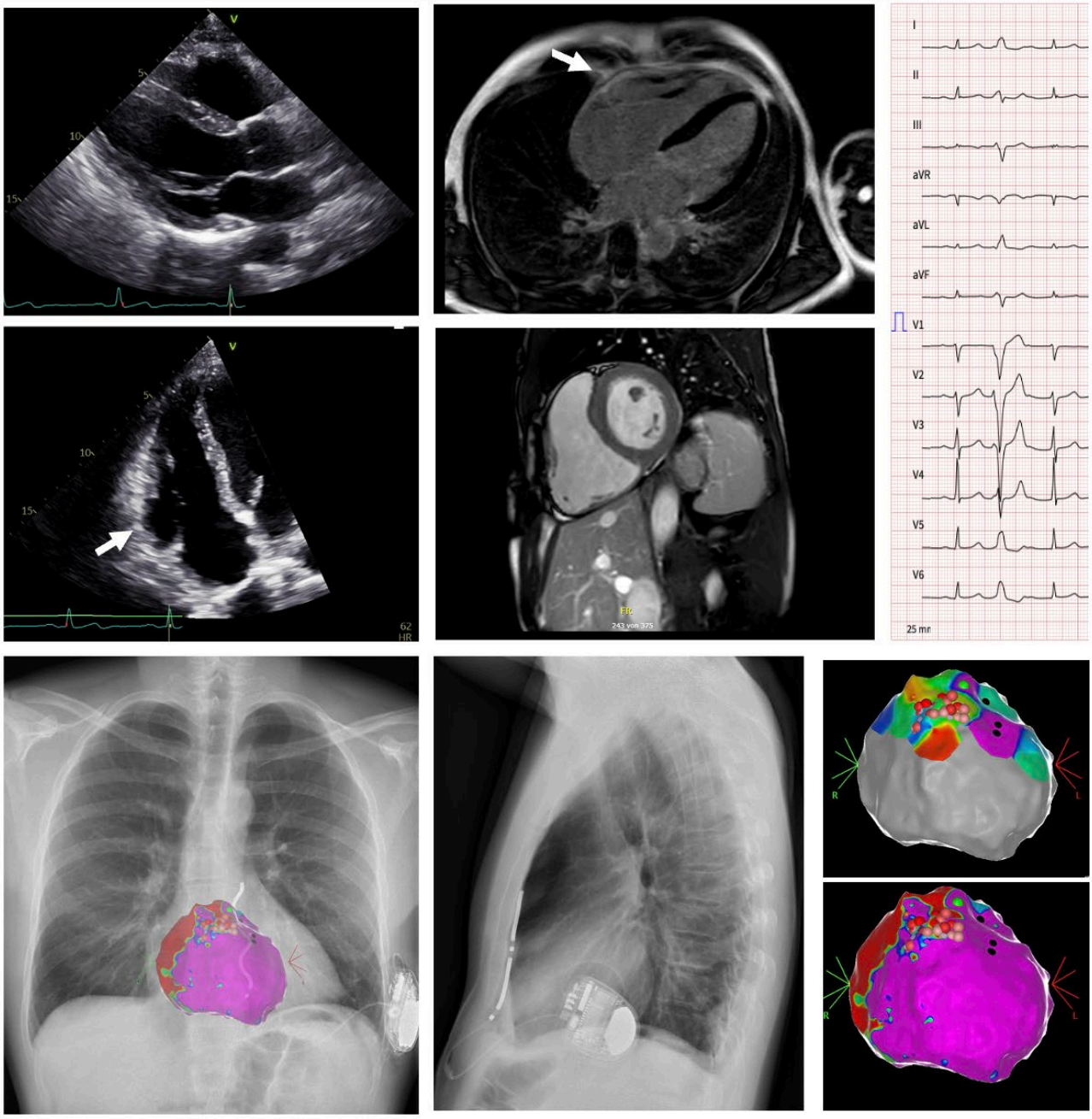
Introduction: Arrhythmogenic cardiomyopathy (ACM) is an inherited disease characterized by fibrofatty replacement, most often involving the right ventricle but potentially affecting the left or both ventricles. It predisposes to severe ventricular arrhythmia (VA). Differentiating ACM from athlete's heart remains difficult, particularly in gene-elusive patients. Implantable cardioverter defibrillators (ICD) and catheter ablation are key therapeutic options. Epicardial ablation may be required for VA circuits inaccessible in an endocardial approach, but data on its feasibility in patients with extravascular ICD (EV-ICD) featuring a retrosternal lead is scarce.

Material & Methods: Results: A 42-year-old male athlete was admitted for syncope due to sustained ventricular tachycardia (VT) during exertion. Cardiac magnetic resonance imaging (CMR) showed a dilated right ventricle (RV) with wall akinesia and fibrosis involving the RV outflow tract (RVOT). Genetic testing was negative. The diagnosis of borderline gene-elusive ACM was made. An EV-ICD was implanted for secondary prevention. Recurrent sustained VT prompted endocardial and epicardial ablation, which was feasible and successful, but radiofrequency energy application was hampered close to the retrosternal lead due to electromagnetic interference.

Conclusion: This case highlights the diagnostic and therapeutic challenges of ACM in athletes. Epicardial ablation is feasible even with an EV-ICD, although electromagnetic interference may complicate ablation near the lead. High-level endurance exercise can induce ACM-like remodeling, creating phenotypic overlap with gene-elusive ACM. Early recognition, tailored ablation strategies, and appropriate device therapy are essential for the management of these patients.

COI: No

Graphical abstract: Electromagnetic interference during epicardial ablation in a patient with an extravascular implantable cardioverter defibrillator and arrhythmogenic right ventricular cardiomyopathy.



- **Epicardial ablation in patients with EV-ICD is feasible**, although the lead is positioned retrosternally.
- Close to the lead, ablation can be hampered due to **electromagnetic interference** between the radiofrequency catheter and ICD lead.
- **Extreme endurance athleticism** can cause a distinct cause form of arrhythmogenic cardiomyopathy affecting the RVOT, showing overlap with ARVC, even in gene elusive patients.

EV-ICD: extravascular implantable cardioverter defibrillator; **ARVC:** arrhythmogenic right ventricular cardiomyopathy; **RVOT:** right ventricular outflow tract.

O069

A silent instigator: a common but largely unknown cause of ST depression

Cristina Pop[1], Peter Ammann[1], Roman Brenner[1], Sebastian Seidl[1], Florian Franzeck[1]

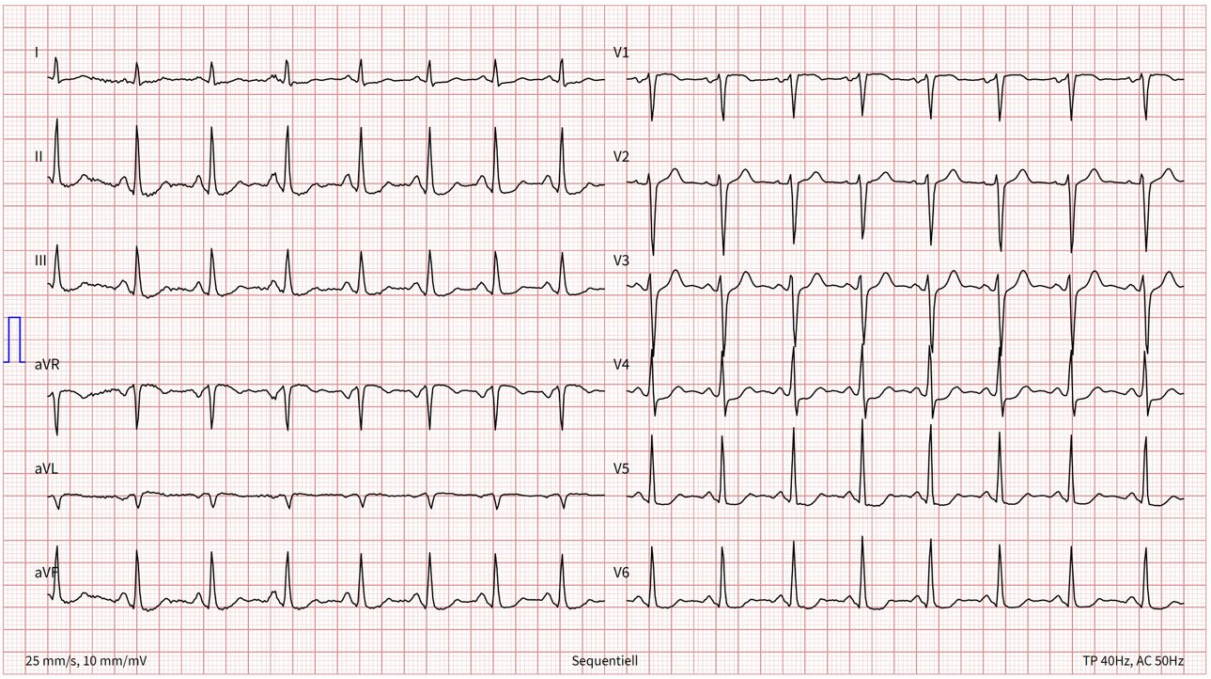
[1]*Klinik für Kardiologie, Kantonsspital St. Gallen, HOCH Health Ostschweiz, St. Gallen, Switzerland*

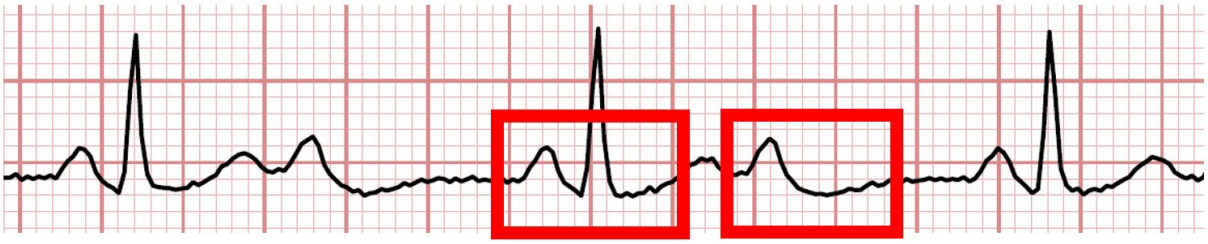
Introduction: ST segment depression is a significant electrocardiographic finding commonly associated with myocardial ischemia. However, the differential diagnosis is broad, including bundle branch block, ventricular hypertrophy, Takotsubo syndrome, digoxin effect, preexcitation, electrolyte disturbances and the recently described Bundgaard syndrome (familial ST depression syndrome). In this report, we describe a typical finding of a common yet under-recognized cause of ST depression.

Material & Methods: A 37-year-old asymptomatic male was referred for further evaluation as significant ST depression was noted during a routine medical assessment before starting a train driver training program. Medical history revealed that these repolarisation changes were already present at a military screening examination at age 17 and remained unchanged since then. Echocardiographic evaluation showed normal findings and no dynamic repolarisation changes were observed during a bicycle stress test. Due to the strict medical requirements for train driver eligibility, additional diagnostic imaging including coronary CT and cardiac MRI was performed and yielded normal results. Notably, the ST depression was most pronounced in leads with high P-wave amplitude and showed a strict discordance with the P-wave. To further investigate this pattern, adenosine testing was performed to induce temporary AV block. During 2:1 AV block a marked atrial repolarization wave was identified as the cause of the apparent ST depression.

Results: Conclusion: The existance of an atrial repolarization wave T(a) was first described in 1925. The T(a) wave is typically of low amplitude (0.1 -0.2 mV) and obscured by superimposition with the QRS. Importantly, the polarity of the P-wave is always opposite to that of the T(a) wave unlike the relationship between the P-wave and T-wave. It does not constitute a pathology and should not be misinterpreted as a sign of myocardial disease. Additionally, in cases of inferior ectopic atrial rhythm with a negative P-wave, also ST elevation may occasionally be observed.

COI: No





O071

Pediatric Giant Left Atrium with Secondary Mitral Regurgitation: Case Report and Surgical Insights

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Introduction: Giant left atrium is a rare cardiac pathology, particularly in the pediatric population. It is most commonly associated with rheumatic or degenerative valvular disease and rarely congenital, with few pediatric cases reported. Patients may present with arrhythmias, thromboembolic events, or heart failure. Progressive left atrial enlargement can cause secondary mitral regurgitation despite structurally normal mitral valve leaflets. Given its rarity, there is no established consensus on optimal treatment in children.

Material & Methods: We present the case of a 13-year-old girl with no prior medical history who underwent diagnostic evaluation for palpitations, exertional tachycardia, dizziness, and a syncopal episode. No sustained arrhythmias were documented, although an arrhythmic etiology of syncope was suspected. MRI and echocardiographic assessment demonstrated a giant left atrium (approximately 78 × 103 mm) with dextrorotation and secondary atrial mitral regurgitation, without evidence of rheumatic or intrinsic mitral valve disease. Surgical management consisted of left atrial reduction plasty achieved by plication of the aneurysmal segment using two running polypropylene sutures reinforced with bovine pericardium on either side of the coronary sinus, along with left atrial appendage occlusion and mitral valve ring annuloplasty. Atrial tissue samples were obtained for histopathological analysis.

Results: Surgery resulted in effective reduction of left atrial size and restoration of mitral valve competence without obstruction. Histological examination revealed endocardial fibrosis with cardiomyocyte hypertrophy and atrophy, interstitial fibrosis, and no evidence of malignancy. There were no findings suggestive of storage disease, granulomas, or inflammatory infiltrates. The postoperative course was uneventful, with sinus rhythm and preserved ventricular function. The patient was discharged home in good clinical condition.

Conclusion: In the absence of established treatment guidelines, left atrial reduction plasty combined with left atrial appendage occlusion and mitral valve annuloplasty proved to be a safe and effective surgical strategy for giant left atrium with secondary mitral regurgitation in our pediatric patient.

COI: No

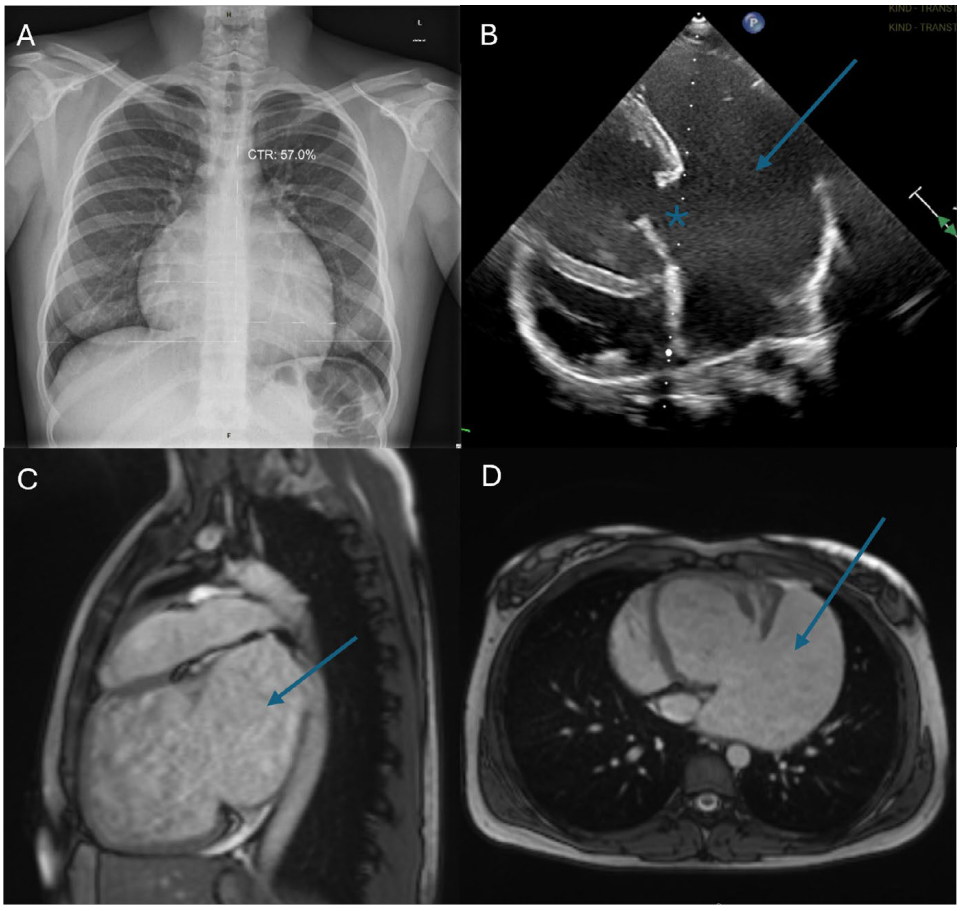

Figure 1 Preoperative diagnostic: (A) chest x-ray p.a. view, (B) transthoracal echocardiographic apical four-chamber view with arrow to left atrium and asterisk at the mitral valve, (C) MRI sagittal view with arrow to left atrium, (D) MRI axial view with arrow to left atrium.

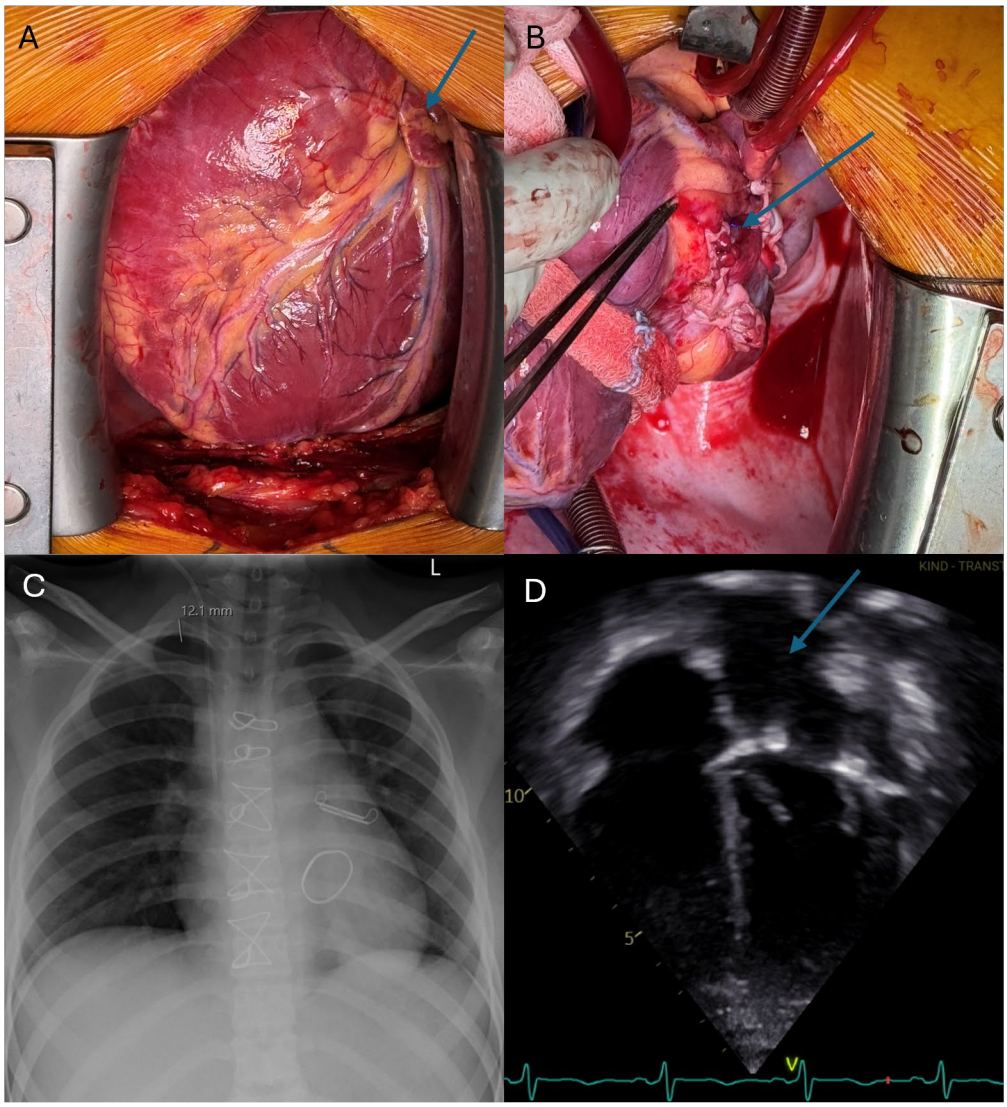

Figure 2 Intra- and postoperative images: (A) intraoperative view prior to surgical correction with dextrorotation of the heart and arrow towards left atrium, (B) intraoperative view after left atrial reduction plasty with arrow to plicated left atrium, (C) postoperative chest x-ray p.a. view, (D) transthoracal echocardiographic four-chamber view with arrow to the left atrium.

O072

When cardiac arrest unmasks complete adult aortic coarctation: a case report

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Introduction: Aortic coarctation is a congenital narrowing of the aorta that is typically diagnosed in childhood. Survival into adulthood without repair is uncommon and is associated with extensive collateral circulation, systemic hypertension, and premature coronary artery disease. Adult diagnosis is rare in developed countries due to routine screening, and coarctation is seldom discovered during acute cardiovascular emergencies

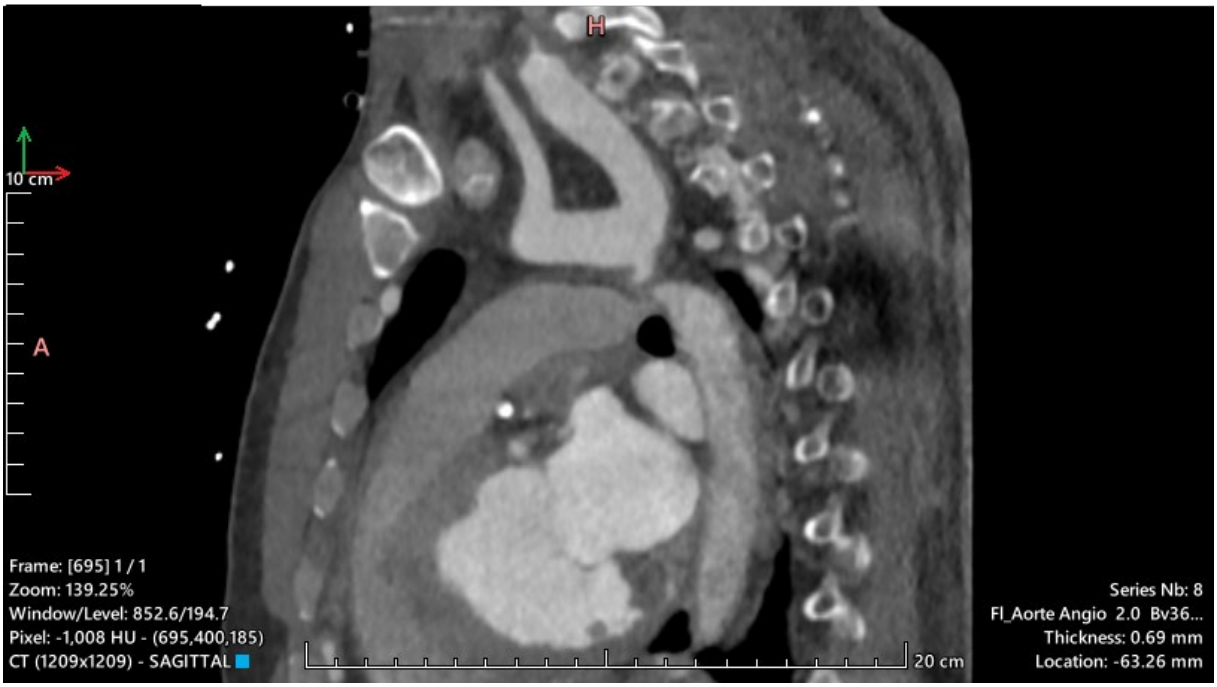
Material & Methods: A 51-year-old man with longstanding hypertension, dyslipidemia, and severe multivessel coronary artery disease suffered out-of-hospital cardiac arrest with an initial rhythm of ventricular fibrillation. After return of spontaneous circulation, he was transferred for suspected anterior STEMI.

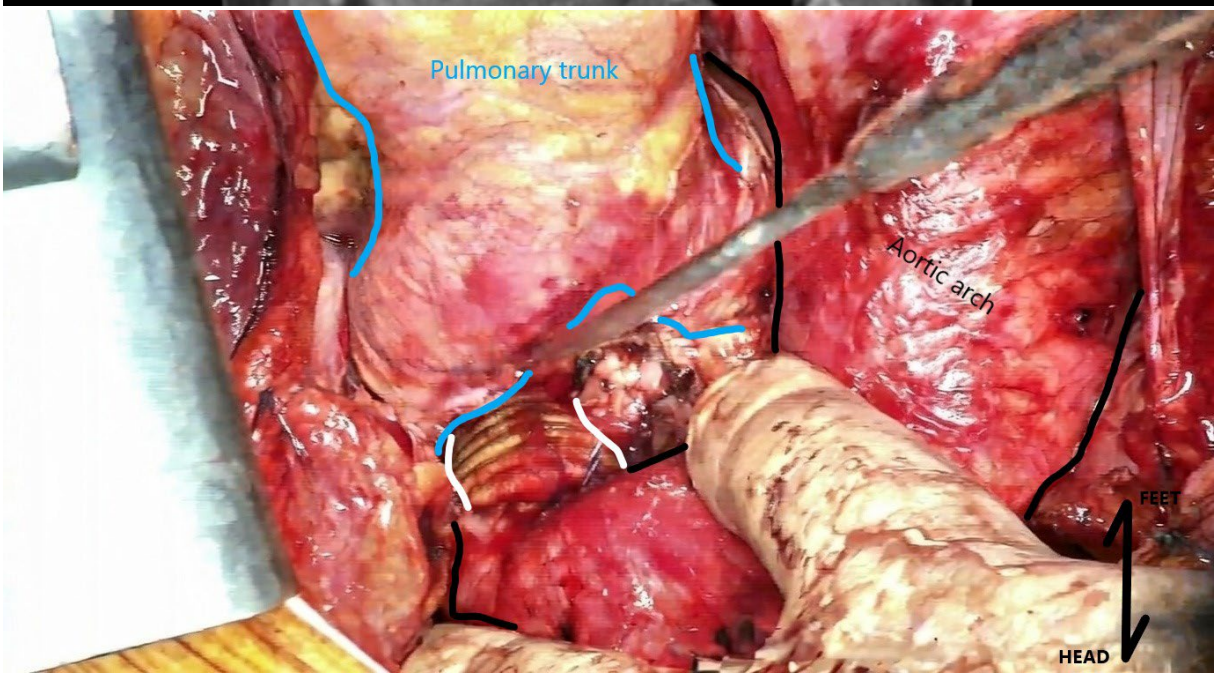
ECG showed anterior ST-segment elevation and echocardiography revealed severe left ventricular dysfunction (LVEF ~25%) with apical akinesia. Emergency coronary angiography demonstrated chronic three-vessel disease without an acute culprit lesion. Refractory cardiogenic shock prompted planned Impella implantation, but transfemoral access failed due to unexpected aortic obstruction. CT angiography revealed complete congenital aortic coarctation distal to the left subclavian artery with extensive collateral circulation (Image 1).

Results: After multidisciplinary discussion, urgent surgery was performed via median sternotomy. The coarctation was resected without cardiopulmonary bypass and reconstructed with an 18-mm interposition graft (Image 2). Coronary artery bypass grafting and apical aneurysm repair were completed in the same session under cardiopulmonary bypass. The patient was successfully weaned from bypass, extubated 8 hours postoperatively, and discharged from intensive care without neurological deficits or major cardiovascular complications.

Conclusion: This case highlights the rare occurrence of undiagnosed adult coarctation presenting during cardiac arrest, which limited percutaneous mechanical support. Longstanding hypertension and premature coronary disease were likely consequences of chronic proximal pressure overload. Combined single-stage repair via sternotomy is feasible and may be lifesaving when percutaneous support is not possible.

COI: No





O073

Emergency total aortic arch repair for acute aortic arch rupture using a minimally invasive technique

Lilly Ilcheva Burger[1], Petar Risteski[1], Igor Tudorache[1], Milan Milojevic[1], Héctor Rodríguez Cetina Biefer[1 2], Omer Dzemali[1 2]

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Introduction: Aortic arch pathology remains a major surgical challenge. Over the last decade, total aortic arch replacement with the frozen elephant trunk (FET) technique has become an established option for both aneurysmal and dissecting arch disease.We report an emergency minimally invasive ascending aortic surgery combined with FET implantation in a patient with a ruptured aortic arch.

Material & Methods: Results: A 78-year-old man presented with a 2-day history of severe chest and interscapular pain. His medical history included idiopathic pulmonary fibrosis treated with nintedanib, cerebrovascular occlusive disease with stenting of the common and internal carotid arteries, arterial hypertension, and a family history of cardiovascular disease. Emergency computed tomography angiography (CTA) revealed a contained rupture of the distal aortic arch *(Figure 1)*. Given his severe frailty, emergency aortic repair was performed via partial upper sternotomy. The distal arch rupture was located 2–3 cm proximal to the descending aorta, and definitive treatment required total arch replacement using a zone 0 FET combined with ascending aortic replacement. Bilateral antegrade cerebral perfusion was established during arch repair. Aortic cross-clamp time was 59 minutes, visceral ischemia time 17 minutes, and total cardiopulmonary bypass time 144 minutes. The patient was extubated on the first postoperative day (POD). Postoperative CT confirmed successful aortic arch repair (*Figure 2*). His subsequent postoperative course was uneventful, and he was discharged on 10 POD to a local rehabilitation facility in accordance with standard postoperative care. He returned home on POD 38.

Conclusion: Aortic arch surgery remains highly challenging particularly in acute setting, and minimally invasive FET procedures have been reported mainly in small elective series. This case illustrates that in carefully selected patients and in centres with dedicated expertise in minimally invasive aortic surgery, emergency FET with total arch replacement can be a feasible and safe treatment approach.

COI: No

Figure 1. Preoperative computed tomography reconstruction demonstrating rupture of the distal aortic arch.

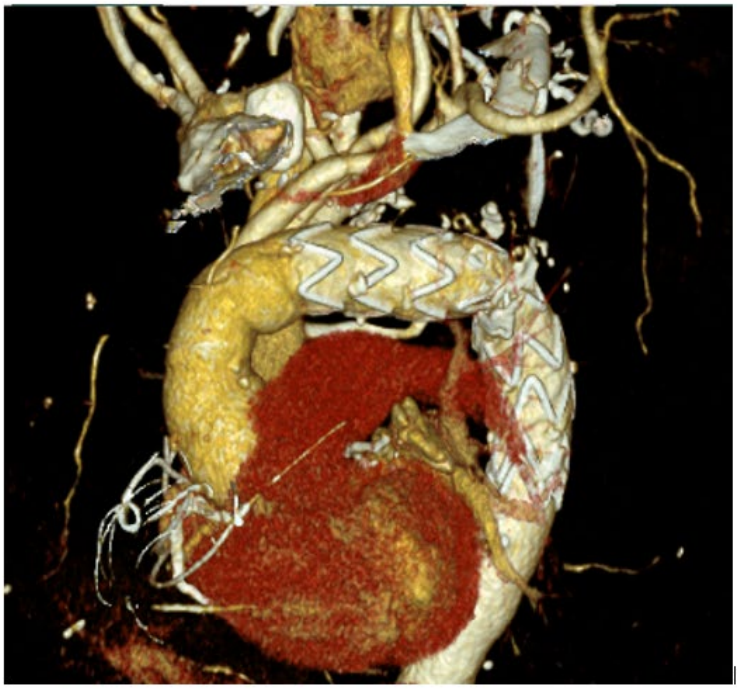

Figure 2. Postoperative computed tomography reconstruction demonstrating ascending aortic and total arch repair performed via a minimally invasive approach through partial upper sternotomy.

O074

Transcatheter Aortic Valve Implantation for Severe Aortic Insufficiency in LVAD-Supported Patients: A High-Risk but Feasible Strategy

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Introduction: Severe aortic insufficiency (AI) is a frequent and deleterious complication in patients supported with left ventricular assist devices (LVAD), resulting in ineffective forward flow, recirculation, worsening heart failure, and recurrent pulmonary edema. Management remains challenging, and redo surgery carries substantial risk. Transcatheter aortic valve implantation (TAVI) has emerged as an alternative, although procedural complexity and thromboembolic risk are major concerns.

Material & Methods: We report the case of a 66-year-old woman with an LVAD implanted for ischemic cardiomyopathy as a bridge to transplantation. Three months after implantation, she developed recurrent pulmonary edema despite optimized LVAD parameters. Echocardiography revealed newly developed severe AI due to cusp degeneration, associated with a thrombus along the non-coronary cusp. To avoid redo sternotomy prior to transplantation, a transfemoral TAVI was performed under general anesthesia with transesophageal echocardiographic guidance and bilateral cerebral embolic protection. A 26-mm Sapien 3 Ultra Resilia valve was successfully implanted. During the procedure, a thrombus adherent to the left-ventricular guidewire was aspirated using an 8F catheter, and a large thrombus was captured in the right-sided cerebral protection filter.

Results: Valve deployment was optimal, with complete elimination of aortic regurgitation. Upon awakening, the patient developed an acute right-sided hemisyndrome. Emergency cerebral imaging demonstrated occlusion of the right middle cerebral artery. Mechanical thrombectomy was promptly performed, achieving complete reperfusion (eTICI 3). The patient showed rapid neurological recovery and sustained hemodynamic improvement, with complete resolution of pulmonary edema.

Conclusion: This case highlights the feasibility and clinical value of transfemoral TAVI for severe AI in LVAD-supported patients, even in the presence of intracardiac thrombus. Despite a major thromboembolic complication, prompt recognition and intervention allowed a favorable outcome. TAVI represents a valuable alternative to redo surgery, preserving future surgical options in patients managed as bridge to transplantation or destination therapy.

COI: No

RAPID FIRE ABSTRACTS: PREVENTION, REHABILITATION & SPORTS CARDIOLOGY

O090

Intra-individual Lipoprotein(a) Variability and Risk Reclassification in Patients with Cardiovascular Disease

Chunyan Zhang[1], Gabrielle Huré[2], Arnaud Champetier[2], Felix Mahfoud[2], Gregor Leibundgut[2], Jasper Boeddinghaus[2], Jan Stouwe[2], Danielle Gualandro[2], Emel Kaplan[2], Michael Zellweger[2], Anna Marsano[3], Christian Müller[2]

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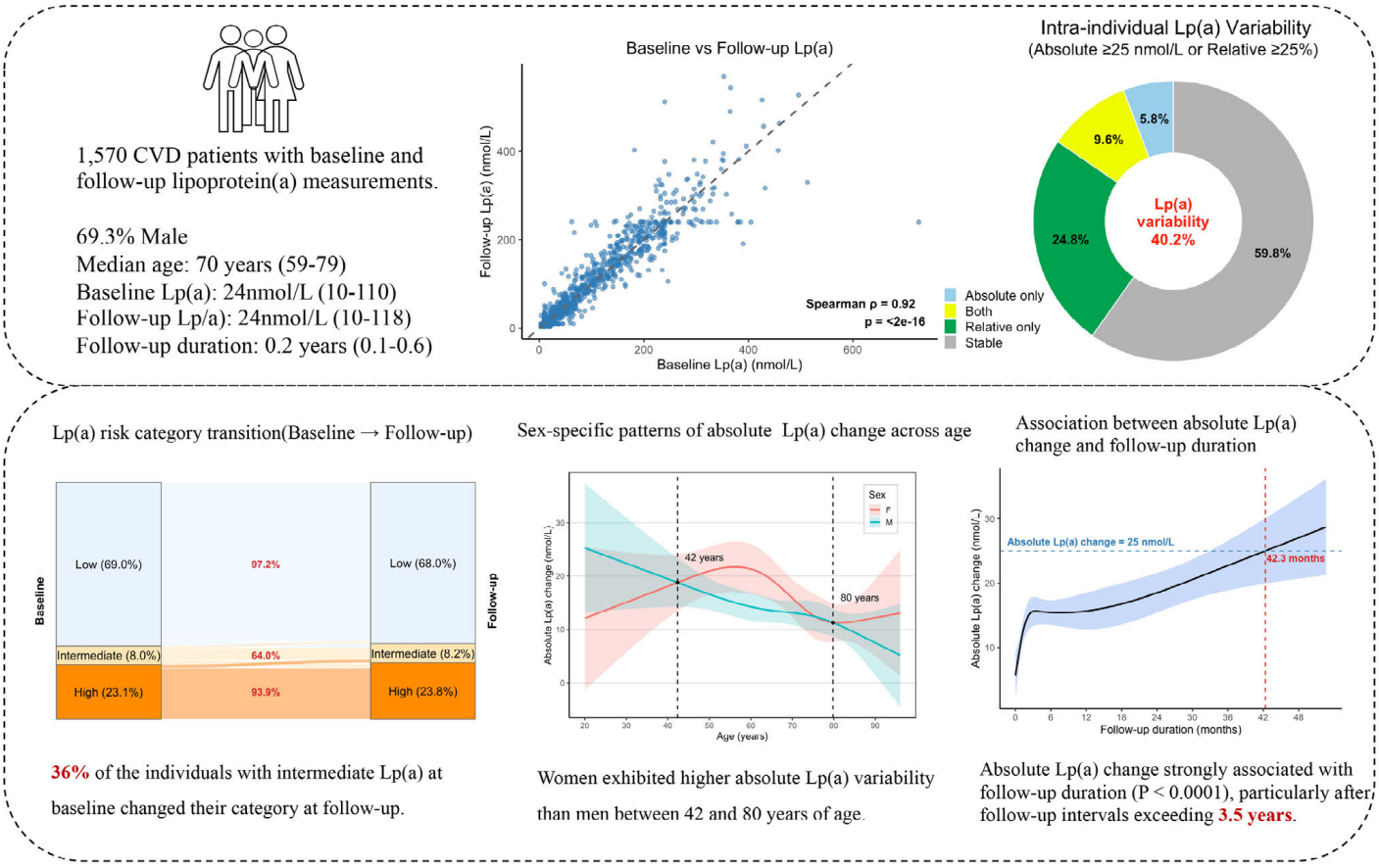
Introduction: Lipoprotein(a) (Lp(a)) is widely regarded as a genetically determined and stable cardiovascular risk factor, assumed to require only a single lifetime measurement. However, emerging evidence suggests that Lp(a) levels may fluctuate over time. This study aims to investigate the magnitude and prevalence of intra-individual Lp(a) variability in patients with established cardiovascular disease (CVD).

Material & Methods: We analyzed longitudinal Lp(a) data from 1,570 CVD individuals between 2020 and 2025 at University Hospital Basel. Intra-individual Lp(a) variability was defined as an absolute change ≥25 nmol/L or a relative change ≥25%. Baseline Lp(a) levels were categorized as low (<75 nmol/L), intermediate (75–124 nmol/L), or high (≥125 nmol/L), and category transitions were assessed. Associations between Lp(a) variability and age, sex, BMI, lipids, renal function, and follow-up duration were examined.

Results: Baseline and follow-up Lp(a) levels were strongly correlated (Spearman $\rho = 0.92$, $p < 0.001$), indicating overall population-level stability. Nevertheless, 15.4% of individuals exhibited an absolute Lp(a) change ≥25 nmol/L and 34.6% demonstrated a relative change ≥25%. Notably, 36% of individuals with intermediate baseline Lp(a) transitioned to a different risk category at follow-up. Lp(a) variability was more pronounced in females aged between 42 and 80 years than males of the same age. Lp(a) variability occurred significantly more often when the interval between measurements exceeded 3.5 years. In multivariable logistic regression models incorporating restricted cubic splines, changes in LDL-C were independently associated with Lp(a) variability ($P < 0.01$, nonlinear $P < 0.01$).

Conclusion: Intra-individual Lp(a) variability is substantial among CVD patients. Individuals with intermediate baseline Lp(a) levels are particularly prone to risk category reclassification. These findings challenge the concept of lifelong Lp(a) stability and support consideration of repeated Lp(a) measurements in selected patients.

COI: No



O091

Prognostic Value of Systolic Blood Pressure One Year After An Acute Coronary Syndrome: Insights from a Swiss Prospective Cohort

Henri Lu[1], Cédric Follonier[2], Louise Artels[1], Akshay Desai[3], David Carballo[2], David Nanchen[4], Thomas F. Lüscher[5], Christian Matter[6], Lorenz Räber[7], Olivier Muller[1], Nicolas Rodondi[8], Grégoire Wuerzner[1], François MacH[2], Baris Gencer[1]

[1]CHUV, Lausanne, Switzerland, [2]HUG, Geneva, Switzerland, [3]Brigham and Women's Hospital, Boston, United States, [4]Unisanté, Lausanne, Switzerland, [5]Royal Brompton and Harefield NHS Trust, London, United Kingdom, [6]University Heart Center, Zurich, Switzerland, [7]University Hospital Bern, Bern, Switzerland, [8]BIHAM, Bern, Switzerland

Introduction: Current guidelines recommend targeting a systolic blood pressure (SBP) below 130 mmHg in patients at high cardiovascular risk, including those with a prior acute coronary syndrome (ACS), although this target is largely extrapolated from trials in the general hypertensive population. We aimed to assess the prognostic value of SBP measured 1 year after ACS on subsequent cardiovascular outcomes.

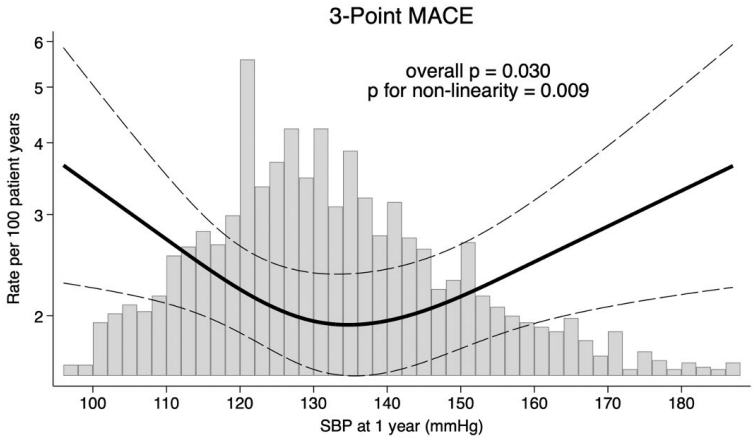
Material & Methods: The ELIPS cohort prospectively enrolled ACS patients between 2009 and 2017 across four Swiss tertiary centers. We included patients alive at 1 year post-enrollment with available SBP measurements at that timepoint. The association between continuous SBP and the risk of the composite outcome of 3-point major adverse cardiovascular events (MACE) – cardiovascular death, non-fatal stroke/transient ischemic stroke, non-fatal myocardial infarction – was assessed using restricted cubic splines, adjusted for age, sex, estimated glomerular filtration rate, cardiovascular risk factors and antihypertensive medication classes.

Results: A total of 2,074 patients (age 63±12 years; 82% male; SBP 132±19 mmHg) were included. Over a median follow-up of 4.0 years, 229 MACE occurred (event rate 2.7 [2.3–3.0] per 100py). The association between 1-year SBP and the risk of MACE demonstrated a nonlinear, J-shaped pattern ($P_{non\text{-}linearity}$ = 0.009; Figure 1), with lowest risk observed for SBP between ~125 and 135 mmHg (23.9% of patients), and an increase in risk for SBP above 135 mmHg (39.7% of patients). Above 135 mmHg, each 5-mmHg SBP increment was associated with a 7% higher risk of MACE (HR 1.07; 95%CI 1.00–1.14). The J-shaped association was consistent across age and sex, with no significant interactions ($P_{interaction}$ = 0.34 and 0.35, respectively; Figure 2).

Conclusion: In this well-characterized cohort of post-ACS patients, the lowest risk of cardiovascular events was observed with SBP levels between ~125 and 135 mmHg. Our results support a balanced SBP target after ACS that avoids both over- and undertreatment.

COI: No

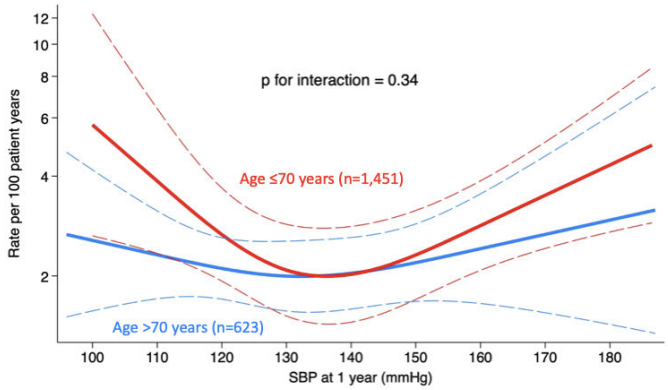
Figure 1. Risk-Adjusted Absolute Incidence Rates for 3-Point MACE According to Systolic Blood Pressure Measured at 1 Year



Adjustment for age, sex, BMI, use of RAAS inhibitors, beta-blocker, calcium channel blocker, diabetes, LDL-C level (all at 1 year), eGFR at baseline.
Abbreviations: BMI = body mass index; eGFR = estimated glomerular filtration rate; LDL-C = low-density lipoprotein cholesterol; MACE = major adverse cardiovascular events; RAAS = renin-angiotensin-aldosterone-system; SBP = systolic blood pressure.

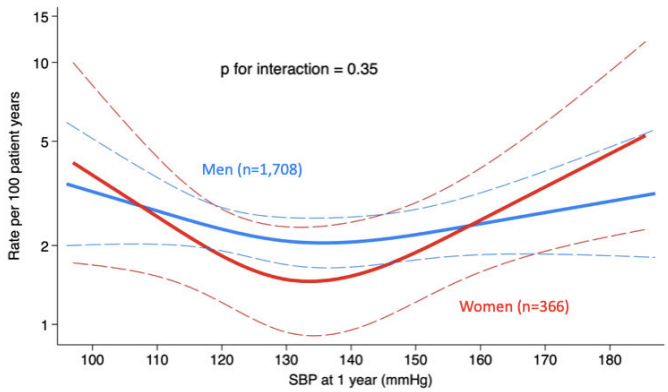
Figure 2. Adjusted Incidence Rates for 3-Point MACE Across the Spectrum of Systolic Blood Pressure Measured at 1 Year, (A) According to Age Groups; (B) According to Sex

A.



Adjustment for sex, BMI, use of RAAS inhibitors, beta-blocker, calcium channel blocker, diabetes, LDL-C level (all at 1 year), eGFR at baseline.

B.



Adjustment for age, BMI, use of RAAS inhibitors, beta-blocker, calcium channel blocker, diabetes, LDL-C level (all at 1 year), eGFR at baseline.
Abbreviations: BMI = body mass index; eGFR = estimated glomerular filtration rate; LDL-C = low-density lipoprotein cholesterol; MACE = major adverse cardiovascular events; RAAS = renin-angiotensin-aldosterone-system; SBP = systolic blood pressure.

O092

Association between lipoprotein(a) and ischemic brain lesions in patients with atrial fibrillation: insight from the Swiss-AF cohort

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[1]Geneva University Hospitals, Geneva, Switzerland, [2]Basel University Hospital, Basel, Switzerland, [3]Triemli Hospital Zurich, Zurich, Switzerland, [4]Bern University Hospital, Bern, Switzerland, [5]Cardiology Division, Ente Ospedaliero Cantonale (EOC) Cardiocentro Ticino Institute, Regional Hospital of Lugano, Lugano, Switzerland, [6]Population Health Research Institute, McMaster University, Hamilton, Canada, [7]Kantonsspital Baden, Baden, Switzerland, [8]Lausanne University Hospital, Lausanne, Switzerland

Introduction: Lipoprotein(a) (Lp(a)) exhibits both proatherosclerotic and prothrombotic properties and could be a prognostic biomarker in patients with atrial fibrillation (AF). We assessed the association between Lp(a) serum concentration and the presence of ischemic brain lesions in patients with AF.

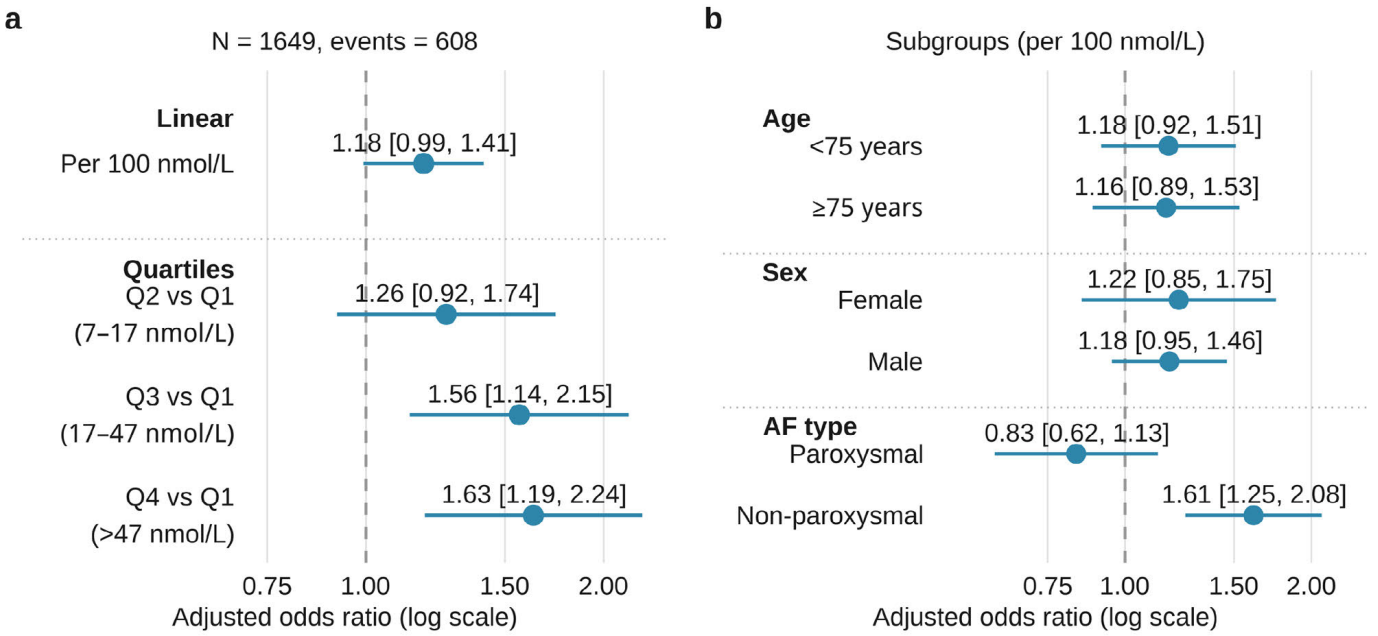
Material & Methods: Data were derived from the Swiss-AF cohort (N = 2,415), a multicenter prospective observational cohort of patients aged ≥65 years with documented AF. We included participants with Lp(a) serum concentration and brain magnetic resonance imaging (MRI) at baseline. Multivariable logistic regression was used, adjusting for prespecified confounders. Baseline Lp(a) was treated as continuous and categorized in quartiles. MRI-detected brain infarcts at baseline (with or without prior clinical stroke) were classified as large non-cortical or cortical infarcts (likely embolic) or small non-cortical infarcts (lacunar infarcts). Prespecified subgroup analysis included notably age category, sex, and AF type.

Results: Among 1'649 patients with baseline Lp(a) and MRI (72.6±8.4 years, 72.6% male, 54.7% non-paroxysmal AF), 608 (37.1%) exhibited ischemic brain lesions at baseline MRI. Median Lp(a) serum concentration was 16.5 nmol/l (IQR 6.7-47.1). Higher Lp(a) was associated with prevalent ischemic brain lesions, with a dose-response pattern across quartiles (p for trend = 0.001), driven by upper quartiles: adjusted OR for highest versus lowest quartile, 1.60; 95%CI, 1.17-2.20 (Figure, Panel A). The association appeared driven by likely embolic infarcts (OR per 100 nmol/l, 1.22; 95%CI, 1.00-1.48) rather than lacunar infarcts (OR per 100 nmol/l, 1.00; 95%CI, 0.82-1.22). There was significant effect modification by AF type (p for interaction = 0.002): per 100 nmol/l, adjusted OR was 1.60 (95%CI, 1.24-2.06) in non-paroxysmal AF versus 0.83 (95%CI, 0.61-1.12) in paroxysmal AF (Figure, Panel B).

Conclusion: In patients with AF, higher Lp(a) was associated with increased prevalence of ischemic brain lesions on MRI, especially in those with non-paroxysmal AF.

COI: This project was supported by the Swiss Atherosclerosis Association (Swiss Lipid Research Award 2024)

Lp(a) and ischemic brain lesions at baseline



Adjusted for age, sex, BMI, LDL-cholesterol, smoking, CKD, diabetes, hypertension, AF type, vascular disease, prior stroke/TIA, heart failure, major bleeding, oral anticoagulation, antiplatelet and lipid-lowering therapy.

O093

Use of Mediterranean Diet Adherence and Physical Activity for Refining Cardiovascular Risk Prediction in the General Population

Louise Artels[1], Pedro Marques-Vidal[2], Julien Vaucher[2 3], Peter Vollenweider[2], Roxane De La Harpe[2], Olivier Muller[1], Orestis Efthimiou[4], Baris Gencer[1]

[1]Department of Cardiology, Lausanne University Hospital and University of Lausanne, Lausanne, Switzerland, [2]Department of Internal Medicine, Department of Medicine, Lausanne University Hospital and University of Lausanne, Lausanne, Switzerland, [3]Department of Internal medicine, Department of Internal Medicine and Specialties, Fribourg Hospital and University of Fribourg, Fribourg, Switzerland, [4]Institute of Primary Health Care (BIHAM), University of Bern, Bern, Switzerland

Introduction: The SCORE2/SCORE2-OP, recommended for estimating 10-year risk of first atherosclerotic cardiovascular disease (ASCVD), does not include lifestyle factors. We investigated whether adding self-reported physical activity and Mediterranean diet adherence improves prediction beyond SCORE2/SCORE2-OP.

Material & Methods: We analyzed participants from the population-based CoLaus cohort with data for Physical Activity Frequency Questionnaire (PAFQ) and Mediterranean Diet Score (MedDietScore). Participants with CVD history, lipid-lowering therapy, LDL-cholesterol ≥4.9 mmol/L, diabetes or chronic kidney disease were excluded. Different Cox-based prediction models were tested: SCORE2 or SCORE2-OP depending on the participant age, alone or in addition to PAFQ, MedDietScore or both. Discrimination (Harrell's C), calibration slope and net reclassification improvement (NRI) were calculated. The primary outcome was first incident ASCVD (myocardial infarction, ischemic stroke and cardiovascular death).

Results: 2,587 participants (59% women; median age 55 years) were followed for a median duration of 9.0 years (IQR: 8.13-11.6), during which 145 ASCVD events occurred. Of these, 2,348 were younger than 70 years and evaluated using SCORE2, with 106 events. 239 participants were aged 70 years or older and assessed with SCORE2-OP, with 39 events. Adding physical activity or diet or both to SCORE2 did not improve discrimination (Harrell's C: 0.74 for all models), calibration (slope: 1.06 for all) nor reclassification (continuous NRI: -1.2, 95%CI: -2.23-17.7%) among participants aged <70. In contrast, adding lifestyle factors to SCORE2-OP improved discrimination (Harrell's C: 0.63 to 0.66), calibration (slope: 1.34 to 1.16) and reclassification (continuous NRI: 41%; 95%CI 7–74%). In older participants, event NRI was 28% (95%CI -3-59%) and non-event NRI was 13% (95%CI -1-27%). The decision curves analyses are presented in Figures 1a-b.

Conclusion: The addition of self-reported physical activity and Mediterranean diet may be clinical useful to improve ASCVD risk prediction in older adults beyond the traditional SCORE2-OP.

COI: No

Figure 1a: Decision curve analysis (<70 years)

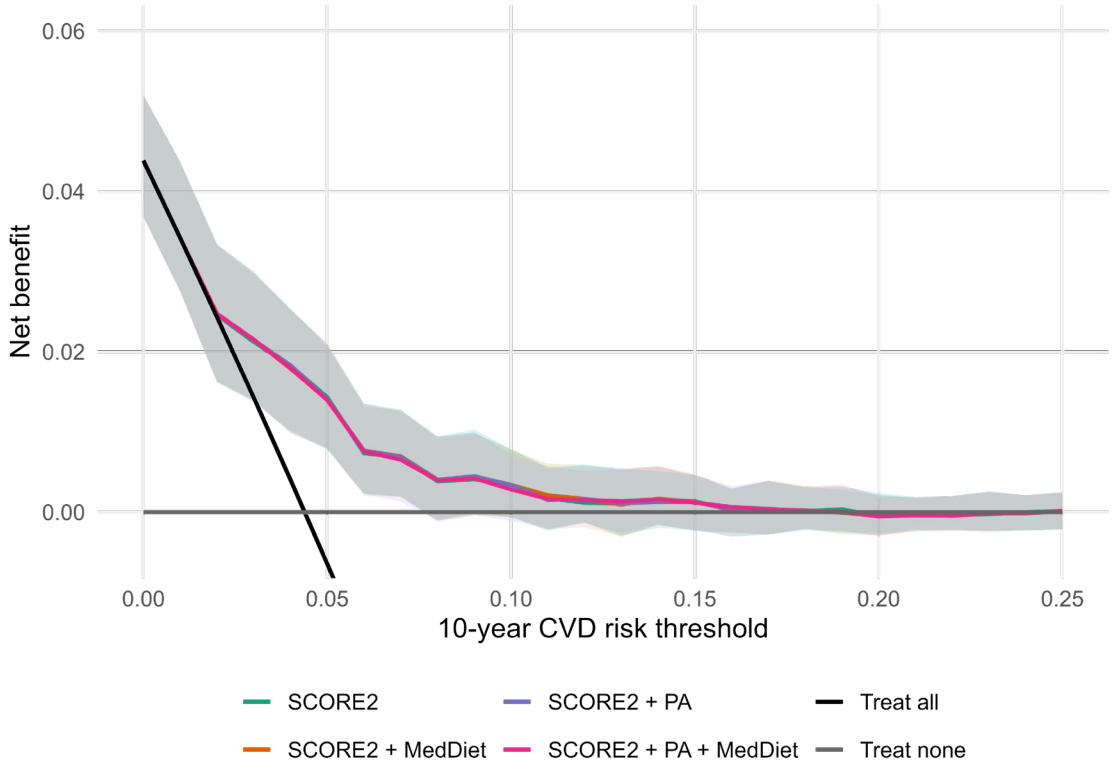
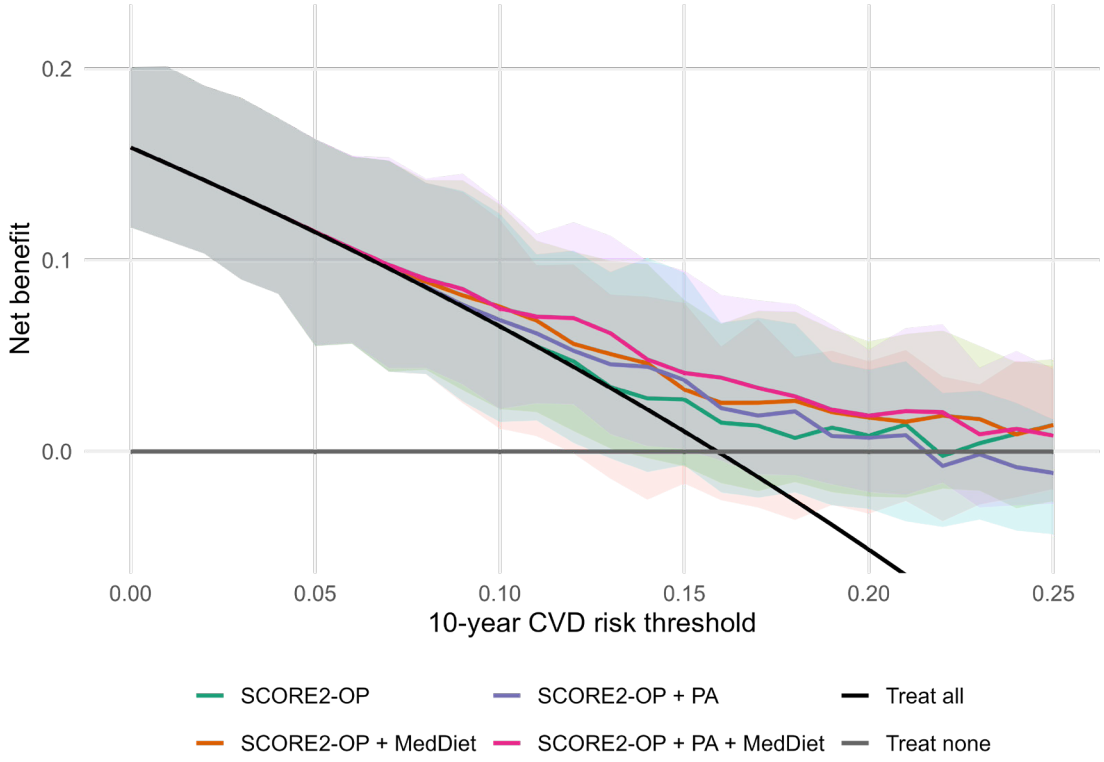


Figure 1b: Decision curve analysis (≥70 years)



O094

Epigenetic Aging and its association with cardiovascular and diseases: Systematic review and meta-analysis of cohort studies

Antoine Saudan[1 2], Cédric Follonier[1 2], Orestis Efthimiou[3], David Nanchen[4], Jonviea Chamberlain[4], Baris Gencer[5]

[1]Hôpitaux universitaires de Genève, Geneva, Switzerland, [2]Université de Genève, Geneva, Switzerland, [3]Universität Bern, Bern, Switzerland, [4]UNIL – Université de Lausanne, Lausanne, Switzerland, [5]CHUV Centre hospitalier universitaire vaudois, Lausanne, Switzerland

Introduction: Epigenetic clocks are models based on biomarkers developed to estimate an individual's biological age, independently of chronological age. These tools could eventually be useful to assess cardiovascular (CV) risk, but the evidence for their clinical utility remains limited.

Material & Methods: We conducted a systematic review of cohort studies reporting epigenetic clocks as exposure (with first-generation clocks Horvath and Hannum, and second-generation clocks PhenoAge and GrimAge) and ASCVD (atherosclerotic cardiovascular disease), including CVD incidents, coronary heart disease, and stroke as outcomes. We searched MEDLINE, Embase, and Google Scholar up to 01.08.2024. Adjusted hazard ratios (HRs) for each clock were extracted and standardized in 5-year increments. Pooled effect estimates were then derived using a random-effects meta-analysis.

Results: Of the 928 screened records, 18 were eligible for inclusion, of which 9 were included in the meta-analysis; this corresponds to a total of 29,009 participants, with 16,075 women (55.4%) and 3,445 ASCVD events. First-generation clocks showed modest associations. Six studies reported Horvath (N = 19,916, HR = 1.05, 95% CI = 1.02–1.08, I^2 = 0.1%), and seven reported Hannum (N = 26,916, HR = 1.08, 95% CI = 1.02–1.14, I^2 = 64.7%). Second-generation clocks demonstrated stronger associations. Five studies reported GrimAge (N = 22,402, HR = 1.31, 95% CI = 1.11–1.55, I^2 = 97.7%). Five studies reported DNAmPhenoAge (N = 22,402, HR = 1.14, 95% CI = 1.11–1.17, I^2 = 0.1%).

Conclusion: Epigenetic age acceleration measured using GrimAge is associated with an increased risk of CV disease. These findings support the potential utility of integrating epigenetic clocks into CV risk assessment or as potential surrogate markers in studies investigating interventions aimed at reducing CV risk.

COI: No

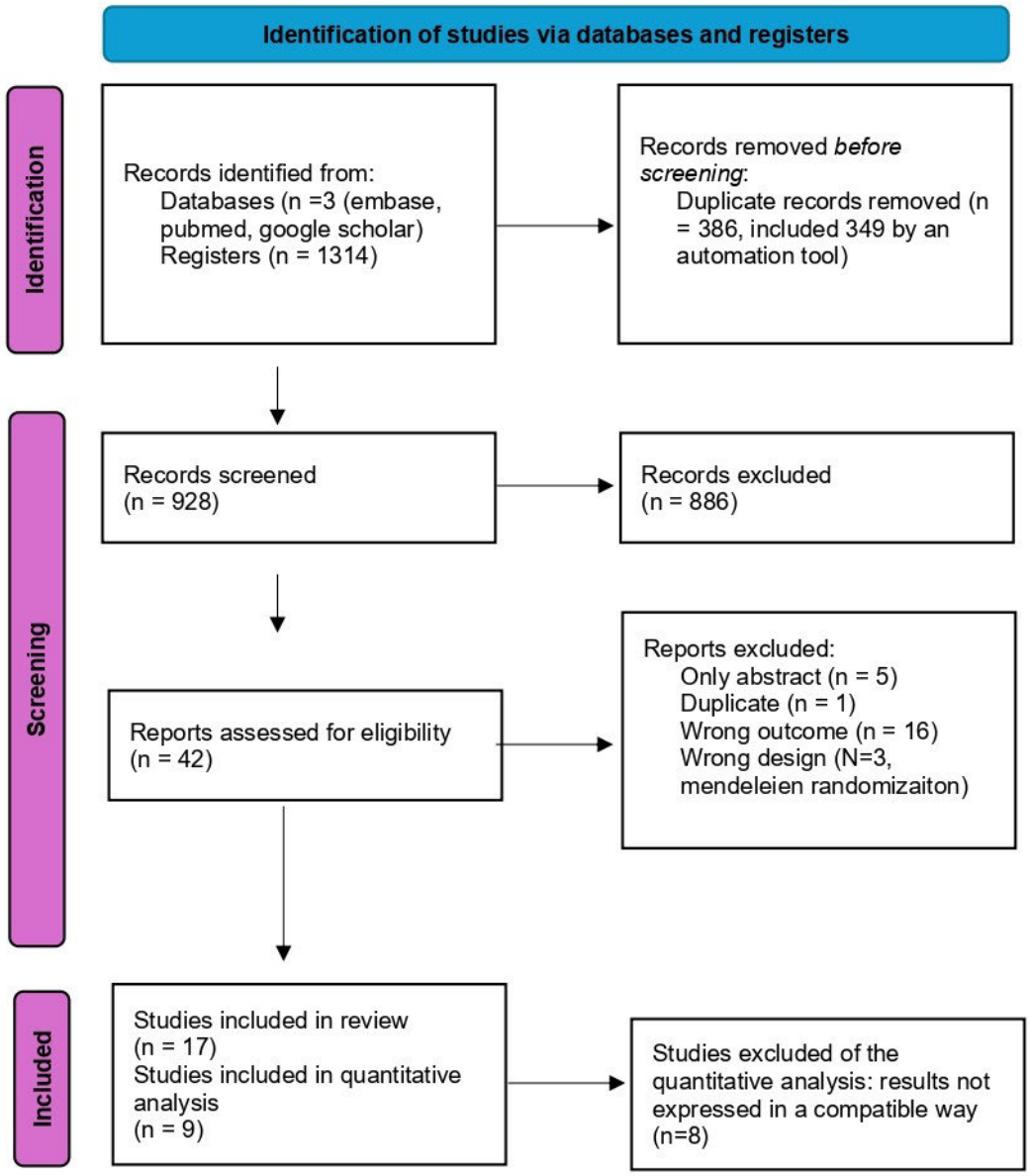
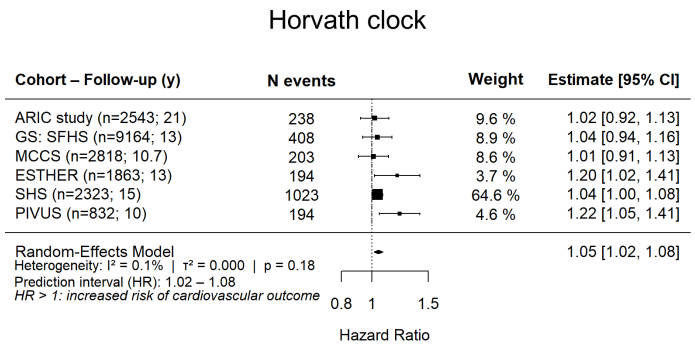
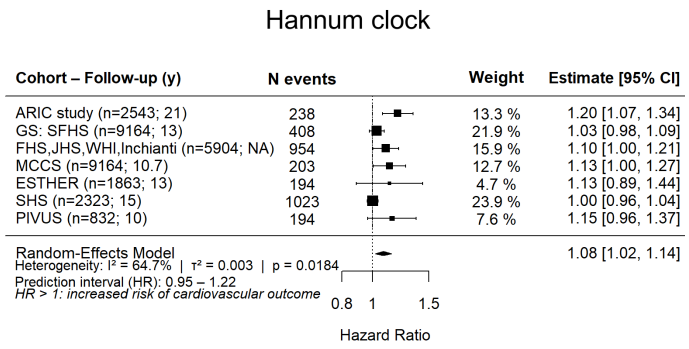
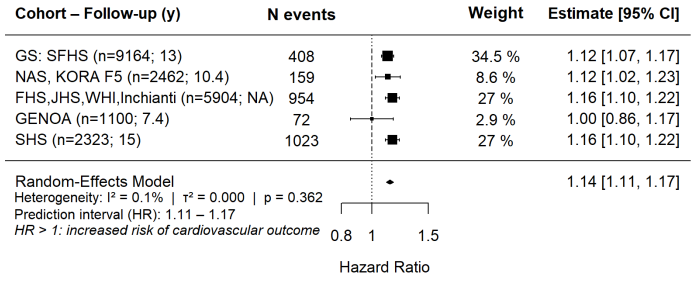
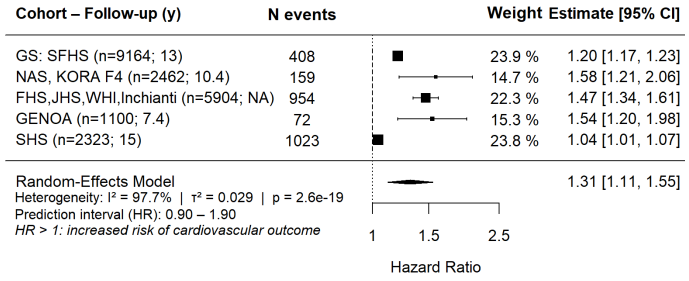


Figure 1 : Prisma flow diagram









O095

Circulating Extracellular Vesicles as Unconventional Biomarkers for Cardiovascular Risk Stratification and MACE Prediction

Elena Caporali[1], Jacopo Burrello[2], Valentina Guerini Giusteri[1], Jessica Goi[2], Gabriele Leone[2], Silvia Monticone[2], Lorenzo Grazioli Gauthier[1], Giovanni Bianco[3], Marco Vabanesi[3], Carlo W. Cereda[3], Giovanni Pedrazzini[1], Lucio Barile[1]

[1]Cardiocentro Ticino Institute, Ente Ospedaliero Cantonale, Lugano, Switzerland, [2]Department of Medical Science, University of Torino, Torino, Italy, [3]Neurocenter of Southern Switzerland, Ente Ospedaliero Cantonale, Lugano, Switzerland

Introduction: Extracellular vesicles (EVs) are nanosized bilayer membrane particles involved in the pathophysiology of cardiovascular diseases. Their molecular cargo reflects cellular origin and activation state of the parental cells, making EVs a promising source of novel biomarkers. This study aimed to identify unconventional biochemical biomarkers associated with the occurrence of major adverse cardiovascular events (MACE) at long-term follow-up and to compare their predictive performance with conventional clinical and biochemical parameters.

Material & Methods: We performed a retrospective analysis of an international cohort of patients referred to tertiary centers for cardiovascular (CV) risk assessment. Surface antigens carried by EVs were measured by flow cytometry and compared between patients who experienced a MACE (including CV death, acute myocardial infarction, stroke, or coronary revascularization) and those with an event-free follow-up.

Results: Of the 323 patients included, 42 (13%) experienced a MACE over a median follow-up of 6.7 years. Among conventional biomarkers, LDL cholesterol and C-reactive protein levels were higher in patients who developed a MACE (3.4 vs 3.0 mmol/L and 6.2 vs 4.7 mg/L, respectively). Of the 37 evaluated EV surface antigens, endothelial- and platelet-derived markers – including CD105, CD62P, CD42b, CD42a, and CD31 – were significantly increased in the MACE group compared with controls ($p \leq 0.01$). EV markers demonstrated a reliable predictive performance, with an AUC of up to 0.807 and an accuracy of 86.4%, outperforming CV risk stratification based on the SCORE-2 system ($p < 0.05$).

Conclusion: The evaluation of unconventional biomarkers, such as EV surface antigens, may improve CV risk stratification and enhance the prediction of MACE. After validation in prospective longitudinal studies, EV profiling could be integrated into CV risk assessment.

COI: No

O096

Cardiometabolic risk factor management and adherence to antihypertensive therapy in patients with severe obesity prior and after bariatric surgery: a cohort study

Michael Kunz[1], Juel Maalouli Schaar[2], Lara Keller[3], Verena Keller[4], Lucas Lauder[1], Felix Götzinger[1], Michael Böhm[3], Christian Müller[1], Martin Schulz[5], Ammar Al-Ali[6], Philipp Scherber[6], Sebastian Holländer[6], Markus Meyer[2], Felix Mahfoud[1]

[1]Clinic of Cardiology, University Heart Center Basel, University Hospital Basel, Basel, Switzerland, [2]Department of Experimental and Clinical Toxicology and Pharmacology, Center for Molecular Signaling (PZMS), PharmaScienceHub (PSH), Saarland University, Homburg, Germany, [3]Department of Internal Medicine III, Cardiology, Angiology and Intensive Care Medicine, University Hospital Saarland, Saarland University, Homburg, Germany, [4]SHG Kliniken Sonnenberg, Klinik für Geriatrie, Saarbrücken, Germany, [5]German Institute for Drug Use Evaluation (DAPI), Berlin, Germany, [6]Department of General, Visceral, Vascular and Pediatric Surgery, University Clinic of Saarland, Homburg, Germany

Introduction: Patients with obesity continue to experience excess mortality. Inadequate cardiovascular risk factor (CVRF) management and poor medication adherence may contribute to this risk. This study aimed to assess (i) CVRF management, (ii) prescription of guideline-directed medical therapy (GDMT), and (iii) adherence to antihypertensives (AHT) in patients scheduled for bariatric surgery.

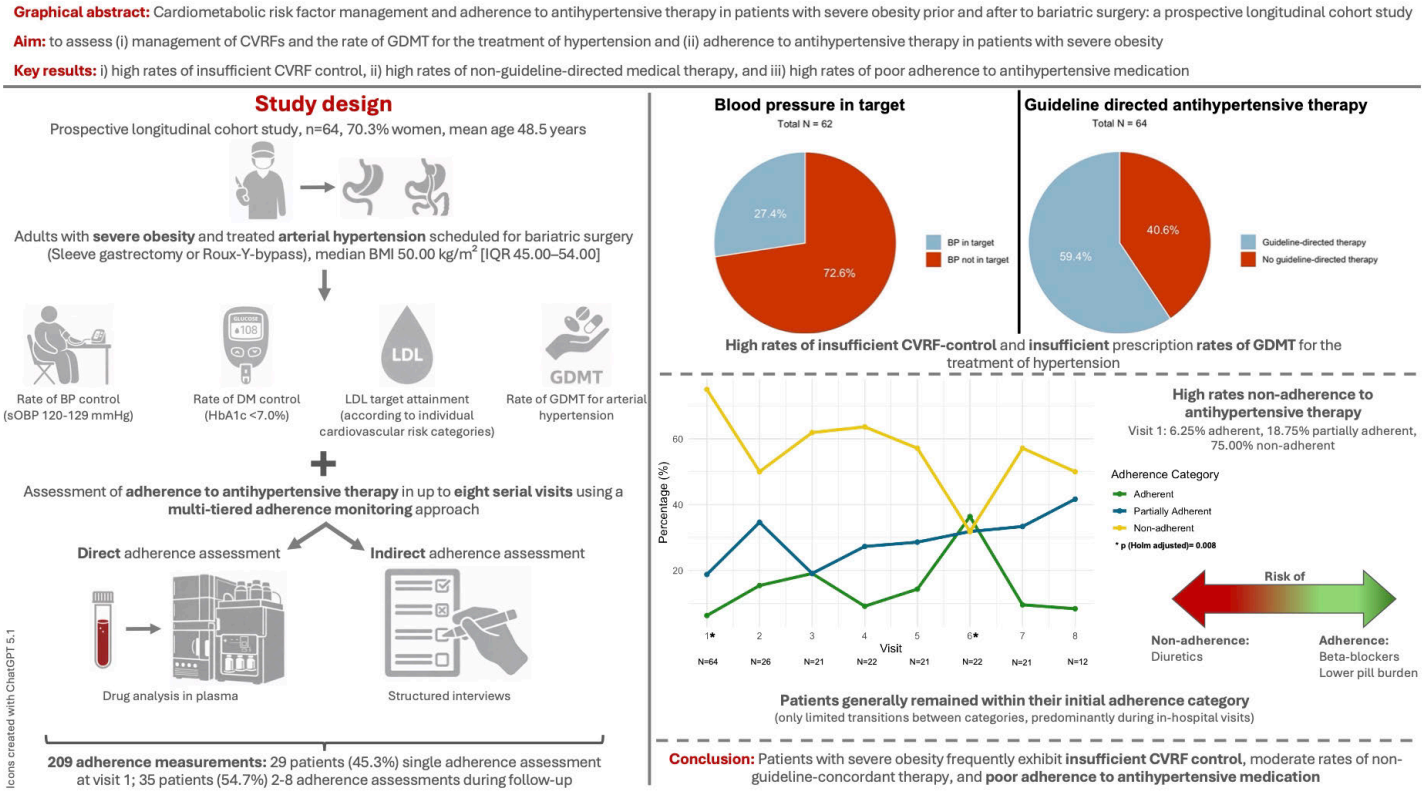
Material & Methods: In this cohort study, adults with body mass index (BMI) >35 kg/m^2 and arterial hypertension scheduled for

bariatric surgery were enrolled. CVRF control was evaluated and adherence was assessed in up to eight serial visits (V1-4 prior and V5-8 after surgery) using liquid chromatography-high-resolution mass spectrometry-based plasma drug analysis. Factors associated with adherence were analyzed using mixed-effects logistic regression and Cox proportional hazards models.

Results: Of 80 screened patients, 64 were included (70% women; mean age 48.5±10.4 years; median BMI 50.0 kg/m² [IQR 45.0–54.0]) with 209 available adherence assessments. At V1, guideline-recommended targets were achieved in 27% for blood pressure, 40% for glycaemic control in patients with diabetes, and 16% for LDL-cholesterol. GDMT for hypertension was prescribed in 59% of patients. At V1, only 6% were fully adherent, 19% partially, and 75% non-adherent. Adherence remained largely stable during follow-up, with few transitions between adherence categories, primarily during in-hospital stays. Improvement of BP control after bariatric surgery was not attributable to improvement in adherence (p = 1.0). Diuretic therapy was associated with lower odds of adherence (OR 0.13, 95% CI 0.04-0.47, p = 0.0019), while beta-blocker therapy was associated with a reduced hazard of non-adherence over time (HR 0.38, 95% CI 0.20-0.73, p = 0.0034). Each additional three drug classes were associated with a reduced likelihood of adherence (OR 0.18, 95% CI 0.04-0.71, p = 0.0148).

Conclusion: Among patients undergoing bariatric surgery, CVRF control is frequently insufficient, GDMT is inconsistently prescribed, and adherence to AHT is poor. These findings underscore the need for structured adherence-enhancing interventions.

COI: MK has received consulting fees from Merck (Merck KGaA).



Graphical abstract

BMI – body mass index in kg/m², BP – blood pressure, CVRF – cardiovascular risk factor, DM – diabetes mellitus, GDMT – guideline directed medical therapy, LDL - low-density lipoprotein cholesterol, sOBP – systolic office blood pressure.

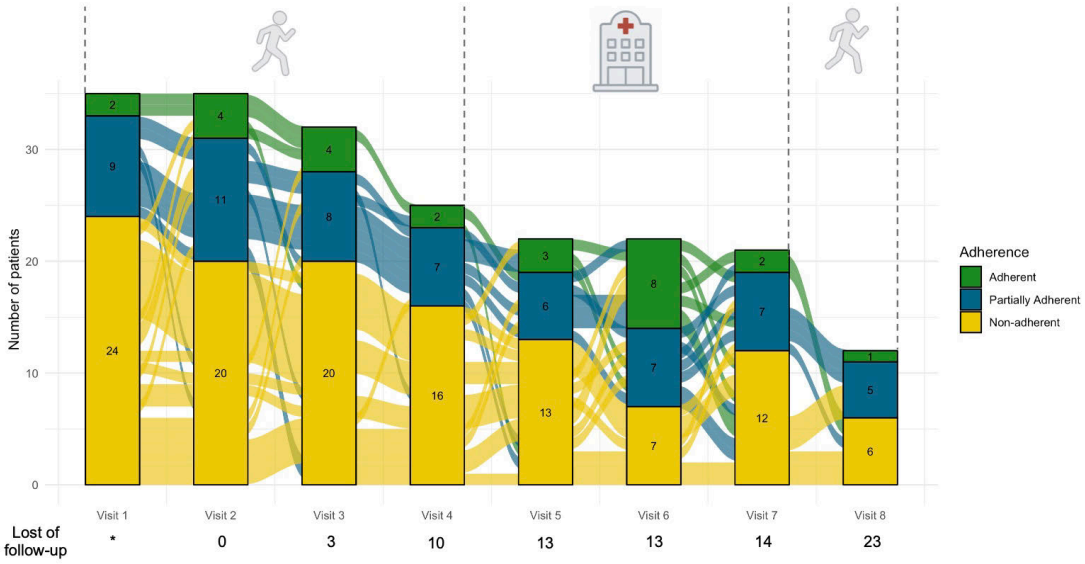


Figure 4: Adherence trajectories across visits

The walking figure icon denotes outpatient visits, whereas the hospital icon indicates in-hospital assessments.

The absolute numbers shown in Figure 4 do not correspond to the number of patients assessed at each visit. If a patient missed one or more intermediate visits, the adherence category from the last available visit was carried forward until the next documented assessment. For example, a patient classified as adherent at visit 1 who did not attend visit 2 but was classified as non-adherent at visit 3 was considered adherent at visits 1 and 2 and switched to non-adherent at visit 3. This approach in the figure reflects the absence of information for missed visits and represents a methodological limitation, as the true adherence status at the missed visit cannot be determined.

ABSTRACT SESSION: CONGENITAL & PEDIATRIC CARDIOLOGY

O099

Loeys-Dietz Syndrome: Outcome of 121 Patients in the Current Era

Jasraj Singh[1], James Howick[1], Benjamin Gochanour[1], David Harmon[1], Christine Attenhofer Jost[2], David Deyle[1], Heidi Connolly[1], Christopher Scott[1], Juan Bowen[1]

[1]Mayo Clinic, Rochester MN, United States, [2]HerzGefässZentrum Im Park, Hirslanden Klinik Im Park, Zurich, Switzerland

Introduction: To evaluate clinical, genetic, echocardiographic features, survival and cardiovascular complications in patients with Loeys-Dietz syndrome (LDS).

Material & Methods: Retrospective study analyzing data from 121 LDS patients (50% females) from 95 families with various LDS mutations [TGFBR1 (n = 35 patients), TGFBR2 (n = 34), SMAD3 (n = 33), and TGFB2/TGFB3 (n = 19)]. Kaplan Meier analysis was used to assess rates of survival and aortic or other dissection. The mortality outcome was left truncated at the patient's first visit to Mayo Clinic Cardiology.

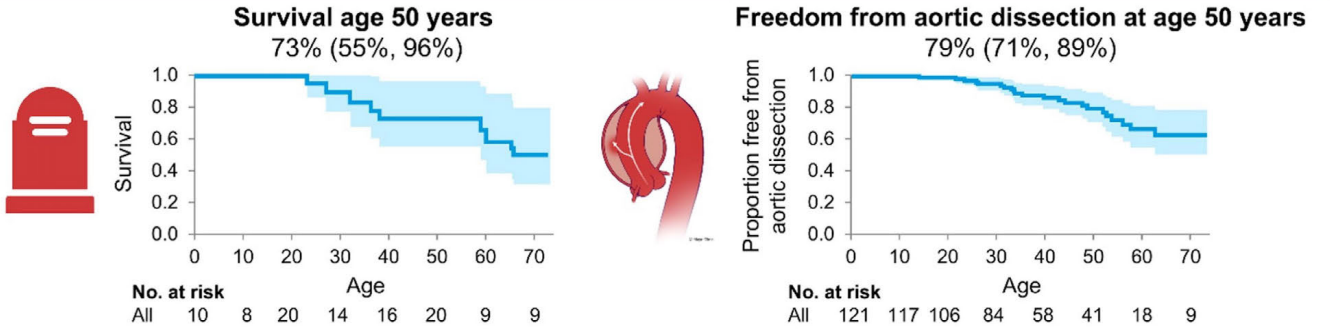
Results: Median age at first Mayo CV visit was 37 years (IQR 21, 50), age at last contact was 43 years (IQR 29, 58). Prevalence of aneurysm of aortic root (69%) and ascending aortic aneurysm (47%) was high. The median maximum aortic root size was 43 mm (37, 49), ranging up to 80 mm. Aneurysms were also observed in abdominal aorta (18%), cerebral (17%), iliac (16%), visceral (4%) and/or brachial arteries (2%). Aortic dissection occurred in 23 patients (74% type A, 17% type B, 9% abdominal): rates were similar across genetic subgroups. Dissection of vessels other than the aorta/iliac artery occurred in 24 patients. In Kaplan Meier analysis, only 36% (95% CI 24-55%) of patients were dissection free at 70 years; rates of dissection did not differ significantly between mutations. In patients with at least one cardiac surgery (n = 80), first surgery was elective in most (75%). The most common surgeries were hemiarch replacement (47%), valve sparing aortic root replacement (45%), and aortic root/valve replacement (30%). Overall survival at 70 years was 50% (95% CI 31-80%).

Conclusion: Outcome in LDS patients followed at our center is better than previously reported due to advances in surveillance and surgical management. However, aortic and other vessel aneurysms and dissection remain high and special attention is needed to identify these complications.

COI: No

Retrospective Analysis of 121 Loeys-Dietz Patients seen at Mayo Clinic

Clinical outcome



	TGFBR1 (N=35)	TGFBR2 (N=34)	SMAD3 (N=33)	TGFB2/TGFB3 (N=19)	Total (N=121)	P
Aortic root aneurysm	27 (77%)	23 (68%)	22 (67%)	11 (58%)	83 (69%)	0.519
Ascending aorta aneurysm	18 (51%)	16 (47%)	17 (52%)	6 (32%)	57 (47%)	0.501
Aortic dissection (n = 23) by type	7	6	8	2	23	0.462
• Type A	5 (71%)	4 (67%)	7 (88%)	1 (50%)	17 (74%)	
• Type B	1 (14%)	2 (33%)	0 (0%)	1 (50%)	4 (17%)	
• Abdominal	1 (14%)	0 (0%)	1 (12%)	0 (0%)	2 (9%)	
Carotid dissection	4 (11%)	3 (9%)	3 (9%)	1 (5%)	11 (9%)	0.971
Urgent/emergency aortic surgery	7 (25%)	4 (23%)	8 (36%)	1 (8%)	20 (24%)	0.272
Number of cardiovascular surgeries						0.038
• Single	20 (57%)	6 (18%)	11 (33%)	5 (26%)	42 (35%)	
• Multiple	8 (23%)	12 (35%)	11 (33%)	7 (37%)	38 (31%)	
Aortic valve and root replacement	12 (34%)	10 (29%)	10 (30%)	4 (21%)	36 (30%)	0.792
Valve sparing aortic root replacement	21 (60%)	13 (38%)	14 (42%)	7 (37%)	55 (45%)	0.222

Cause of death is often cardiovascular (7 of 11 patients)

O100

Accuracy of CT-Derived Anomalous Coronary Arteries Anatomy Versus 3D-Reconstructed IVUS Reference

Anselm Stark[1], Ali Mokhtari[1], Ryota Kakizaki[1], Marius Bigler[1], Marc Ilic[1], Lorenz Räber[1], Christoph Gräni[1]

[1]*Department of Cardiology, Inselspital, Bern University Hospital, University Bern, Bern, Switzerland*

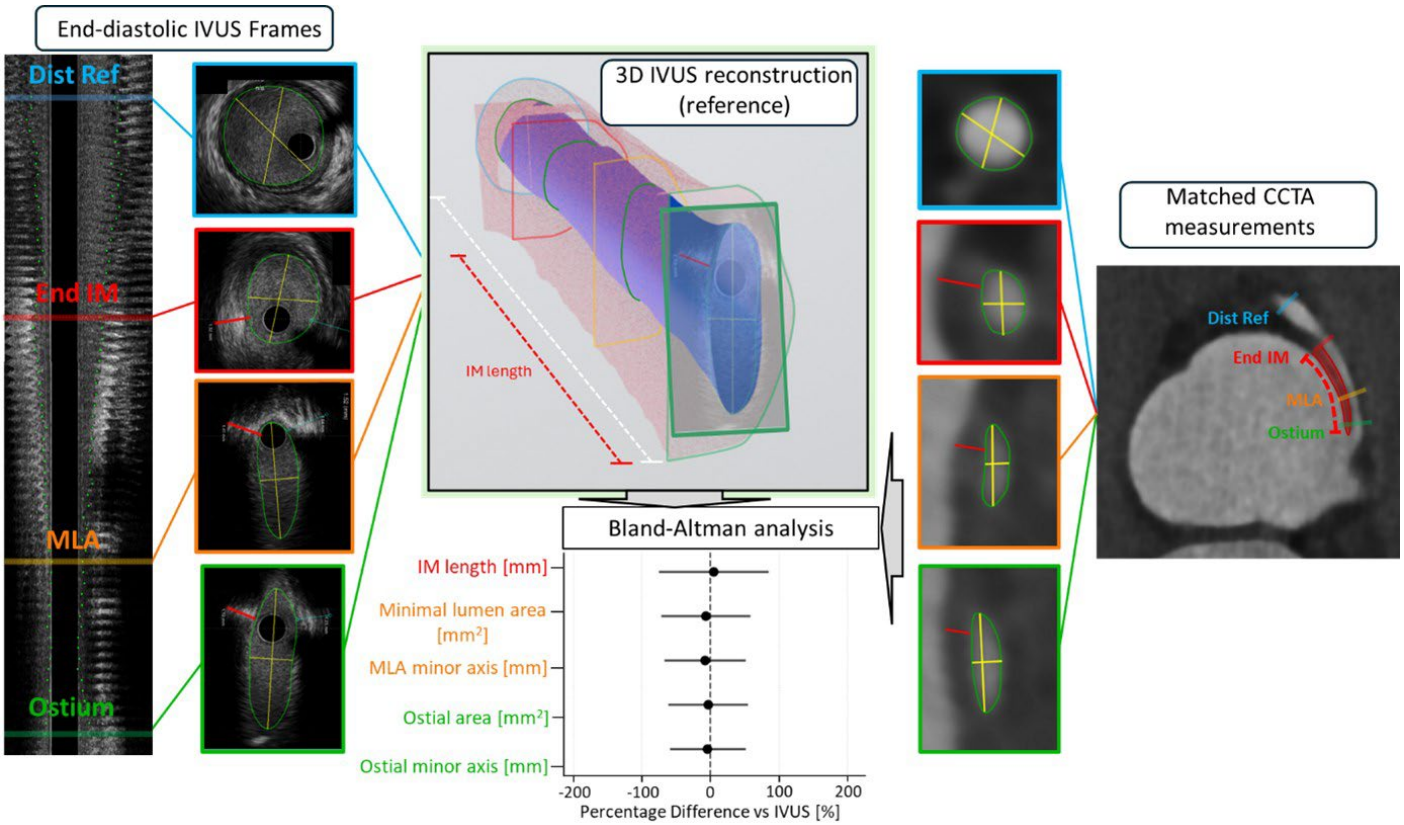
Introduction: In anomalous aortic origin of a coronary artery (AAOCA) with an interarterial and intramural course, noninvasive anatomical characterization is primarily performed using coronary CT angiography (CCTA). However, the accuracy of its anatomical measurements lacks validation against a high-fidelity in-vivo standard. The aim of this study is to validate CCTA-derived lumen dimensions and intramural length (IML) against a 3D-reconstructed intravascular ultrasound (IVUS) reference standard.

Material & Methods: Consecutive patients with AAOCA underwent CCTA and IVUS. IVUS pullbacks were automatically segmented, end-diastole identified, and 3D models reconstructed. CCTA measurements (i.e. ostial area, minor axis, major axis, elliptic ratio, IML) were obtained using clinical techniques, including several methods for IML estimation (i.e. interluminal space thickness (ILS), elliptic ratio and pericoronary fat sign). Agreement was assessed via Pearson correlation and Bland-Altman analysis; reliability via intraclass correlation coefficients (ICCs) and geometric reliability with point-by-point distances.

Results: A total of 50 AAOCA patients (mean age 52.3±11.3 years; 30% female) were analyzed. Strongest CCTA-IVUS correlations were for linear ostial dimensions (circumference r = 0.66, major axis r = 0.62; p <0.001). For IML, the ILS method correlated best (r = 0.64, p <0.001) but showed wide limits of agreement (bias +4.8%; limits of agreement -74.1% to +83.7%). All CCTA IM-length methods overestimated versus IVUS. CCTA intra-reader reliability was good for ostial measures (ICC 0.83-0.93) and ILS (ICC 0.92). The IVUS 3D reconstruction demonstrated excellent geometric reliability (geometric error 0.16 (0.10-0.26) mm) and ICC ranging from 0.92-0.97.

Conclusion: While CCTA remains the essential, non-invasive test for AAOCA screening, this study reveals significant limitations in its precision for detailed anatomical quantification, particularly for IML measurements. The ILS method shows promise as the most accurate CCTA technique. The superior accuracy and reproducibility of 3D IVUS highlight it as a valuable reference.

COI: No



O101

Predictive Value of CT-FFR in Right Anomalous Aortic Origin of Coronary Artery in a middle-aged population

Shea Grob[1], Marius Bigler[1], Anselm Stark[1], Adriana Ferroni[1], Ryota Kakizaki[1], Lorenz Räber[1], Ronny Büchel[2], Isaac Shiri[1], Christoph Gräni[1]

[1]Department of Cardiology, University Hospital Bern (Inselspital), University of Bern, Bern, Switzerland, [2]Department of Nuclear Medicine, Cardiac Imaging, University Hospital Zurich, Zurich, Switzerland

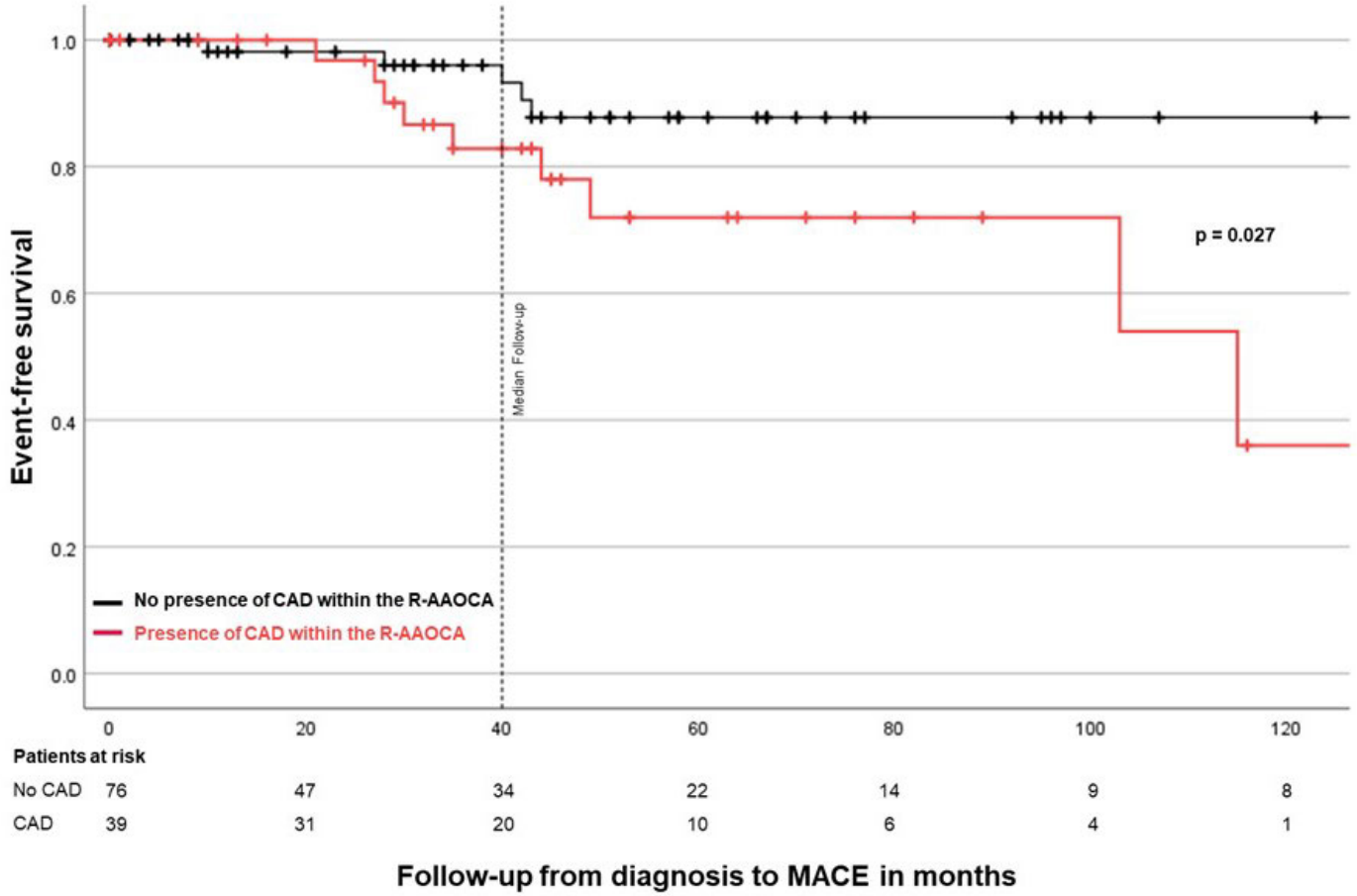
Introduction: Right anomalous aortic origin of a coronary artery (R-AAOCA) is a rare congenital anomaly potentially associated with adverse events. The growing use of coronary computed tomography angiography (CCTA) in middle-aged patients with suspected coronary artery disease (CAD) has led to increased detection of AAOCA and a noninvasive tool to predict cardiovascular outcomes in AAOCA is needed. However, the prognostic value of CCTA-derived fractional flow reserve (CT-FFR) in this population remains unclear.

Material & Methods: In this study, consecutive patients with R-AAOCA were selected from two registries (University Hospital of Bern and Zurich). All participants underwent CT-FFR analysis using cFFR software (Siemens-Healthineers) alongside additional CCTA-based quantification of CAD and anatomical features. Follow-up assessments were conducted through telephone interviews and clinical reports. Major adverse cardiac events (MACE) were defined as death of all cause, (survived) sudden cardiac death, myocardial infarction and urgent/non-urgent revascularization of the anomalous vessel.

Results: 116 patients (25% female; mean age 58±13years) were included in the final analysis. Of these, 31 patients (26%) underwent AAOCA-related treatment. After a median follow-up of 40months (IQR 9-68months), 14 patients (12%, thereof 64% with a treatment) experienced a first MACE, including 7 deaths, 1 myocardial infarction, and 6 non-urgent revascularizations. No anatomical feature could independently predict MACE. CT-FFR emerged as a significant predictor (odds ratio (OR) 0.022, 95%-CI 0.001-0.650, p = 0.027), but this association lost significance after adjusting for CAD within the anomalous vessel (OR 0.103; p = 0.229). OR for presence of CAD within the anomalous vessel was 4.181 (95%-CI 1.296-13.490, p = 0.017). Kaplan-Meier analysis demonstrated a significant difference in event-free survival based on the presence of CAD in the anomalous vessel, but not for CT-FFR.

Conclusion: In middle-aged patients with R-AAOCA, only CAD in the anomalous vessel, but not CT-FFR or anatomical features, was predictive of adverse outcomes, highlighting the prognostic relevance of atherosclerosis in this population.

COI: No



Kaplan-Meier estimates of major adverse cardiac event (MACE)-free survival stratified by the presence or absence of coronary artery disease in the anomalous right coronary artery. Statistical significance was assessed using the log-rank test.

O102

Long-Term Stroke Recurrence And Safety After Percutaneous Patent Foramen Ovale Closure: Results From The Lucerne Registry

David Möller[1], Federico Moccetti[1], Nina Conrad[1], Matthias Bossard[1], Adrian Attinger-Toller[1], Florim Cuculi[1], Stefan Toggweiler[1], Mathias Wolfrum[1]

[1]*Canton hospital of Lucerne, Heart Center Lucerne, Department of Cardiology, Lucerne, Switzerland*

Introduction: Percutaneous patent foramen ovale (PFO) closure is the gold-standard treatment for patients with PFO and cryptogenic stroke as well as other dedicated indications; however, long-term outcome data remain limited.

Material & Methods: Consecutive patients undergoing percutaneous PFO closure between September 2017 and December 2024 at the Heart-Center-Lucerne were enrolled and evaluated for recurrence of cerebrovascular events (primary endpoint), procedural success, procedure-related complications, echocardiographic and clinical outcomes. Follow-up consisted of echocardiographic assessment at 6 months as well as clinical follow-up of up to 8 years using electronic-health-records and a structured telephone interview.

End Points	Number (%)
Combined Endpoints/Events	12 (3.63%)
Stroke	5 (1.5%)
Ischemic	3 (0,9%)
Hemorrhagic	2 (0.6%)
TIA	5 (1.5%)
Peripheral embolism	0
Death	2 (0.6%)
Complications	
Bleeding	9 (2.7%)
Minor	8 (2.4%)
Major	1 (0.3%)

Table 1: Primary and secondary outcomes.

Results: 331 patients underwent PFO closure (mean age 51±12 years, 39% female) with cryptogenic stroke (80%) as the primary indication. An atrial-septal-aneurysm was present in 31% and grade 3 shunt in 72% of cases, with a median RoPE-Score of 7 (IQR 6-7). Acute technical success was achieved in 99% of procedures. Amplatzer-devices were most frequently used (52%), followed by Figulla-Flex-devices (42%), Noble Stitch (6%) and Gore-Devices (1%). The rate of effective-closure (residual shunt >1) differed between double-disc occluders and suture-based devices (97% vs 68%, p <0.001). Thirty-three percent of patients treated with Noble Stitch (7/21 patients) had a relevant residual shunt and subsequently underwent treatment with a double-disc-device. During a median follow-up of 3.9 years (IQR 2.1-5.7), 10 cerebrovascular events occurred, including 5 strokes (3 ischemic, 2 hemorrhagic) and 5 transient ischemic attacks (Figure 1) and 2 deaths (Table 1), corresponding to a recurrence rate of 0.39 strokes/100 patient-years. Patients with a residual shunt >1 showed a trend toward increased cerebrovascular risk during follow-up (HR 4.3, CI 0.9–20.9, p = 0.068). Post-interventional atrial fibrillation was detected in 5.4% of patients.

Conclusion: This registry demonstrates durable long-term efficacy and an acceptable safety profile of percutaneous PFO closure in a real-world cohort. A significant residual shunt was associated with a trend toward increased cerebrovascular event rates.

COI: Dr Toggweiler serves as a consultant and proctor for Medtronic, Boston Scientific, Edwards Lifesciences and Biosensors; a proctor for Edwards Lifesciences and Abbott Vascular; and as a consultant for Medira, Shockwave, Teleflex, AtHeart Medical, Veosource, and Polares Medical; has received institutional research grants from Boston Scientific, Fumedica and Novartis; and holds equity in Hi-D Imaging. Dr. Wolfrum serves as proctor for Biosensors and holds equity in Hi-D Imaging. The remaining authors report no financial relationships or conflicts of interest regarding the content herein.

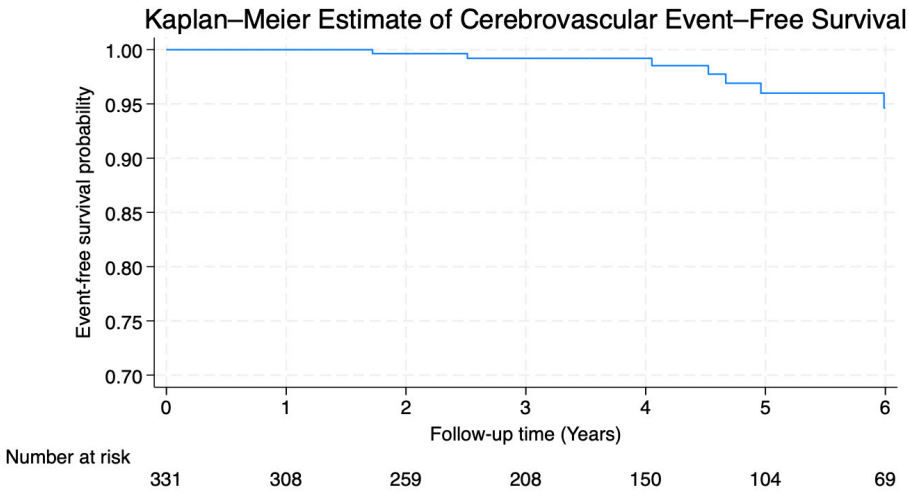


Figure 1: A total of 10 cerebrovascular events including 5 strokes and 5 TIA. Y-axis truncated for clarity.

O103

Patients' perceptions of routine depression and anxiety screening during adult congenital heart disease follow-up visits: A mixed-methods evaluation

Sven Schmutz[1], Michelle Andreas[1], Mathias Possner[1], Fabienne Schwitz[1]

[1]*Inselspital, Bern University Hospital, Bern, Switzerland*

Introduction: Depression and anxiety are prevalent in adults with congenital heart disease (ACHD), adversely affecting quality of life and clinical outcomes. Although guidelines recommend routine questionnaire-based screening in outpatient care, patients' acceptance, perceived burden, and perceived value of such screening remain insufficiently understood. This study presents preliminary data on patients' perceptions of routine depression and anxiety screening during ACHD follow-up visits.

Material & Methods: In this cross-sectional mixed-methods evaluation, 55 ACHD patients completed routine screening for depression (Patient Health Questionnaire-9, PHQ9) and anxiety (Generalized Anxiety Disorder-7, GAD7) during regular outpatient visits. After the consultation, patients evaluated the screening using the Questionnaire Experience Questionnaire (QxQ; satisfaction and workload) and three single-item measures assessing perceived meaningfulness, discomfort, and burden (7-point Likert scale). Additionally, three semi-structured questions explored positive aspects, negative aspects, and suggestions for improvement. Quantitative analyses included descriptive statistics, Wilcoxon signed-rank tests comparing ratings against the scale midpoint, and Spearman correlations between screening perceptions and PHQ9/GAD7 scores. Qualitative data were analyzed using structured content analysis.

Results: Screening was perceived as highly meaningful (median 7, IQR 1) with minimal discomfort and burden (both median 1, IQR 0). QxQ results indicated high satisfaction (median 6, IQR 1) and low perceived workload (median 1, IQR 0.33). All ratings differed significantly from the scale midpoint (p <0.05). Screening perceptions were not associated with depression or anxiety symptom severity scores (p >0.05). Qualitative analyses highlighted appreciation for the structured approach and the integration of psychological aspects into cardiac care. Reported limitations included limited opportunities to contextualize questionnaire responses, with suggestions for brief optional elaboration.

Conclusion: Routine depression and anxiety screening during ACHD outpatient visits was perceived as meaningful, satisfactory, and low-burden. These findings support the integration of such screenings into ACHD care. Allowing optional elaboration on questionnaire responses may further enhance patient-centered implementation.

COI: No

BEST ABSTRACT – RHYTHM DISORDERS

O040

Contemporary trends and outcomes in European ICD recipients: A 15-year analysis

Andrea Papa[1], Salvatore Chianese[2], Sing-Chien Yap[3], Michael Kühne[1], Christian Sticherling[1], Felix Mahfoud[1], Dominic Amj Theuns[3], Beat Schär[1]

[1]Department of Cardiology, University Hospital Basel, Basel, Switzerland, [2]A. Cardarelli Hospital, Cardiology Division, Department of Advanced Biomedical Sciences, "Federico II" University, Naples, Italy, [3]Erasmus MC, Department of Cardiology, Cardiovascular Institute, Rotterdam, Netherlands

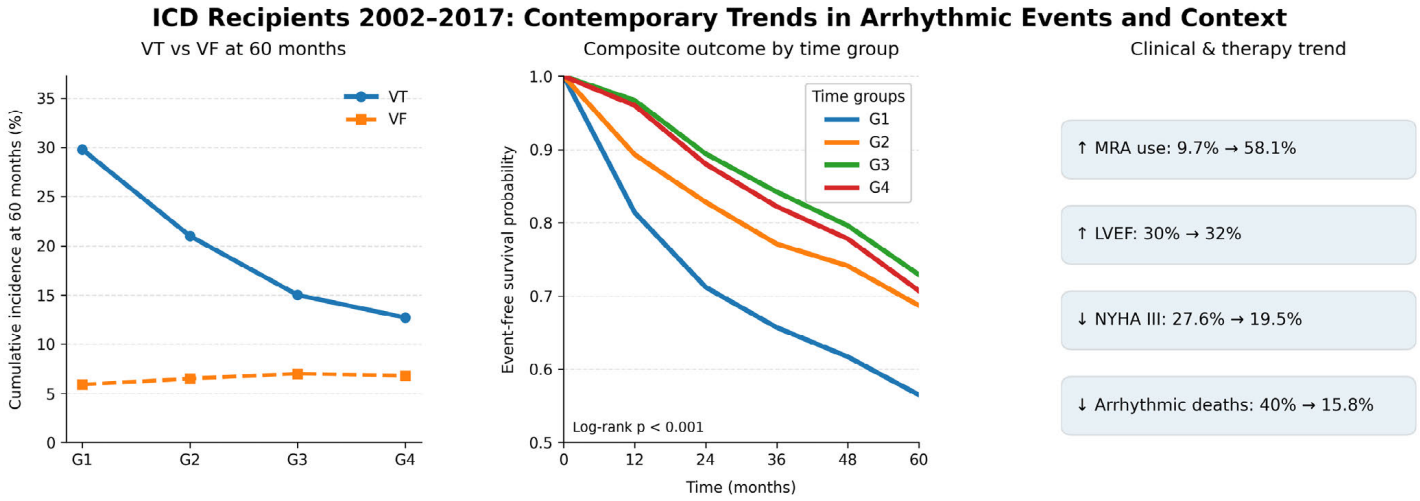
Introduction: Implantable cardioverter-defibrillators (ICDs) prevent sudden cardiac death, but wider adoption of guideline-directed medical therapy (GDMT) may have altered arrhythmic risk among ICD recipients. We examined 15-year trends in patient characteristics, pharmacotherapy, outcomes, and ICD utilization.

Material & Methods: We retrospectively analyzed consecutive ICD recipients (2002–2017) at two European centers, grouped into four time groups. Baseline demographics, comorbidities, device type, and medications were recorded. Primary outcomes were appropriate ICD therapies (antitachycardia pacing or shock), heart transplantation, and death, censored at 60 months. Kaplan–Meier estimates and multivariable logistic regression were applied.

Results: Among 2,184 patients (mean age 65.6 years, 13.7% female), 61.4% received ICDs for primary prevention. From the earliest to latest period, left-ventricular ejection fraction increased from 30% to 32% (p <0.001) and NYHA III decreased from 27.6% to 9.5% (p = 0.028). GDMT rose substantially (e.g., mineralocorticoid receptor antagonist use increased from 9.7% to 58.1%, p <0.001). Appropriate ICD therapies declined from 35.7% to 20.2% (p <0.001), driven by fewer sustained VT events from 29.8% to 12.7% (p <0.001), while VF remained low and unchanged (p = 0.21). The proportion of arrhythmic deaths fell from 40.0% to 15.8% (p = 0.0297), and 60-month event-free survival improved from 56.3% to 67.3% (log-rank p <0.001). In adjusted analyses, wider QRS duration independently predicted appropriate therapy (OR 1.05 per 10-ms increase, 95% CI 1.01–1.09, p = 0.015), whereas diabetes was associated with lower odds (OR 0.69, 95% CI 0.55–0.87, p = 0.002).

Conclusion: Over 15 years, ICD recipients exhibited fewer arrhythmic events alongside broader GDMT uptake, likely reflecting improved patient selection, pharmacotherapy and device programming.

COI: No



Abbreviations: ICD = implantable cardioverter-defibrillator; VT = ventricular tachycardia; VF = ventricular fibrillation; GDMT = guideline-directed medical therapy. Composite outcome = appropriate ICD therapy (shock/ATP), heart transplantation, or all-cause death. MRA = mineralocorticoid receptor antagonist; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association.

BEST ABSTRACT – CLINICAL CASES

O041

Acute myocarditis with severe left ventricular dysfunction caused by Francisella tularensis infection – A case report

Sarah Bissig[1], Matthias Paul[1], Bart De Boeck[1], Adrian Attinger-Toller[1], Matthias Bossard[1]

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Background: Acute myocarditis is mostly caused by viral infections. However, in patients with epidemiologic exposure and systemic symptoms, bacterial and zoonotic pathogens should be considered. *Francisella tularensis*, a gram-negative, aerobic coccobacillus is one of them, but only exceptionally associated with cardiac involvement.

Case summary: We report the case of a middle-aged farmer presenting with fever, dyspnea and mild chest discomfort. 2 days prior to admission he developed generalized limb pain, neck and headache and profound fatigue. Laboratory testing revealed markedly elevated cardiac biomarkers and inflammatory parameters (high-sensitivity troponin T up to 1,165ng/L, C-reactive protein up to 160mg/L, NT-proBNP 500ng/L). Transthoracic echocardiography demonstrated a rapid decline in left ventricular ejection fraction from initially mildly reduced to 25% within 12 hours. Cardiac magnetic resonance imaging (cMRI) showed typical features of acute myocarditis according to the revised Lake Louise criteria. Coronary angiography excluded obstructive coronary artery disease and an endomyocardial biopsy was nondiagnostic. Given the clinical constellation of a farmer with intermittent fever, a painless eschar on the leg and acute myocarditis, a zoonotic infection was suspected. Serology and PCR of the skin biopsy confirmed infection with *Francisella tularensis*. Guideline-directed heart failure therapy and antibiotic treatment with doxycycline were initiated, resulting in rapid clinical improvement and complete recovery of LV function within one week.

Discussion: In a patient with acute myocarditis and persisting systemic inflammatory symptoms, skin lesions or contact with animals, a thorough clinical assessment and evaluation for bacterial and zoonotic pathogens is crucial. For this purpose, rapid cMRI and extensive laboratory testing are mandatory, especially since the gold standard of endomyocardial biopsy is prone to sampling error and may be nondiagnostic.This case highlights *Francisella tularensis* as a rare but potentially life-threatening etiology. Early antimicrobial treatment and heart failure therapy are crucial for a favorable outcome

COI: No

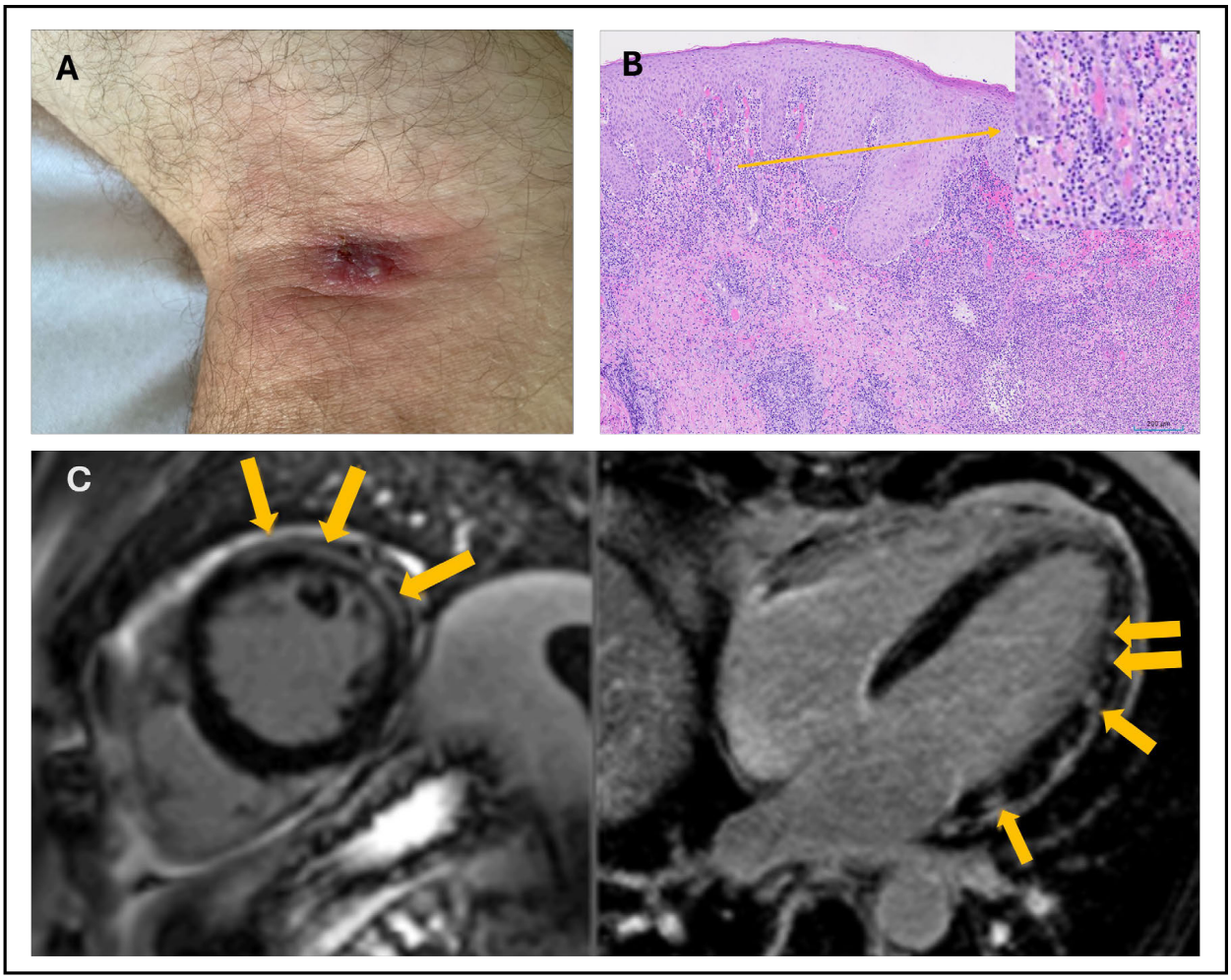

Figure: (A) Eschar in the left popliteal fossa. (B) Histology of the skin biopsy showing a dense inflammatory infiltrate, vasculitis and extensive erythrocyte extravasation. (Francisella sp DNA was detectable). Right corner enlarged image section. (C) cMRI with epicardially oriented, patchy to widely scattered, T1- and T2-positive lesions of the LV free wall and around the midventricular inferior hinge point (yellow arrows)

BEST ABSTRACT – BASIC SCIENCE CARDIOLOGY/CARDIAC SURGERY

O048

Adverse chromatin remodelling by SETD7 drives inflammation and diastolic dysfunction in obese heart failure with preserved ejection fraction

Shafeeq Mohammed[1], Sarah Costantino[1], Era Gorica[1], Natalia Atzemian[1], Frank Ruschitzka[1 2], Francesco Paneni[1 2]

[1]Centre for Translational and Experimental Cardiology, Department of Cardiology, University Hospital of Zurich, Zürich, Switzerland, [2]University Heart Center, Cardiology, University Hospital Zurich, Zürich, Switzerland

Introduction: Obese heart failure with preserved ejection fraction (obHFpEF) is expected to rise considerably over the next decades, and associates with high mortality. Yet, the underlying mechanisms are poorly understood, and breakthrough therapies remain to be developed. Epigenetic modifications have recently emerged as key players in the pathophysiology of cardiovascular disease. Post-translational modification of histones by chromatin modifying enzymes (CME) play a pivotal role in regulating gene transcription. Purpose: To investigate the role of chromatin remodeling in obHFpEF.

Material & Methods: Differential expression of chromatin-modifying enzymes was assessed in left ventricular myocardium from obHFpEF patients and controls (n = 10/group). Based on these data, cardiomyocyte-specific SETD7 knockout mice and littermate controls were subjected to a diet- and nitric oxide synthase inhibition–based model of obHFpEF, followed by in vivo functional assessment. SETD7-dependent chromatin remodeling was evaluated using epigenomic profiling. Mechanistic validation was performed in lipid-challenged neonatal rat cardiomyocytes with genetic and pharmacological SETD7 modulation, and translational relevance was examined using selective SETD7 inhibition in human cardiomyocytes from obHFpEF hearts.

Results: SETD7 was the top-ranking transcript in myocardial specimens from obHFpEF patients as compared to controls. ChIP-Seq in CMs showed a strong redistribution of the SETD7-dependent chromatin mark (H3k4me1) in HFpEF vs control CMs, with enrichment at inflammatory gene loci, including the NF-κB p65 promoter, and increased IL-1β and IL-6 expression (Fig. 1). In HFpEF mice, cardiomyocyte-specific deletion attenuated left ventricular hypertrophy, diastolic dysfunction, pulmonary congestion, and exercise intolerance (Fig. 2), while suppressing inflammatory signaling and apoptosis. In cultured CMs exposed to PA, SETD7 inhibition by (R)-PFI-2 prevented H3k4me1-driven p65 upregulation, whereas SETD7 overexpression mimicked HFpEF features. Of clinical relevance, (R)-PFI-2 reduced passive stiffness in skinned CMs isolated from obHFpEF patients.

Conclusion: SETD7 promotes obHFpEF by driving epigenomic reprogramming and pro-inflammatory gene activation.

COI: No

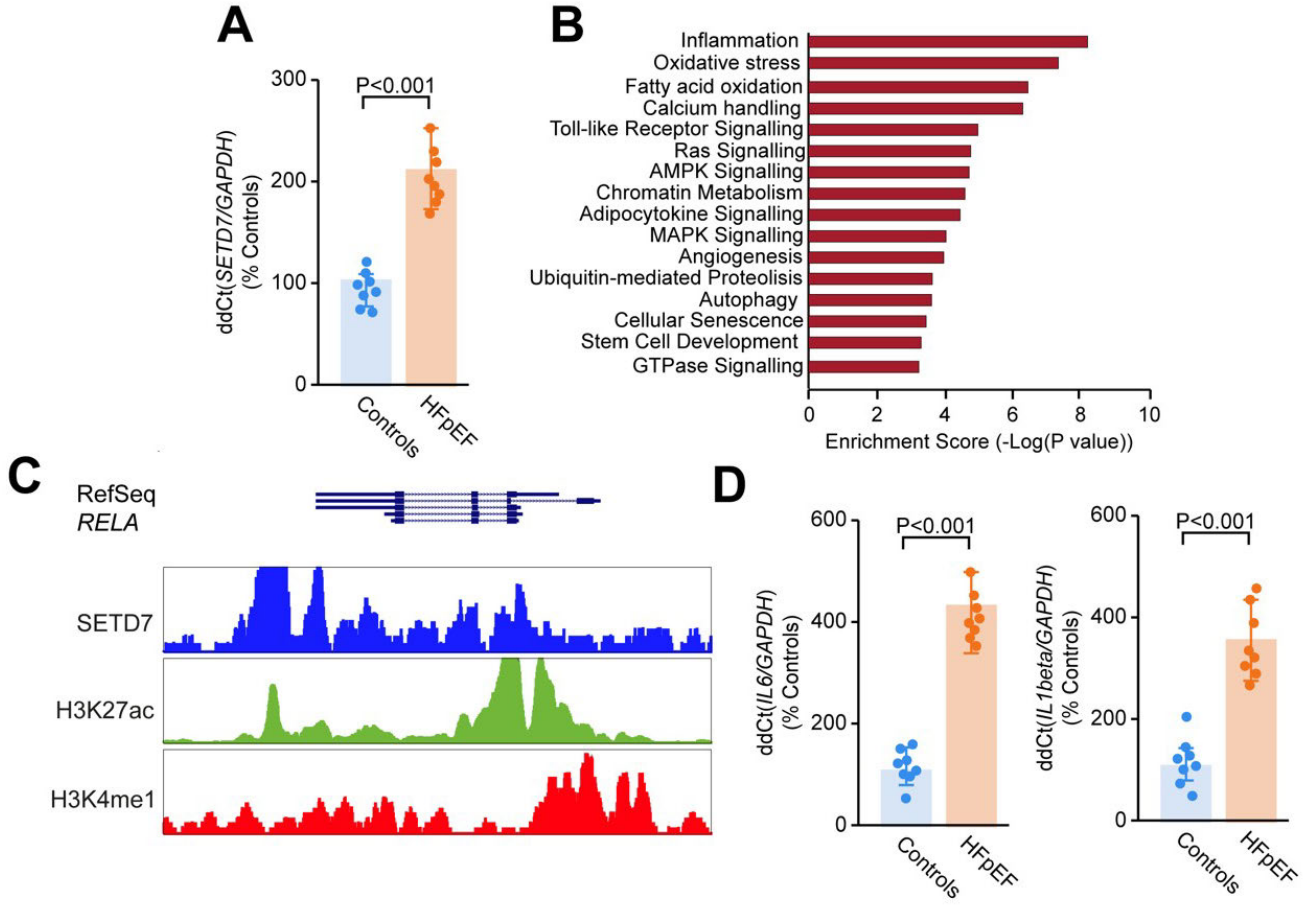

Figure 1. SETD7 orchestrates inflammatory transcriptional programs in cardiomyocytes. A) SETD7 gene upregulation is CMs from HFpEF mice. **B**) Gene ontology analysis of ChIP-seq dataset showing the transcriptional programs affected by SETD7 in human CMs. **C**) SETD7 occupancy and H3K4me1/H3K27ac enhancer remodelling at genes regulating inflammation (RELA NFkB p65); **D**) upregulation of NFkB genes in HFpEF CMs.

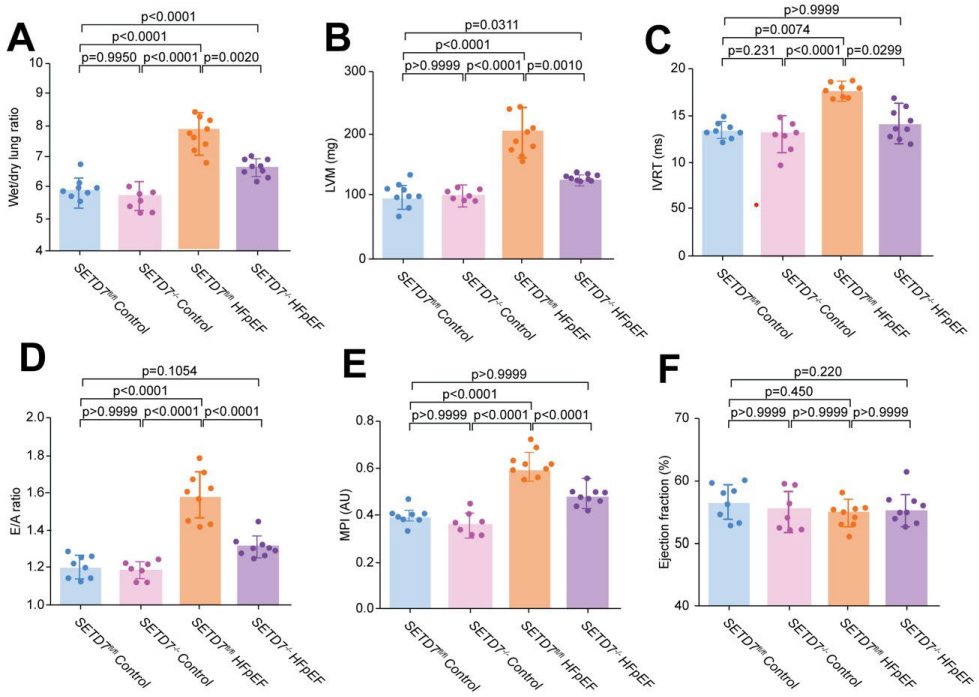

Figure 2. Cardiomyocyte-specific deletion of SETD7 protects against cardiometabolic HFpEF. **A**) Lung wet/dry ratio in HFpEF and control mice, with and without CM-specific deletion of SETD7. **B**) Left ventricular mass assessed by echocardiography in the 4 experimental groups. **C-D**) Diastolic function as assessed by IVRT and E/A ratio. **E**) Myocardial performance index. **F**) Systolic function assessed by ejection fraction. Results are presented as mean±SD and compared by one-way analysis of variance (ANOVA) followed by post-hoc analysis with Bonferroni correction. Adjusted p values (alpha/g) are shown. A p-value <0.05 was considered significant. LVM: Left ventricular mass, IVRT: Isovolumic relaxation time; MPI: Myocardial performance index.

BEST ABSTRACT – CARDIAC SURGERY

O058

Aortic Root Enlargement using the H technique: A Retrospective Study

Christoph Huber[1 2], Rares Eniu[1], Tomasz Nalecz[1], Daniela Dumitriu La Grange[1 2], Jalal Jolou[1 2], Nicolas Murith[1 2], Mustafa Cikirikcioglu[1 2]

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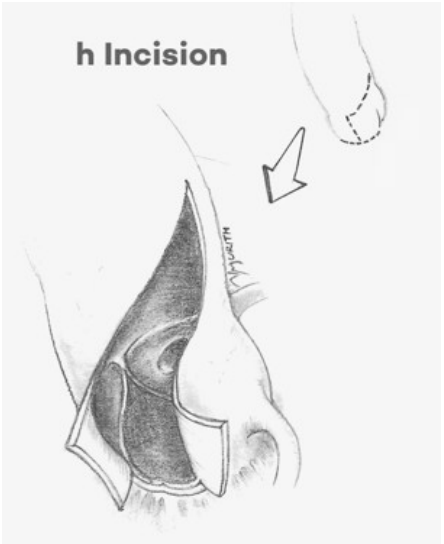
Introduction: SAVR is essential for treating aortic stenosis and regurgitation but is limited by patient-prosthesis mismatch, in patients with a small aortic annulus. Although several aortic root enlargement techniques exist to address this issue, many are technically demanding or associated with increased morbidity. We describe a novel aortic root enlargement method, the H-incision technique, and evaluate its safety, feasibility, and early hemodynamic performance in patients undergoing SAVR. The technique consists of a tailored J-shaped aortotomy combined with an H-shaped incision through the non-coronary sinus and annulus (Image 1), creating a controlled double trapdoor enlargement of the aortic root that is reconstructed with a pericardial patch to allow implantation of an upsized prosthesis while preserving root integrity.

Material & Methods: We conducted a retrospective cohort study including 18 patients who underwent SAVR with concomitant aortic root enlargement using the H-incision technique between January 2022 and October 2024. Pre- and postoperative transthoracic echocardiography was used to assess valve hemodynamics and left ventricular function. Endpoints included prosthesis size, occurrence of PPM, postoperative transvalvular gradients, indexed effective orifice area, complications, and left ventricular ejection fraction.

Results: Mean age was 67.5 ± 11.3 years, and 50% of patients were female. Aortic stenosis was present in 77.7% of cases. Prosthesis upsizing by 1-3 sizes was achieved in all patients, most commonly by two sizes. No patient developed postoperative PPM, with a mean iEOA of 0.99 cm²/m². Mean postoperative transvalvular gradient was 10.5 mmHg, and mean valve area was 1.9 cm². LVEF was preserved. Postoperative complications occurred in 44.4% of patients, mainly self limitting atrial fibrillation 27.7% and conduction disturbances, with no mortality, permanent pacemaker implantation, or paravalvular leak.

Conclusion: The H-incision technique is a simple and reproducible approach to aortic root enlargement that enables reliable valve upsizing and prevents PPM, with excellent early hemodynamic results and low morbidity.

COI: No



BEST ABSTRACT – CORONARY ARTERY DISEASE & ACUTE CARDIOVASCULAR CARE

O075

Training and Validation of a 12-lead ECG-Based Deep-Learning Model for Myocardial Infarction Subtypes

Tobias Zimmermann[1 2 3], Ivo Strebel[1 3], Pedro Lopez Ayala[1 3], Wayne Zeng[1], Sven Knecht[1], Luca Koechlin[1 3 4], Arnaud Champetier[1 3], Dominik Lemm[5], Paolo Bima[1 3 6], Karin Wildi[1 3], Michael Christ[7], Stefan Osswald[1], Volker Roth[8], Felix Mahfoud[1], Christian Müller[1 3], Jasper Boeddinghaus[1 3 9]

[1]Cardiovascular Research Institute Basel (CRIB) and Department of Cardiology, University Hospital Basel, University of Basel, Basel, Switzerland, [2]Intensive Care Unit, Department of Acute Medicine, University Hospital Basel, University of Basel, Basel, Switzerland, [3]GREAT network, Rome, Italy, [4]Clinic for Cardiac Surgery, University Hospital Zurich, Zurich, Switzerland, [5]Gradient Zero, Vienna, Austria, [6]Department of Medical Sciences, University Of Turin, Turin, Italy, [7]Emergency Department, Cantonal Hospital Lucerne, Switzerland, Lucerne, Switzerland, [8]Department of Mathematics and Informatics, University of Basel, Basel, Switzerland, [9]BHF Centre for Cardiovascular Science, University of Edinburgh, Edinburgh, Scotland, United Kingdom

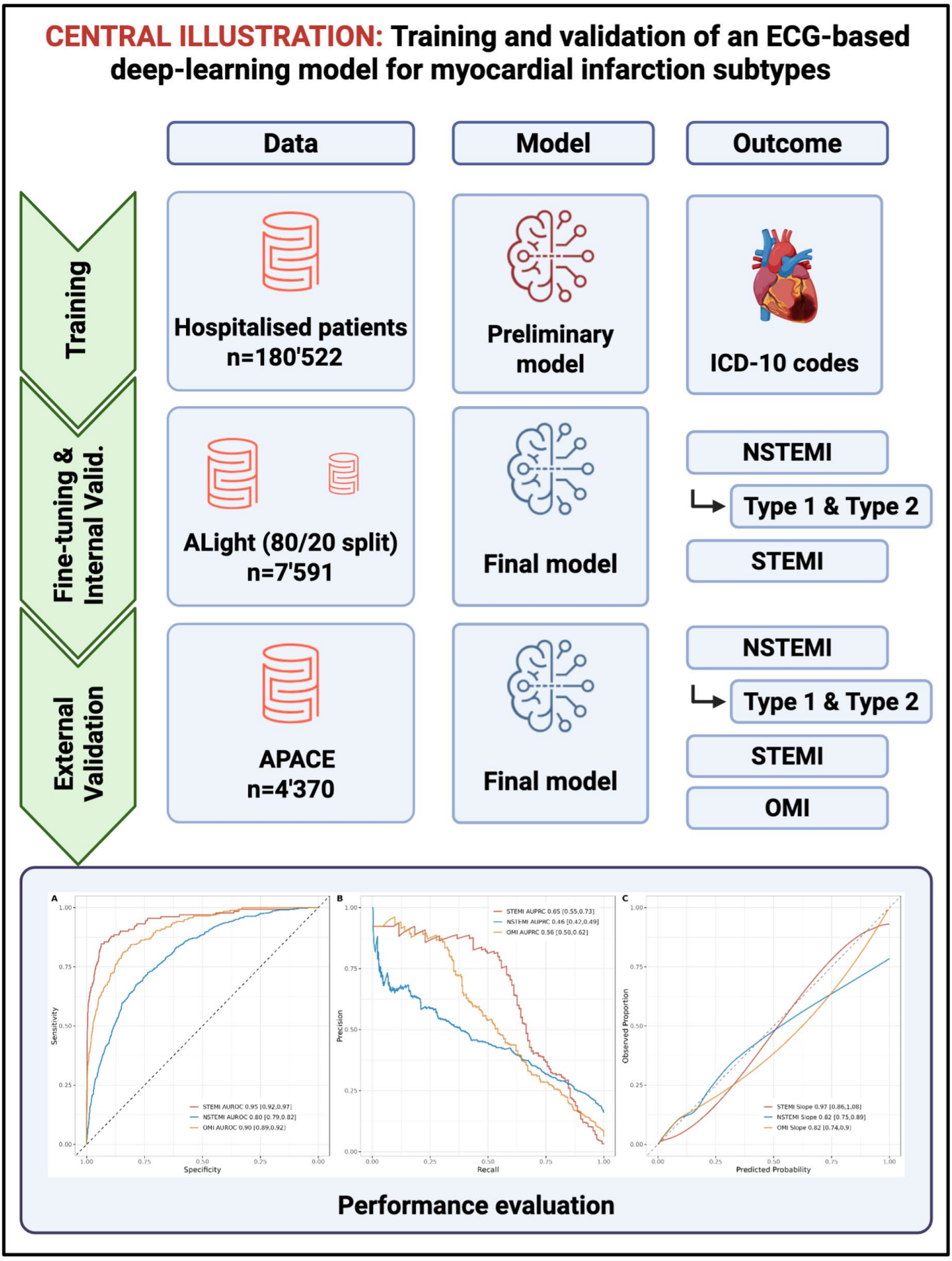
Introduction: The 12-lead electrocardiogram (ECG) is essential for diagnosing acute myocardial infarction (AMI), yet its interpretation largely depends on reader experience and can be unreliable. Consequently, delayed or missed diagnoses may occur, underscoring the need for more objective reproducible assessment methods. This study aims to derive and validate an artificial intelligence (AI)-powered 12-lead ECG-based deep learning model for AMI subtypes.

Material & Methods: We trained a convolutional neural network on digital 12-lead ECG data of 180,522 hospitalized patients. The model was fine-tuned and internally validated in a prospective diagnostic study of 7,591 patients with acute chest discomfort and externally validated in a prospective multicentre diagnostic study of 4,370 patients with acute chest discomfort, both with central diagnostic adjudication. The multi-label outcome was a diagnosis of non-ST-segment elevation MI (NSTEMI, type 1 and type 2), STEMI, and occlusion myocardial infarction (OMI). The model's performance and interpretability were evaluated overall and in pre-specified subgroups.

Results: External validation showed good diagnostic discrimination for NSTEMI (area under the receiver operating characteristic curve (AUROC) 0.80 [95%-CI 0.79-0.82]), with higher discrimination for NSTEMI type 1 (0.80 [0.78-0.82]) than type 2 (0.74 [0.78-0.71]). Model performance for NSTEMI was better in younger patients without previous heart disease. For STEMI, the model performed better than 12-lead ECG interpretation by physicians (0.95, [95%-CI 0.92-0.97]) versus 0.90 [0.86-0.94], p = 0.030). The model also achieved high diagnostic accuracy for OMI (0.90 [0.89-0.92]). Overall calibration was good for NSTEMI (slope 0.82 [0.78-0.95]), STEMI (slope 0.97 [0.86-1.08]), and OMI (slope 0.82 [0.74-0.90]).

Conclusion: This 12-lead ECG-based deep learning model showed good discrimination and calibration for different MI subtypes, and outperformed physician-based STEMI diagnosis.

COI: No



BEST ABSTRACT – VALVULAR HEART DISEASE

O076

Longitudinal Trends and Outcomes in Patients Undergoing Transcatheter Aortic Valve Implantation: a report from the SwissTAVI Registry

Thorald Stolte[1], Jakob Reichl[1], David Tueller[2], Felix Götzinger[1], Max Wagener[1], Stefan Stortecky[3], Raban Jeger[2], Stefan Toggweiler[4], Enrico Ferrari[5], Christoph Huber[6], Peter Wenaweser[7], Stephan Windecker[3], Felix Mahfoud[1], Stéphane Noble[6], Thomas Nestelberger[1]

[1]Department of Cardiology and Cardiovascular Research Institute Basel (CRIB), University Hospital Basel, University of Basel, Basel, Switzerland, [2]Department of Cardiology, Triemli Hospital Zurich, Zurich, Switzerland, Zürich, Switzerland, [3]Department of Cardiology, Inselspital, Bern University Hospital, University of Bern, Bern, Switzerland, [4]Department of Cardiology, Heart Center, Cantonal Hospital Lucerne, Lucerne, Switzerland, [5]Department of Cardiovascular Surgery, Cardiocentro Ticino Institute, Lugano, Switzerland, [6]Division of Cardiology and Cardiovascular Surgery, University Hospital, Geneva, Switzerland, [7]Heart Clinic Hirslanden, Hirslanden Clinic Zurich, Zürich, Switzerland

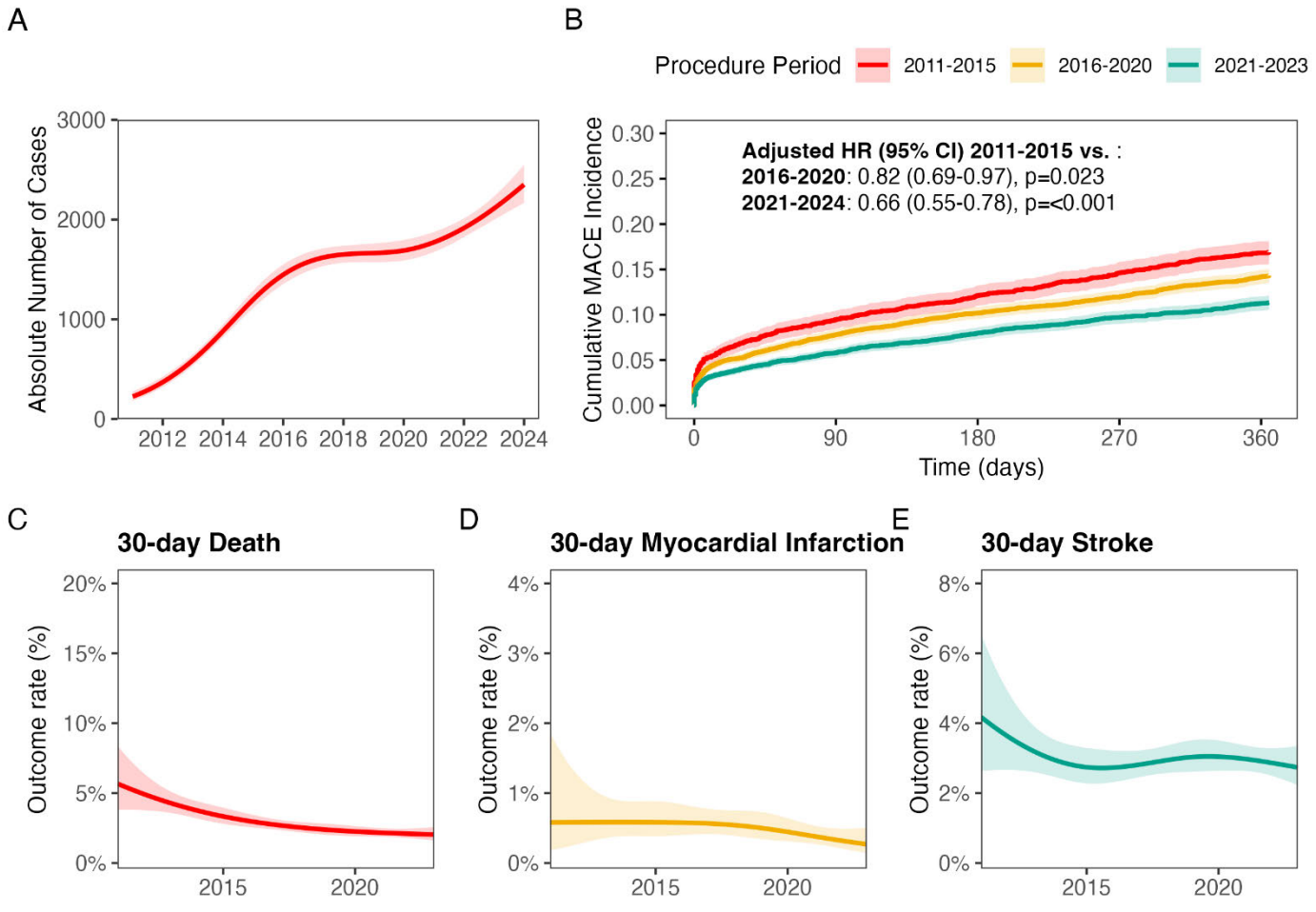
Introduction: The widening of indications for Transcatheter Aortic Valve Implantation (TAVI) across lower-risk and younger populations has raised concerns about a potential outcome dilution with increasing procedural volumes. Real-world evidence addressing this concern is limited. The objective of the present study was to assess longitudinal trends and changes in patient characteristics and procedural outcomes in a large nationwide TAVI cohort.

Material & Methods: The SwissTAVI Registry prospectively enrolled all patients undergoing TAVI in Switzerland. Baseline characteristics, procedural complications and outcomes were compared across three periods: 2011-2015, 2016-2020 and 2021-2024. The primary endpoint was major adverse cardiovascular events (MACE: all-cause death, non-fatal myocardial infarction, and non-fatal stroke) at one year. Secondary endpoint was 30-day MACE.

Results: Among 19452 patients, TAVI volume increased 11-fold with 27% yearly growth. Patient age (82 years) and comorbidities remained stable, while female representation increased (51% to 58%) and median STS score decreased (4.5% to 3.3%). Despite this expansion to lower risk, 1-year MACE declined significantly from 16% to 14% to 9.1% across periods (adjusted HR 0.69, 95%CI 0.62-0.78, p <0.001). Similarly, 30-day MACE decreased from 6.9% to 5.2% to 4.0% (adjusted HR 0.66, 95%CI 0.55-0.78, p <0.001). Procedural mortality declined from 2.9% to 1.2%, periprocedural myocardial infarction from 0.4% to 0.2% and stroke from 3.2% to 2.0% (all p <0.001) (*Figure 1*).

Conclusion: Risk-adjusted TAVI outcomes improved continuously despite an 11-fold volume increase, with improvements exceeding those expected from treating lower-risk patients alone. These findings contradict STS/ACC registry data showing outcome stagnation and support the volume-outcome hypothesis, demonstrating that expansion through high-volume centers maintains rather than dilutes procedural quality.

COI: No



BEST ABSTRACT – PREVENTION, REHABILITATION & SPORTS CARDIOLOGY

O089

Cardio-Kidney-Metabolic Drivers of Residual Risk After Acute Coronary Syndromes: The SMART-ACS Model

Yifan Wang[1], Marianne Zeller[2], Deepak Bhatt[3], Maud Maza[4], Giovanni G. Camici[1], Yves Cottin[2], Frédéric Chague[2], Thomas F. Lüscher[5], Simon Kraler[6]

[1]Center for Molecular Cardiology, University of Zurich, Wagistrasse 12, 8952, Schlieren, Switzerland, [2]Department of Cardiology, CHU Dijon Bourgogne, Physiolopathologie et Epidémiologie Cérébro-Cardiovasculaire (PEC2), EA 7460, Univ Bourgogne Europe, Dijon, France, [3]Mount Sinai Fuster Heart Hospital, Icahn School of Medicine at Mount Sinai, New York, United States, [4]Department of Cardiology, CHU Dijon Bourgogne, Dijon, France, [5]Center for Molecular Cardiology, University of Zurich, Wagistrasse 12, 8952 Schlieren, Switzerland, Royal Brompton and Harefield Hospitals and National Heart and Lung Institute, Imperial College, London, United Kingdom, [6]Center for Molecular Cardiology, University of Zurich, Wagistrasse 12, 8952 Schlieren, Switzerland, Department of Cardiology, Swiss Heart Center, Inselspital, Bern, Switzerland

Introduction: Residual cardiovascular risk reflects a complex interplay of lipid, low-grade inflammatory, and cardio-kidney-metabolic (CKM) pathways and remains substantial in patients with acute coronary syndromes (ACS) despite intensive low-density lipoprotein cholesterol (LDL-C)-lowering therapy. A more holistic characterization of ACS patient phenotypes is therefore needed to enable more targeted risk stratification and intervention. Accordingly, this study aimed to identify distinct ACS phenotypes using machine-learning approaches and to quantify their individual contributions to residual cardiovascular risk.

Material & Methods: We analysed 4,787 Swiss statin-treated acute coronary syndrome (ACS) patients from the Special Programme University Medicine Acute Coronary Syndrome (SPUM-ACS) study and 15,283 French statin-treated ACS patients from the obseRvatoire des Infarctus de Côte-d'Or (RICO) study. By using the Davies–Bouldin index and silhouette coefficient, unsupervised ML identified four patient clusters (k = 4) across lipid, inflammatory, and CKM domains. One-year major adverse cardiovascular events (MACE) were compared across phenotypes using Kaplan–Meier and multivariable Cox analyses.

Results: In SPUM-ACS, four patient clusters were identified by unsupervised ML, namely HDL-, LDL-, HbA1c-, and CRP–creatinine–dominant patient phenotypes (Fig. 1A). In RICO, similar clusters were identified (Fig. 1B). Risk of 1-year MACE differed significantly across clusters (log-rank p <0.001) in both Swiss and French patients (Fig. 1C-D), manifested as a 2.45-(adjusted hazard ratio [aHR], 2.45, 95% CI 1.64–3.66) and 2.40-fold (aHR, 2.40, 95% CI 1.60–3.59) higher MACE risk for HbA1c- and CRP-creatinine-dominant phenotypes, respectively, while LDL- (aHR, 1.07, 95% CI 0.68–1.69) and HDL-dominant (reference) phenotypes showing the best outcomes among statin-treated patients (Fig. 1E-F). Expectedly, the proportion of patients exceeding guideline-recommended risk thresholds differed significantly across clusters, irrespectively whether Swiss or French patients were analyzed (Fig. 2A-B).

Conclusion: ML-derived unsupervised clustering identifies reproducible CKM phenotypes with distinct risk profiles, providing a foundation for phenotype-guided secondary prevention strategies in the contemporary era.

COI: No

Fig. 1

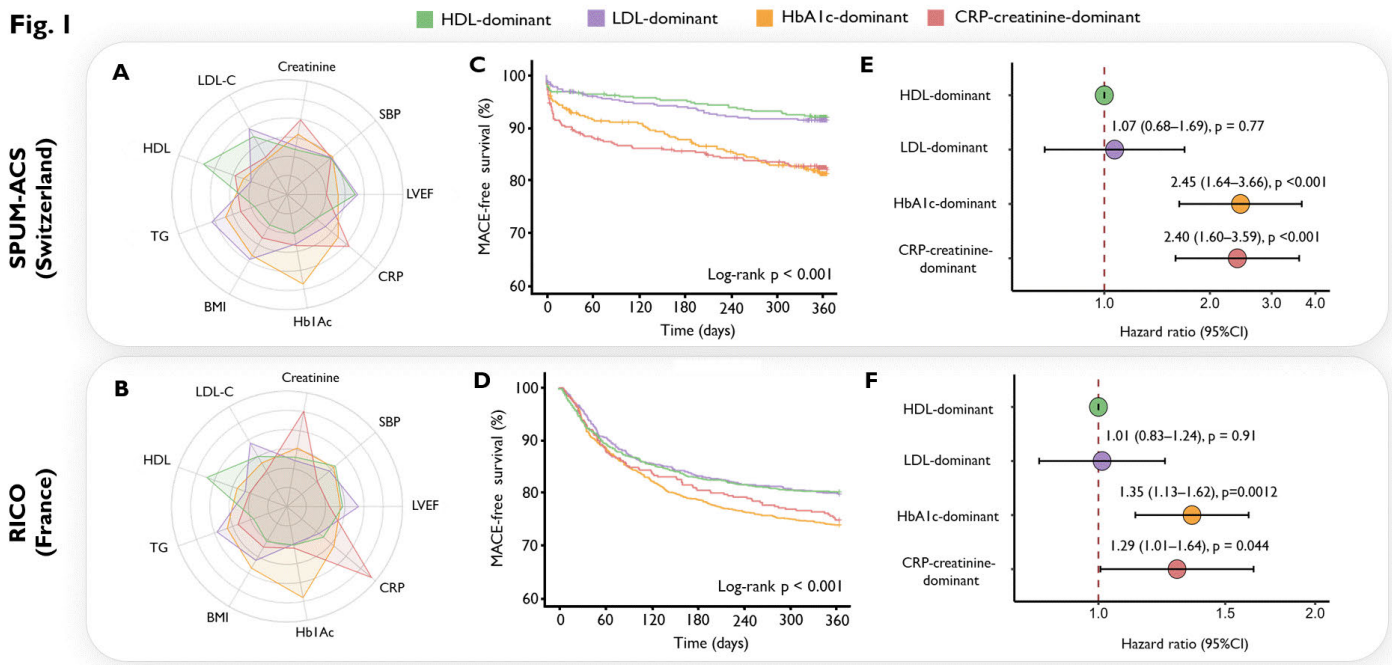


Figure 1. Clustering-derived cardiometabolic phenotypes and 1-year MACE risk. Radar plots display standardized mean values (z-scores) of nine clinical variables across four spectral clustering–derived phenotypes in the **(A)** SPUM-ACS and **(B)** RICO cohorts. Each phenotype shows a distinct clinical profile based on lipid, metabolic, inflammatory, renal, and cardiac markers. Kaplan–Meier curves compare 1-year MACE-free survival across phenotypes in **(C)** SPUM-ACS and **(D)** RICO; differences between groups were assessed using the log-rank test. Cox proportional hazards models estimate the 1-year risk of MACE for each phenotype in **(E)** SPUM-ACS and **(F)** RICO, using the HDL-dominant group as the reference category. **SBP**=systolic blood pressure; **LVEF**=left ventricular ejection fraction; **CRP**=C-reactive protein; **HbA1c**=glycated hemoglobin; **BMI**=body mass index; **TG**=triglyceride; **HDL**=high-density lipoprotein cholesterol; **LDL-C**=low-density lipoprotein cholesterol.

Fig. 2

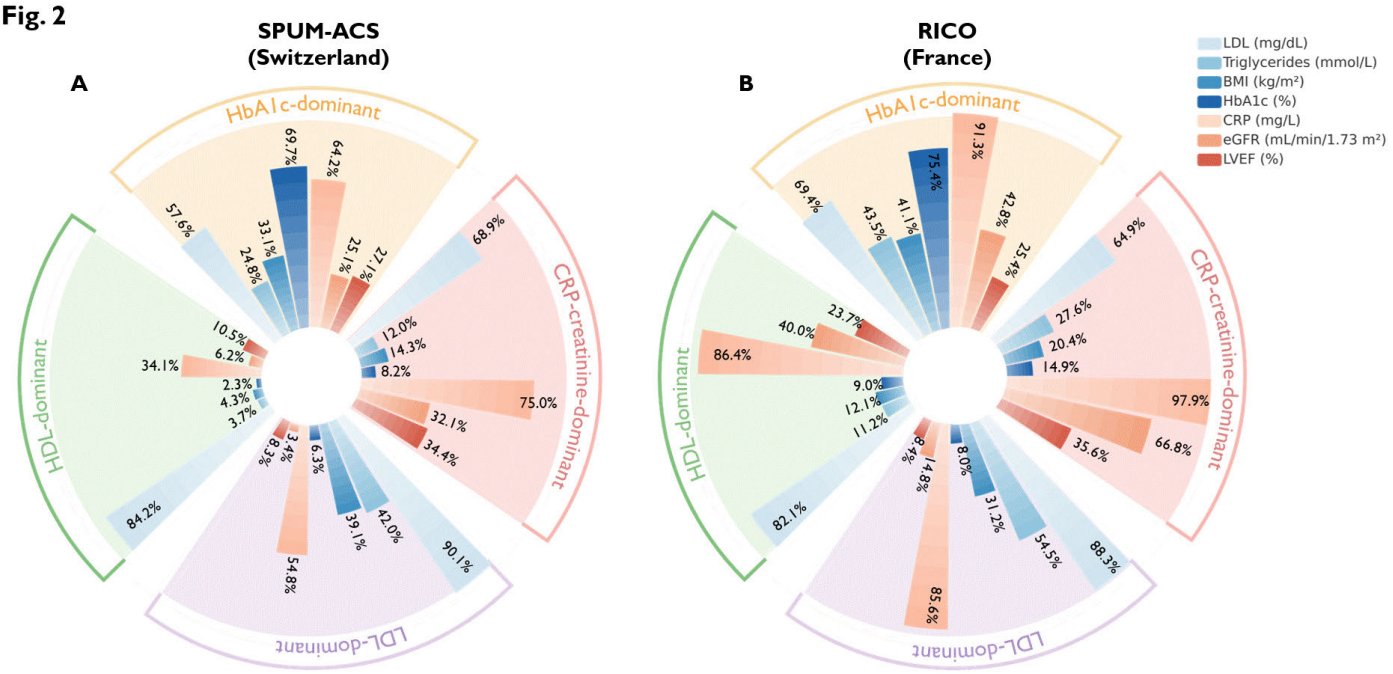


Figure 2. The proportion of individuals within each phenotype exceeded predefined clinical cut-offs. Circular bar plots show the proportion of individuals within each phenotype exceeding predefined clinical thresholds in **(A)** SPUM-ACS and **(B)** RICO. Thresholds were LDL-C≥ 1.8 mmol/L (70 mg/dL), Triglyceride≥ 1.7 mmol/L (150 mg/dL), BMI≥ 30 kg/m², HbA1c≥ 6.5%, CRP≥ 2 mg/L, eGFR < 60 mL/min/1.73 m², and LVEF≤ 40%. Bars represent the percentage of individuals per phenotype above each threshold.

BEST ABSTRACT – ALLIED HEALTH PROFESSIONAL

O097

Building Recovery Together: The ERAS Nurse–Patient Partnership through cardiac surgery journey

Estelle Brahier[1], Nathalie Parietti[1], Tamila Abdurashidova[1], Zied Ltaief[1], Valentina Rancatti[1], Ziyad Gunga[1], Anna Nowacka[1], Alexandra Othenin-Girard[1], Karima Alouazen[1], Florine Valliet[1], Matthias Kirsch[1]

[1]*Centre Hospitalier Universitaire Vaudois, Lausanne, Switzerland*

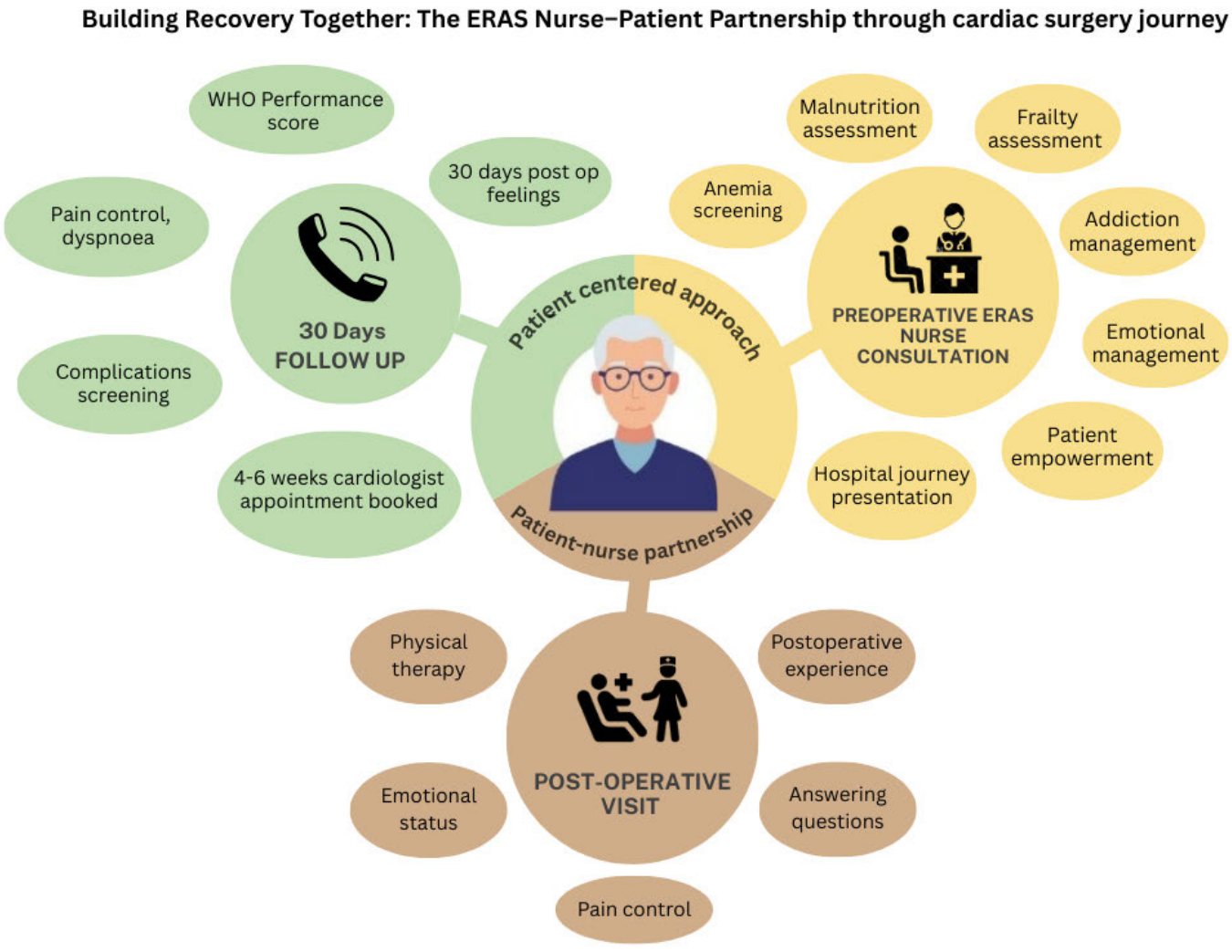
Introduction: Preoperative nurse-led consultation is a cornerstone of Enhanced Recovery After Surgery (ERAS) program. It empowers patients to actively participate in their care through education, enables early identification of perioperative risks, and facilitates targeted interventions to optimize surgical readiness. More than two years ERAS implementation, we report updated outcomes of protocol application.

Material & Methods: All elective cardiac surgery patients with cardiopulmonary bypass between May 2023 and December 2025 were included. The content of the ERAS consultation and nursing follow-up throughout the patient's journey are explained in the figure below.

Results: Of 495 patients included (median age 62.4 years : 25% female), 91.1% received structured education before admission. Nutritional screening was performed in 90.5% and 31% required and received nutritional supplementation. Ferropenic anemia was screened in 97% preoperatively, with ambulatory intravenous iron administered to 5.9%. The frailty score was screened in 92.5% patients : 10.1% had a frailty score ≥4, 7.5% patients required a physiotherapist consultation and 3.8% needed physical prehabilitation. Among patients, 24.4% were active smokers, 9.9% were regular alcohol consumers. Both received targeted cessation counseling. At the 30-day post-operative follow-up, 86.8% patients responded to our calls.

Conclusion: More than two years of program application, the nurse-led preoperative consultation remains a key element of our cardiac ERAS protocol and ensure high adherence to evidence-based interventions. This model promotes a patient-centred approach through therapeutic education provided during consultations and multidisciplinary coordination facilitated by the ERAS nurse. Throughout the patient's surgical journey, the patient-nurse partnership is key to providing comprehensive support and enhancing recovery after surgery. These results support broader adoption of structured nurse-led pre- and post-operative pathways in cardiac surgical ERAS programs and highlight it importance.

COI: No



BEST ABSTRACT – CONGENITAL / PEDIATRIC HEART DISEASE

O098

Detection and quantification of left-to-right shunts by magnetic resonance contrast bolus time intensity curves

Stephanie Brunner[1], Lucca Loretz[1], Stefan Toggweiler[1], Bart De Boeck[1]

[1]*Luzerner Kantonsspital, Luzern, Switzerland*

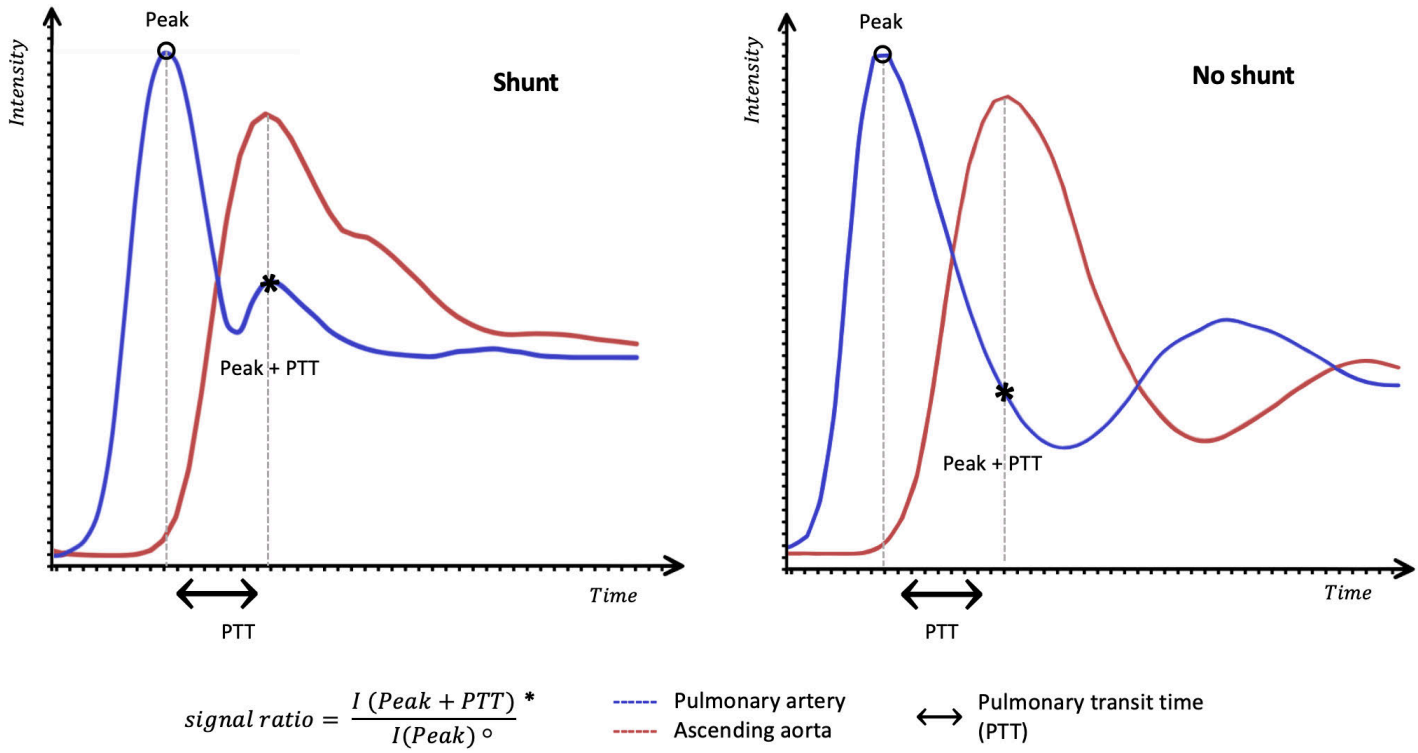
Introduction: Cardiac left-to-right shunts can have significant clinical impact. Cardiac magnetic resonance (CMR), the current non-invasive standard for detection and quantification, has inherent limitations, in particular if a shunt is not anticipated. We adapted historical invasive indicator-dilution principles to develop a novel CMR-based approach and investigated the potential of this novel method for rapid shunt screening (detection), as well as shunt localization and quantification.

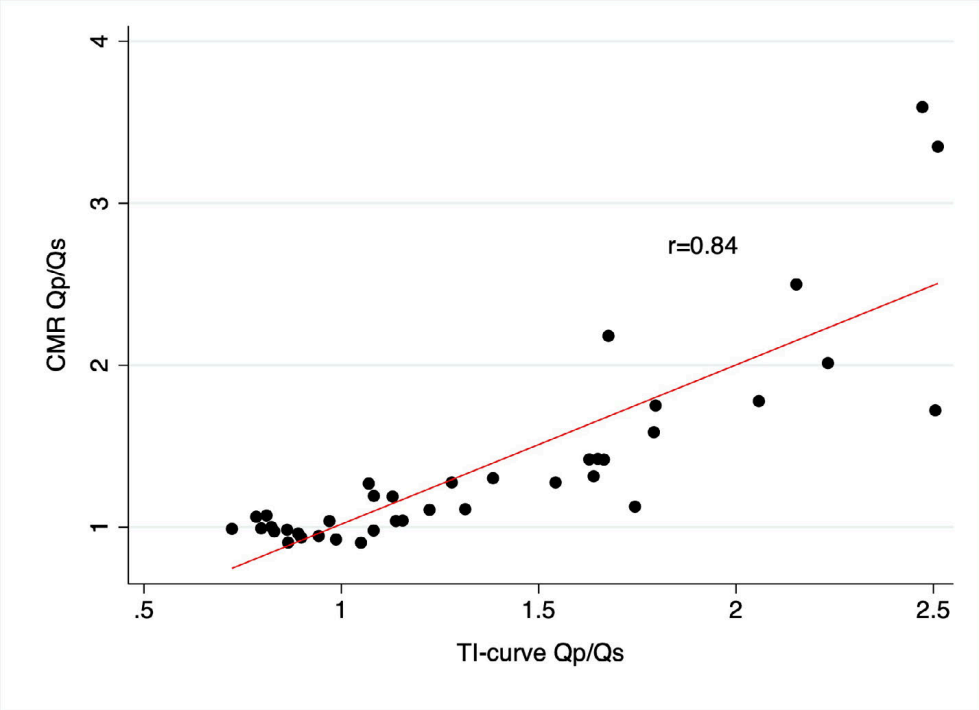
Material & Methods: This prospective study included 39 patients (22 with confirmed left-to-right shunt, 17 without) undergoing clinically indicated CMR. During contrast bolus passage, signal intensity was sampled in right- and left-sided arteries and cardiac chambers to generate time-intensity (TI) curves for shunt assessment as follows: 1) Shunt detection: identification of a premature recirculation sign (“notch”) during signal dilution occurring at [peak + pulmonary transit time (PTT)], Figure 1. 2) Shunt localization: most proximal anatomical site with "notch" in TI-curve. 3) Shunt size: derived from the signal ratio at (Peak + PTT) / (Peak), and compared with Qp/Qs obtained by CMR flow measurements as reference standard.

Results: Shunt detection by the TI-curve method was correct in 21/22 patients (sensitivity 95%), it’s exclusion in 16/17 patients (specificity 94%). No shunt with Qp/Qs >1.2 went undetected. Shunt localization was accurate in 90% of cases (17/19). Correlation between Qp/Qs determined by CMR and approximated Qp/Qs by the TI-curve method was good (r = 0.84).

Conclusion: Our novel CMR-based methodology based on TI-curve analysis demonstrated satisfactory accuracy for screening (detection), localization, and quantification of cardiac shunts compared with phase-contrast CMR as the reference standard, and can easily be implemented in most routine clinical CMR protocols.

COI: No





BEST ABSTRACT – HEART FAILURE

O104

Tricuspid regurgitation and outcomes in arrhythmogenic right ventricular cardiomyopathy: role of tricuspid annulus dilatation.

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Introduction: Tricuspid regurgitation (TR) is associated with adverse outcomes. Although right ventricular remodelling is common in arrhythmogenic right ventricular cardiomyopathy (ARVC), the role of TR remains hardly defined. This study aimed to determine its occurrence, mechanisms, and prognostic relevance in ARVC.

Material & Methods: In this international cohort study the phenotype of definite ARVC patients was characterized by echocardiography, sustained ventricular arrhythmia (VA) and death were considered as outcomes, and Cox or logistic regression models were used for analysis.

Results: The study included 514 patients (mean age 46 years, 45% women), with 58% showing none/trivial, 29% mild, and 13% moderate/severe TR. TR mostly (89%) exhibited ventricular-secondary origin. Tricuspid annulus dilatation was an independent determinant of TR (OR 2.22, 95% CI: 1.83–2.75). The median follow-up was 5.1 years. Overall, 146 VA events and 42 deaths were observed. TR severity and tricuspid annulus dilatation were independently associated with increased risk of either outcome (severe TR HR for VA 3.15, 95% CI 1.87–5.32; HR for death 21.81, 95% CI 9.67–49.23). Primary prevention patients (i.e. without prior VA) with moderate/severe TR or tricuspid annulus dilatation had VA-free survival similar to secondary prevention patients. Tricuspid annulus dilatation predicted VA or death independent of genotype, TR severity, and RV or left ventricular strain. Its discriminatory power for VA in primary prevention patients tended to exceed that of RV strain (time-dependent ROC at 2 years AUC 0.741, 95% CI 0.629–0.852; RV free-wall strain AUC 0.575, 95% CI 0.433–0.716).

Conclusion: TR occurs frequently in ARVC showing ventricular-secondary origin due to dilated tricuspid annulus. TR severity and annulus dilatation identify patients at higher risk irrespective of genotype, history of prior VA and right or left ventricular strain, with similar VA-free survival in primary and secondary prevention patients.

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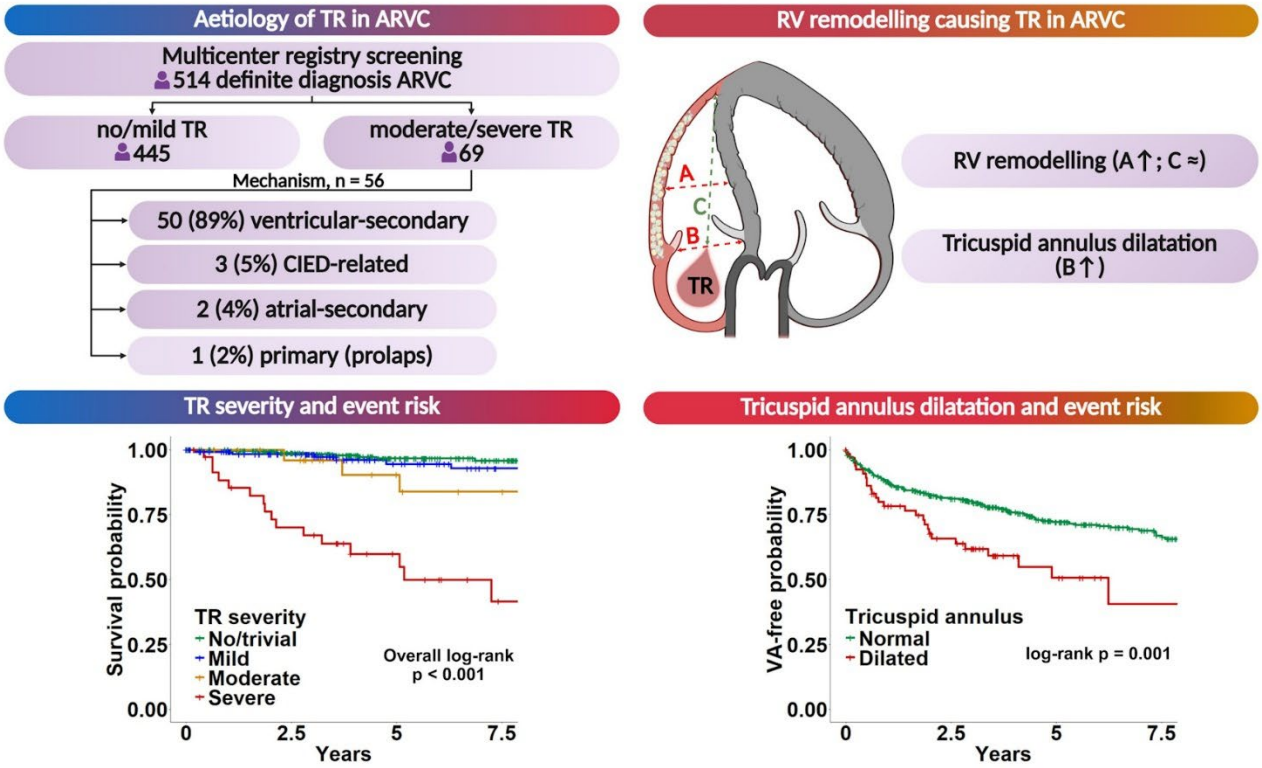
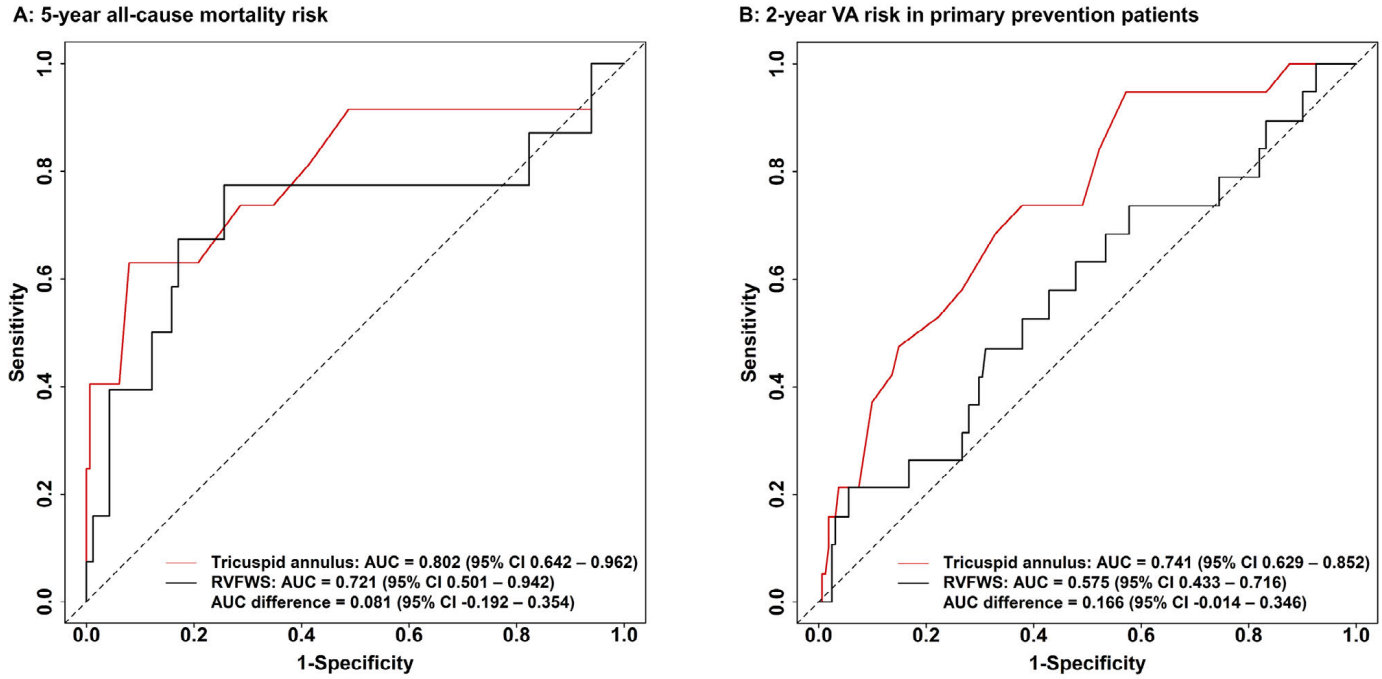


Figure 2: Time-dependent ROC curves



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