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Abstracts

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„Zurück in die Zukunft“

Salzburg,
25. bis 28. Mai 2022

Tagungspräsident
Univ.-Prof. Dr. Bernhard Metzler

Tagungssekretär
Univ.-Prof. Dr. Daniel Scherr

BEST ABSTRACTS

COVID-19 & Herz

Prevalence and duration of SARS-CoV-2 nucleocapsid antibodies in healthcare workers and an unselected all-comer patient population in Austria

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Introduction: Serosurveys are critical to determine prior exposure to SARS-CoV-2 and enable population-level surveillance. Only limited data on the change in SARS-CoV-2 exposure over time and direct comparisons between adult and pediatric patients and healthcare workers are available.

Methods: A longitudinal study enrolling healthcare professionals and concurrent serial cross-sectional studies of unselected all-comer patients were conducted at an Austrian academic tertiary center. Healthcare workers were tested at enrollment and after 1, 2, 3, 6, and 12 months. The cross-sectional studies in patients were conducted at 3 time points, which coincided with the end of the first, second, and third wave of SARS-CoV-2 in Austria (i. e., August 24–September 7, 2020; February 8–22, 2021, and November 9–23, 2021). Antibodies against the SARS-CoV-2 nucleocapsid antigen were measured using a sandwich electrochemiluminescence assay (Elecsys, Roche Diagnostics). We estimated the seroprevalence and 95 % confidence intervals (CI) according to the Wilson's score method.

Results: In total, 2735 and 9284 samples were measured in 812 healthcare workers (median age 40 years, 78 % female) and 9234 patients (median age 55 years, 51 % female), respectively. Over the entire study period, antibodies against SARS-CoV-2 were detected in 98 of 812 healthcare workers resulting in a seroprevalence of 12.1 % (95 % CI 10.0–14.5 %) which did not differ significantly ($P = 0.63$) from that of the all-comer patient population at the end of the study period (407/3184; 12.8 %, 95 % CI 11.7–14.0 %). The seroprevalence between healthcare workers and patients did not differ significantly at any time point (all P-values >0.10) and was 1.5- to 2-fold higher compared with the number of confirmed cases in Austria throughout the pandemic [Panel A and B]. Notably, there was no significant difference in the seroprevalence between pediatric ($n = 371$, median age 13 years, 48 % female) and adult patients at any of the tested time points (all P-values >0.09) [Panel C]. Among patients with a documented positive PCR test and detectable antibodies, the median time between the first documented positive PCR test and positive antibody test was 146 days (25th–75th percentiles 67–298 days).

Conclusion: Throughout the pandemic, healthcare staff and an all-comer patient population at an academic tertiary facility had similar exposure to SARS-CoV-2. There was no difference in the seroprevalence between pediatric and adult patients over the entire study period. These findings emphasize the need of research focusing on the prevention and mitigation of long-term complications of SARS-CoV-2 that can be associated with severe cardiovascular sequelae. (Funded by the Austrian Science Funds; ClinicalTrials.gov number: NCT04407429)

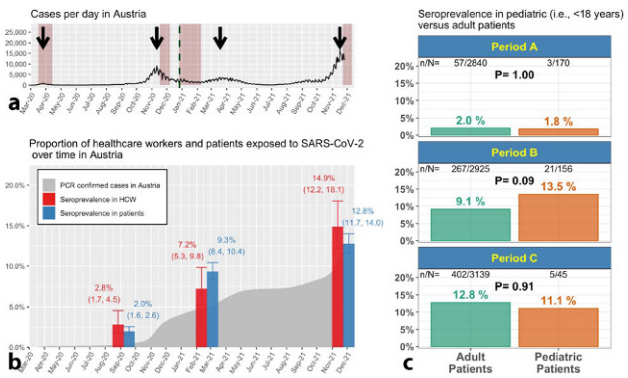

Fig. 1 Prevalence and duration of SARS-CoV-2 nucleocapsid antibodies in healthcare workers and an unselected all-comer patient population in Austria

Cardiovascular complications of long-COVID syndrome

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Introduction: Despite the overwhelmingly mild course of COVID-19 in the majority of the general population, a considerable portion of patients develops lasting symptoms, regardless of the severity of the initial disease episode. Long-COVID syndrome is characterized by the persistence of symptoms for longer than 12 weeks following an acute COVID-19 infection. The aim of this prospective registry is to characterize the clinical presentation of long-COVID syndrome and its clinical course.

Methods: Patients with a confirmed COVID-19 infection and persisting symptoms for longer than 12 weeks who visited the cardiology outpatient clinic were included in this registry. Upon inclusion in the registry, the patients were assessed using standardized questionnaires pertaining to their medical history, past and current symptoms and their general quality of life. Routine laboratory parameters were measured. Patients were divided into two groups in accordance to their vaccinated status at the first presentation.

Results: Totally 197 patients with long-COVID syndrome were included, 110 of them have already received their vaccination at the first clinical presentation. The median age our cohort was 44.5 (IQR: 33.7–54.9) years and the majority of patients were female (70.1 %). More than 50 distinct symptoms were described in this cohort (Fig. 1a). Postural tachycardia was documented in 47 patients (23.9 %). Any abnormalities in ECG (prolonged QRS time as incomplete bundle branch block or AV block or atrial fibrillation or Lown grade ≥2 ventricular arrhythmias) were detected in 58 Patients (29.4 %). Cardiac magnetic resonance imaging was available for 71 patients: 37 (52.1 %) scans showed on or more pathological features. The most com-

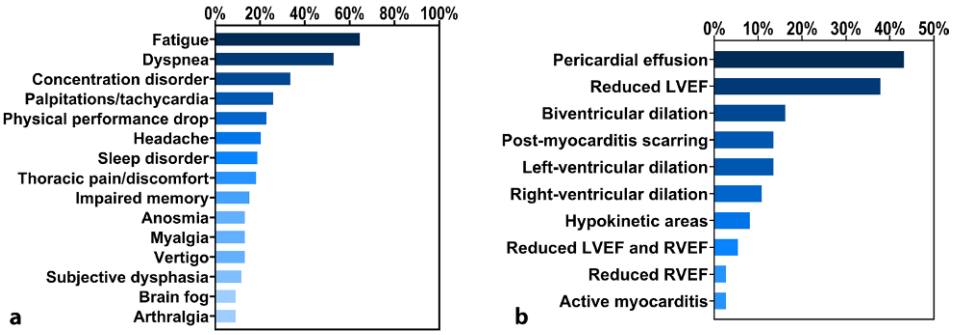

Fig. 1 Cardiovascular Complications of Long-COVID syndrome (**a**) Prevalence of symptoms and (**b**) primary patological cardiac magnetic resonance findings

monly observed changes were: pericardial effusion (43.2 %), a reduced left ventricular ejection fraction (37.8 %), dilation of both ventricles (16.2 %) and post-myocarditis scarring (13.5 %), as shown in Fig. 1b. Positive IgM of either EBV or CMV or Parvovirus or Herpes simplex virus was detected in 16.2 % of patients, suggesting reactivation of different RNA or DNA viruses. Long-COVID patients with vaccination had a significantly lower total IgM (90.1 ± 24.2 vs 197.0 ± 91.3 mg/dL), trend towards less IgG subtype 3 (27.7 ± 10.2 vs 33.9 ± 17.8 mg/dL, $p = 0.07$) and anti-cardiolipin antibody IgG (0.8 ± 0.6 vs 1.1 ± 0.8 mg/dL, $p = 0.098$). There was no difference between the patients with/without vaccination regarding the cardiac MRI abnormalities or clinical symptoms or viral IgM positivity.

Conclusion: Long-COVID is associated with high prevalence of subclinical cardiac abnormalities, which suggests subclinical viral myocarditis in patients not requiring hospitalization due to mild/moderate symptoms. These patients should be controlled clinically, as recent study [1] revealed substantial increase in cardiovascular burden post COVID-19 infection.

References

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Kardiovaskuläre Effekte einer Dexamethasontherapie bei kritisch kranken COVID-19 Patienten

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Einleitung: Die medizinische Versorgung intensivpflichtiger COVID-19 Patienten stellt weiterhin eine klinische Herausforderung dar. Als eine der ersten Therapieoptionen konnte dabei für Dexamethason eine Reduktion der Mortalität bei schwerer COVID-19 Pneumonie nachgewiesen werden. Ob eine Dexamethasontherapie im Kontext einer schweren COVID-19 Erkrankung auch einen Einfluss auf die myokardiale Schädigung sowie die Inzidenz an Thromboembolien hat, verbleibt jedoch bis dato ungeklärt.

Methoden: Insgesamt wurden 178 (129 Männer, 49 Frauen) intensivpflichtige, maschinell beatmete COVID-19 Patienten in 3 europäischen Zentren (Österreich und Deutschland) rekrutiert. Aus dem Studienkollektiv erhielten insgesamt 113 Patienten (63,5 %) eine Dexamethasontherapie (Behandlungsdauer median 10 Tage (IQR 9–10)). Die Kontrollgruppe setzt sich aus 65 Patienten (36,5 %) zusammen, welche keine Dexamethasontherapie erhielten. Patienten welche eine zusätzliche COVID-19 spezifische Therapie erhielten, wurden nicht in die Studie eingeschlossen.

Resultate: Dexamethason bewirkte eine signifikante Reduktion der maximalen Auslenkung der Entzündungsparameter (CRP max: median 20 ng/mL (IQR 12–28) vs. 22 ng/mL (IQR 14–37); $p = 0{,}043$). Ebenso kam es unter Dexamethason zu einer signifikanten Reduktion der maximalen Tropo-

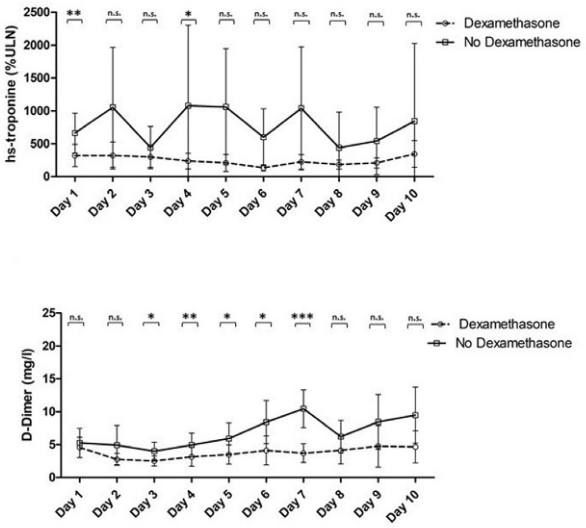

Abb. 1 | 2-7 Verlauf der Plasmalevel von hs-Troponin (%-ULN) und D-Dimer (mg/dl) – Dexamethason vs. kein Dexamethason

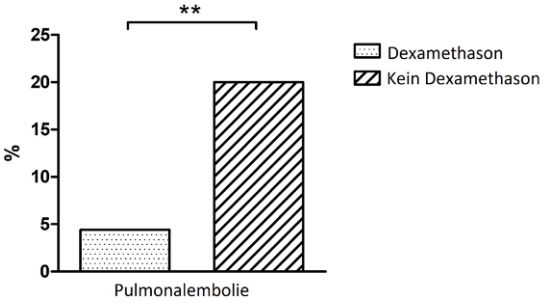

Abb. 2 | 2-7 Inzidenz Pulmonalembolien – Dexamethason vs. kein Dexamethason

ninlevel (Troponin max.: median: 231 % ULN (IQR 89–571) vs. 700 % ULN (IQR 164–2216); $p=0{,}001$) als auch der maximalen D-Dimer Werte (D-Dimer max: median: 2,16 mg/l (IQR 0,94–5,16) vs. 6,14 mg/l (IQR 1,78–16,48); $p=0{,}002$). Dies äußerte sich auch in einer signifikanten Reduktion der Pulmonalembolierate in der Dexamethasongruppe (4.4 % vs. 20.0 %; $p=0{,}001$). Der antithrombotische Effekt der Dexamethasontherapie war dabei auch bei Patienten unter therapeutischer Antikoagulation zu beobachten (6 % vs. 34.4 %; $p<0{,}001$). Es wurden keine signifikanten Unterschiede in den Baselinecharakteristika zwischen dem Dexamethasonkollektiv und der Kontrollgruppe beobachtet.

Schlussfolgerungen: Eine Dexamethasontherapie geht bei schwerer COVID-19 Pneumonie mit einer signifikanten Reduktion an myokardialer Schädigung einher. Ebenso konnte eine signifikante Reduktion an Pulmonalembolien im Dexamethasonkollektiv beobachtet werden. Unsere Ergebnisse unterstreichen damit die vorteilhaften Effekte einer Dexamethasontherapie, als auch den engen Zusammenhang inflammatorischer und prothrombotischer Effekte bei COVID-19.

BASIC SCIENCE

NETosis induced by extracellular vesicles is attenuated by natural IgM recognizing oxidation-specific epitopes

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Introduction: Formation of neutrophil extracellular traps (NETs) has emerged as an important contributor to thrombus formation in acute myocardial infarction (AMI). Many triggers of NET formation have been described, however, the mechanistic understanding of inducers and modulators of NETosis at the culprit site is pivotal for the development of new diagnostic and therapeutic approaches. Oxidations-specific epitopes (OSE), products of lipid peroxidation, accumulate in atherosclerotic plaques and can act as drivers of NET formation. Extracellular vesicles (EV) carrying OSE are elevated in AMI. These OSE-EV are recognized by natural IgM antibodies, which exert protective functions in cardiovascular disease by hampering the pro-inflammatory properties of OSE. We assumed that EV-induced NET formation can be inhibited by OSE-IgM and challenged our hypothesis in vitro and in vivo.

Methods: AMI patients ($n=51$) were recruited at first medical contact. Blood was collected from the culprit site during percutaneous coronary intervention and at several time points afterwards. Myocardial function was documented by cardiac magnetic resonance (CMR) at 72 h and 6 months. EV were isolated by ultracentrifugation and characterized by flow cytometry determining cell origin (leukocyte-, endothelial-, and platelet-derived) and presence of OSE. Isolated EV were used for neutrophil stimulation in vitro and in vivo using a murine injection model. NETs were visualized by immunofluorescence stainings for DNA, histone, citrullinated histone 3 (citH3) and MPO. Natural IgM recognizing OSE (OSE-IgM) and NET markers in murine and patient plasma were measured by ELISA.

Results: EVs of different cellular origins (CD45, CD41a, CD144) revealed a prominent absolute and relative increase of the OSE-carrying subset as recognized by the antibody LR04. Culprit site plasma contained more and proportionally higher levels OSE-EV derived from CD45+ cells than the intra-patient peripheral control. No difference was observed for EV of platelet origin, and interestingly, the endothelial-derived fraction of OSE-EV was decreased at the site of occlusion. NET markers were associated with OSE-EV in the circulation. Decreasing OSE-IgM during hospital stay indicated consumption of protective antibodies. EV isolated from AMI patient plasma were found to induce NETosis in neutrophils in vitro and after injection into mice in vivo, as determined by NET markers and fluorescence microscopy of histone citrullination in neutrophils. The malondialdehyde-specific LR04 IgM antibody, but not a control IgM, reduced the NETogenic effects of EV in both models. Consistently, higher circulating levels of EV and lower OSE-IgM were associated with a reduced ejection fraction in AMI patients at 72 h and six months.

Conclusion: EV from AMI patients induced NET formation in vitro and in vivo, and natural IgM recognizing malondialdehyde-epitopes on EV attenuated this effect. In summary, the balance between OSE-EV and OSE-IgM during AMI may represent a potential prognostic and therapeutic target with impact on heart function.

Post-translational modifications of extracellular matrix protein Biglycan regulate innate immunity and initiate calcific aortic valve disease

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Introduction: Calcified aortic valve disease (CAVD) is the third leading cause of cardiovascular related disease, constituting a major socioeconomic burden in the Western world. Non-invasive, pharmaceutical approaches to stop or slow its progression are urgently needed. Recently, we discovered that the innate immune receptor Toll-Like receptor 3 (TLR3) mediates the initiation of the disease. Our intention was to elucidate the underlying mechanism of TLR3-induced CAVD and test it as a pharmacological target to stop the disease.

Methods: Screening of possible TLR3 ligands was performed in vitro using reporter cells. Interactions were characterized via immunoprecipitation. Mice lacking Tlr3 and its newly identified ligand biglycan (BGN) were subjected to high-fat diet to induce CAVD. Selective digestions of BGN side chains using specific enzymes were performed. Patient samples and aged valvular interstitial cells (VICs) we analyzed for gene and protein expression via qPCR and western blot. Two clinical cohorts (GERA, $n=55{,}192$ with 3469 aortic stenosis cases; UK Biobank, $n=257{,}231$ with 2213 aortic stenosis cases) were examined for genetic variation at 12 genes implicated in the BGN/TLR3 signaling pathway to look for association with aortic stenosis in humans.

Results: We identified an novel endogenous ligand of TLR3, namely the highly decorated proteoglycan biglycan (BGN). Biglycan is a proteoglycan occurring ubiquitously in the extracellular matrix. Both Tlr3–/– and Bgn–/– were protected from

a CAVD phenotype, showing no signs of morphological valve thickening or hemodynamic signs of CAVD in transthoracic echocardiographies. We found that BGN-mediated activation of TLR3 reporter cells was abolished upon inhibition of endocytosis, consistent with BGN-induced TLR3 signaling via endosomes. Biological activity of BGN was primarily dependent on the glycosylation of its serine residues. While a direct interaction was found, the studies revealed that post-translational modifications (PTMs) of BGN by xylosyltranferase 1 (XYLT1) strongly modified its TLR3 activation potential. Levels of XYLT1 were increased in human samples of stenotic valves and aged VICs. Finally, we observed 294 variants which were nominally significant ($p \leq 0.05$) in two clinical cohorts of aortic stenosis. Notably, 14 variants in genes within the BGN/TLR3 pathway demonstrated strong associations ($p \leq 1 \times 10{-}3$) and/or two-fold or greater (up to 5.86-fold) odds of aortic stenosis. We also observed 15 BGN variants (11 independent signals) which were associated with aortic stenosis in the UK Biobank.

Conclusion: Our results uncover the XYLT1-BGN-TLR3 axis as a potential therapeutic target to (a) identify indivuduals at risk vor CAVD and (b) stop the progression of CAVD.

Autophagy causally contributes to the cardioprotective effects of NAD+

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Introduction: Heart failure with preserved ejection fraction (HFpEF) is associated with low cardiac nicotinamide adenine dinucleotide (NAD+), while oral supplementation of the NAD+ precursor, nicotinamide, improves preclinical HFpEF in rodents. However, NAD+ is a pleiotropic molecule and, thus, it remains elusive which cell-autonomous mechanisms are required for HFpEF therapy by NAD+. Because autophagy is an NAD+-dependent mechanism that is essential for maintaining cellular homeostasis, especially in long-lived cardiomyocytes, we set out to test whether cardiac autophagy is involved in NAD+ repletion therapy.

Methods: Here, we show that moderate elevation of NAD+ using a clinically feasible dose of nicotinamide (0.3 % w/v in the drinking water) effectively improved cardinal signs of HFpEF in ZSF1 obese rats, a model of metabolic syndrome-induced HFpEF. Specifically, nicotinamide reduced cardiac hypertrophy, diastolic dysfunction, and pulmonary congestion. These cardioprotective effects correlated with increased expression of several mRNA species involved in the autophagic-lysosomal pathway in the heart. Consistently, nicotinamide stimulated the autophagy marker LC3B, leading to an increase in its electrophoretic mobility (LC3B-II) and reduced the autophagic substrate p62, indicative of increased autophagic flux. To examine whether autophagy is causally required for the anti-HFpEF effects of nicotinamide, we used Atg5-deficient (Atg5–/–) mice, which lack constitutive autophagy specifically in cardiomyocytes. Atg5–/– mice and their control littermates (Atg5+/+) were subjected to a 'two-hit' HFpEF model using a high-fat diet (HFD) and the nitric oxide synthase inhibitor N-nitro-l-arginine methyl ester (L-NAME). After 5 weeks of HFD+L-NAME administration, both Atg5–/– and Atg5+/+ mice developed left ventricular diastolic dysfunction and remodeling, but preserved ejection fraction. Although nicotinamide significantly attenuated diastolic dysfunction and ventricular remodeling in HFD+L-NAME-fed Atg5+/+ mice, it failed to exert similar cardioprotective effects in Atg5–/– mice.

Results: Mechanistically, autophagy activation was not associated with changes in the acetylation levels of essential autophagy-related proteins, as evaluated by immunoblotting and confirmed in a comprehensive acetylome analysis. By contrast, the insulin/IGF-1 pathway, which inhibits autophagy, emerged amongst the top hits in an unbiased gene-set enrichment analysis of the cardiac transcriptome. Accordingly, nicotinamide reduced the expression of cardiac IGF-1 receptor, and diminished the activity of IGF-1 signaling, as indicated by reduced phosphorylation of the downstream effector protein kinase Akt at Thr308 and Ser473. Importantly, co-administration of IGF-1 to nicotinamide-fed mice attenuated the pro-autophagic action of nicotinamide in vivo, confirming that IGF-1 inhibition is mechanistically involved.

Conclusion: These results indicate that activation of cardiac autophagy is essential for HFpEF therapy by nicotinamide.

CLINICAL SCIENCE

Association between inflammation and left ventricular thrombus formation following ST-elevation myocardial infarction

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Introduction: Current evidence suggests a link between the inflammatory state and left ventricular thrombus (LVT) formation following ST-elevation myocardial infarction (STEMI). However, a comprehensive study investigating the association between inflammatory biomarkers and LVT diagnosed by cardiac magnetic resonance (CMR) is lacking. Therefore, the present study aimed to investigate the association of biochemical markers of inflammation with LVT as assessed by CMR imaging among patients with STEMI.

Methods: We studied 309 patients with acute STEMI treated with primary percutaneous coronary intervention (pPCI) from the prospective MARINA-STEMI cohort study. Concentrations of high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), white blood cell count (WBCc), fibrinogen and D-dimer were measured two days after STEMI. Infarct characteristics and presence of LVT were assessed with the use of contrast-enhanced CMR at a median of 4 (interquartile range [IQR] 3–5) days after pPCI.

Results: In total, 309 STEMI patients (18 % female) with a median age of 57 (IQR 52–65) years were included. An LVT was observed in 8 % ($n = 24$) of the overall cohort and in 15 % of patients with an anterior STEMI. Hs-CRP (OR: 2.16, 95 % CI:1.54–3.02, $p < 0.001$), IL-6 (OR: 2.38, 95 % CI:1.48–3.81, $p < 0.001$) and fibrinogen levels (OR: 2.05, 95 % CI:1.40–3.00, $p < 0.001$) were significantly associated with presence of LVT.

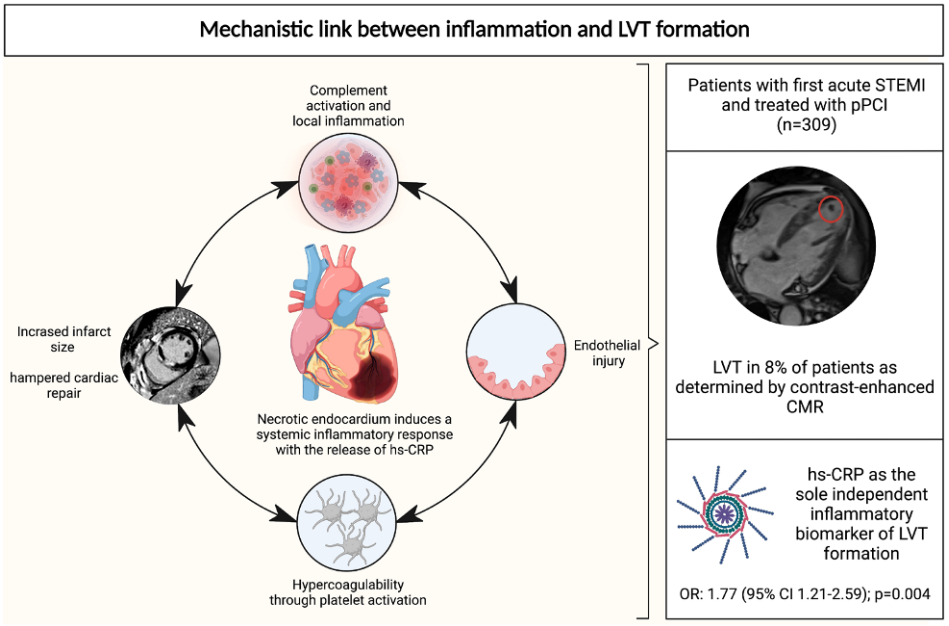

Fig. 1 Association between inflammation and left ventricular thrombus formation following ST-elevation myocardial infarction Association between the inflammatory response and LVT formation

Among all assessed inflammatory biomarkers, only hs-CRP was independently associated with LVT after adjustment for markers of inflammation and CMR parameters (OR: 1.77, 95 % CI:1.21–2.59, $p = 0.004$).

Conclusion: In patients with STEMI treated with pPCI, inflammatory markers (hs-CRP, IL-6 and fibrinogen) are associated with the presence of LVT. However, only hs-CRP was independently associated with the occurrence of LVT, highlighting the key role of CRP as clinical risk marker for LVT formation in STEMI patients treated with pPCI.

Annular remodeling predicts outcome in isolated severe tricuspid regurgitation: A registry-based echocardiographic analysis

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Introduction: Depending on volume status, quantitative parameters of functional tricuspid regurgitation (TR) are known to have a strong dynamic component. In contrast, structural dilatation of the tricuspid annulus and the right heart chambers

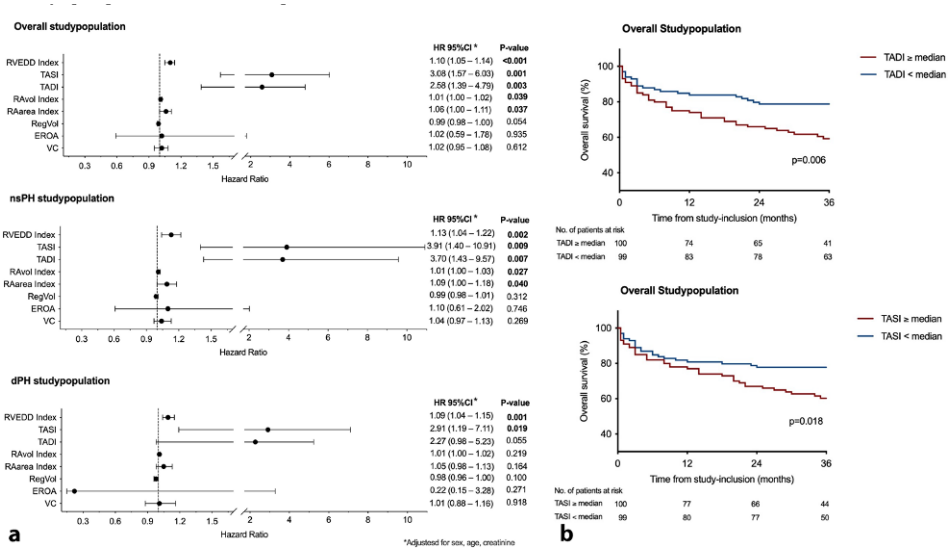

Fig. 1 Annular remodeling predicts outcome in isolated severe tricuspid regurgitation: A registry-based echocardiographic analysis Association of echocardiographic parameters with outcome in severe secondary isolated TR patients. Cox regression analyses of echocardiographic parameters (**a**), and Kaplan-Meier estimates for TADI and TASI (**b**) are shown

Table 1 Annular remodeling predicts outcome in isolated severe tricuspid regurgitation: A registry-based echocardiographic analysis Baseline characteristics and 2D echocardiography data of the entire cohort and for the subgroups nsPH group (TR-Vmax <3.5 m/s) and dPH group (≥3.5 m/s)

	Overall studypopulation	nsPH group: TR-Vmax <3.5 m/s	dPH group: TR-Vmax ≥3.5 m/s	*p*-value
N (%)	220 (100)	108 (49)	109 (50)	
Baseline characteristics				
Age, years (IQR)	69 (52–79)	71 (58–80)	66 (49–77)	0.072
Male sex, *n* (%)	88 (40)	44 (41)	42 (39)	0.739
NYHA class, *n* (%)				0.018
I	122 (56)	64 (64)	54 (50)	
II	24 (11)	13 (13)	9 (8)	
III	33 (15)	13 (13)	18 (17)	
IV	9 (4)	0 (0)	8 (7)	
Creatinine, mg/dl (IQR)	0.97 (0.80–1.34)	0.98 (0.82–1.34)	0.97 (0.78–1.35)	0.594
NT-proBNP, pg/ml (IQR)	1910 (867–3795)	1554 (651–2675)	2448 (1198–5350)	0.001
Follow-up, months (IQR)	35 (19–53)	33 (23–54)	35 (10–53)	0.555
3-year mortality, *n* (%)	70 (32)	30 (28)	39 (36)	0.206
Comorbidities and medication				
Coronary artery disease, *n* (%)	61 (28)	41 (38)	20 (18)	0.003
Diabetes mellitus, *n* (%)	43 (20)	19 (18)	24 (22)	0.290
Arterial hypertension, *n* (%)	129 (59)	65 (60)	62 (57)	0.694
ACE-I, *n* (%)	75 (34)	34 (32)	40 (37)	0.217
Betablockers, *n* (%)	108 (49)	64 (59)	44 (40)	0.016
Diuretics, *n* (%)	148 (67)	71 (66)	75 (69)	0.119
Echocardiography measurements				
RA area, cm² (IQR)	31.8 (26.5–38.8)	31.0 (26.9–39.6)	32.3 (26.9–39.3)	0.406
RV basal diameter, mm (IQR)	45 (40–51)	43 (37–47)	49 (42–55)	<0.001
TV annulus diameter diast. index, mm/m² (IQR)	23 (21–26)	23 (21–25)	24 (22–27)	0.029
TV annulus diameter syst. index, mm/m² (IQR)	21 (19–24)	21 (19–23)	22 (19–24)	0.075
TR-Vmax, m/s (IQR)	3.5 (3.0–4.4)	3.0 (2.6–3.2)	4.4 (3.9–4.9)	<0.001
Tricuspid regurgitation severity				
Vena contracta, mm (IQR)	9.8 (8.3–11.8)	10.5 (8.7–12.8)	9.5 (8.0–11.0)	0.001
EROA (PISA) - cm²	0.40 (0.26–0.54)	0.52 (0.39–0.78)	0.30 (0.22–0.41)	<0.001
RegV, ml (IQR)	44 (33–60)	48 (36–66)	42 (31–57)	0.031

ACE-I angiotensin-converting-enzyme inhibitor; *diast.* diastolic; *EROA* effective regurgitation orifice area; *IQR* interquartile range; *LA* left atrium; *NT-proBNP* N-terminal pro-brain natriuretic peptide; *NYHA* New York Hear Association functional classification; *PISA* proximal isovolumetric surface area; *RA* right atrium; RegV regurgitant volume; *RV* right ventricle; *sPAP* systolic pulmonary artery pressure; *syst.* systolic; *TR* tricuspid regurgitation; *TV* tricuspid valve; *Vmax* maximal velocity

may be less volume/pressure dependent. With this study, we sought to compare the prognostic value of remodeling versus TR severity parameters in patients with isolated severe TR (isoTR).

Methods: A total of 36,000 patients from the longitudinal echocardiographic database of our tertiary center were screened for severe isoTR (VC >7 mm) in the absence of other valve disease and/or reduced systolic left ventricular function (LVF). Echocardiographic examinations were reread, specially focusing on the right ventricular (RV) parameters RV end diastolic diameter (RVEDD), indexed tricuspid valve annulus diameter in diastole (TADI) and indexed tricuspid valve annulus diameter in systole (TASI). Patients were stratified according to the presence of concomitant pulmonary hypertension (dPH; maximal TR velocity signal (TRVmax) ≥3.5 m/s in echocardiography; nsPH: TRVmax <3.5 m/s). Three year all-cause mortality was defined the primary endpoint. Cox-regression and Kaplan-meier analyses were applied.

Results: 220 patients fulfilled the inclusion criteria, 50 % (n=109) revealed a TR Vmax ≥3.5 m/s (dPH group). During a median follow-up of 35 months (IQR: 19–53), all-cause mortality was 32 % (n=70). RVEDD, TADI and TASI were enlarged in the overall study population. Quantitative TR-parameters were significantly larger in the nsPH group compared to the dPH group (VC, EROA, RegV: p ≤0.031 for all; Table 1). Considering the recently proposed further subclassification of TR, most patients in this cohort had severe TR (n=187 [85 %]), and only few had massive (n=24 [11 %]) or torrential TR (n=5 [2 %]). The morphologic parameters TADI and TASI were not associated with TR severity reflected by EROA, VC and RegVol, but with disease severity reflected by NT-proBNP (TADI: r=0.42, p <0.001; TASI: r=0.42, p

<0.001). Multivariate Cox regression analysis revealed no statistically significant association of VC, EROA, or RegV with all-cause mortality. However, indexed RVEDD (HR 1.10, 95 %CI 1.05–1.14, p <0.001), indexed right atrial (RA) dimensions (RAarea index: HR 1.06, 95 %CI 1.00–1.11, p=0.037; RAvol index: HR 1.01, 95 %CI 1.00–1.02, p=0.039), and particularly TADI (HR 2.58, 95 %CI 1.39–4.79, p=0.003) and TASI (HR 3.08, 95 %CI 1.57–6.03, p=0.001) were significantly associated with outcome (Fig. 1a). Kaplan–Meier estimates showed a significant increase of long-term mortality when TADI and TASI were more enlarged (log-rank $p \leq 0.018$; Fig. 1b).

Conclusion: Isolated TR is rare and includes patients with significant pulmonary hypertension. Massive and torrential TR are not common in this population. Parameters of RV remodeling were consistently associated with prognosis, suggesting a "point of no return". Prospective studies should address this question as further evidence may support the therapeutic management of interventional procedures versus conservative recompensation strategy in significant TR without right heart remodelling.

Hepatic T1-time predicts cardiovascular risk in all-comers referred for cardiovascular magnetic resonance

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Introduction: Liver damage is frequently observed in patients with cardiovascular disease (CVD) but infrequently quantified. We hypothesized that in patients with CVD undergoing cardiac magnetic resonance (CMR), liver T1-times indicate liver damage and are associated with cardiovascular outcome.

Methods: We measured hepatic T1-times, displayed on standard cardiac T1-maps, in an all-comer CMR-cohort. At the time of CMR, we assessed validated general liver fibrosis scores. Kaplan-Meier estimates and Cox-regression models were used to investigate the association between hepatic T1-times and a composite endpoint of non-fatal myocardial infarction, heart failure hospitalization, and death.

Results: 1022 participants (58 ±18 y/o, 47 % female) were included (972 patients, 50 controls). Hepatic T1-times were 590 ±89 ms in patients and 574 ±45 ms in controls (p=0.052). They were significantly correlated with cardiac size and function, presence of atrial fibrillation, NT-pro-BNP levels, and gamma-glutamyl-transferase levels (p <0.001 for all). During follow-up (58 ±31 months), a total of 282 (29 %) events occurred. On Cox-regression, high hepatic T1-times yielded a significantly higher risk for events (adj.HR 1.66 [95 %CI: 1.45–1.89] per 100 ms increase, p <0.001), even when adjusted for age, sex, left and right ventricular ejection fraction, NT-proBNP, and myocardial T1-time. On restricted cubic splines, we found that a hepatic T1-time exceeding 610 ms was associated with excessive risk.

Conclusion: Hepatic T1-times on standard CMR scans were significantly associated with cardiac size and function, comorbidities, natriuretic peptides, and independently predicted cardiovascular mortality and morbidity. A hepatic T1-time >610 ms seems to indicate excessive risk.

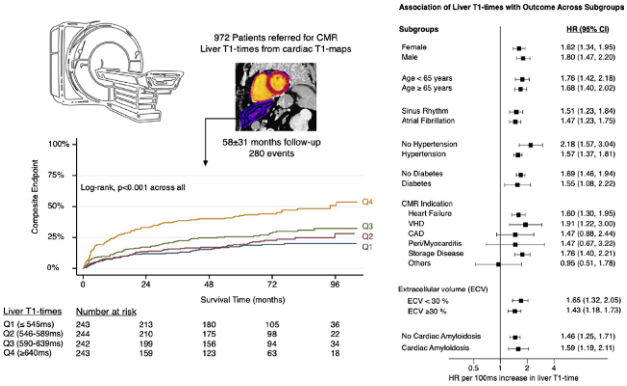

Fig. 1 Hepatic T1-time predicts cardiovascular risk in all-comers referred for cardiovascular magnetic resonance Central Figure

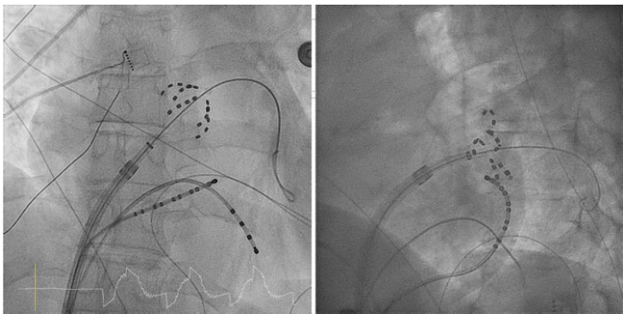

Fig. 2 Hepatic T1-time predicts cardiovascular risk in all-comers referred for cardiovascular magnetic resonance Restricted cubic spline (upper panel) demonstrating a sharp increase in risk for the composite endpoint, starting at 610 ms liver T1-time

Table 1 Baseline clinical characteristics

Variable	All patients (*n* = 2482)	No Trigger (*n* = 717)	Physical Trigger (*n* = 855)	Broken Heart (*n* = 873)	Happy Heart (*n* = 37)	*p*
Age, years	72 (63, 79)	74 (64, 80)	74 (64, 81)	70 (61, 77)	69 (62, 78)	<0.001
Male sex	285/2482 (11.5)	76/717 (10.6)	158/855 (18.5)	44/873 (5.0)	7/37 (18.9)	<0.001
Cardiovascular risk factors						
Hypertension	1684/2473 (68.1)	511/717 (71.3)	574/850 (67.5)	574/869 (66.1)	25/37 (67.6)	0.163
Diabetes mellitus	478/2473 (19.3)	130/717 (18.1)	199/851 (23.4)	147/868 (16.9)	2/37 (5.4)	<0.001
Hypercholesterolemia	984/2333 (42.2)	290/682 (42.5)	314/791 (39.7)	364/826 (44.1)	16/34 (47.1)	0.312
Current Smoking	439/2473 (17.8)	133/717 (18.5)	159/851 (18.7)	142/868 (16.4)	5/37 (13.5)	0.489
Obesity *	351/2137 (16.4)	114/620 (18.4)	134/733 (18.3)	99/748 (13.2)	4/36 (11.1)	0.020
Comorbidity						
Coronary artery disease	204/2148 (9.5)	52/635 (8.2)	59/715 (8.3)	90/762 (11.8)	3/36 (8.3)	0.061
Atrial fibrillation	344/2227 (15.4)	110/650 (16.9)	140/766 (18.3)	89/775 (11.5)	5/36 (13.9)	0.002
Malignancy	310/2132 (14.5)	80/599 (13.4)	146/744 (19.6)	79/761 (10.4)	5/28 (17.9)	<0.001
Pulmonary disease	340/2182 (15.6)	73/609 (12.0)	181/765 (23.7)	83/777 (10.7)	3/31 (9.7)	<0.001
Neurologic disorder	358/1985 (18.0)	99/560 (17.7)	165/708 (23.3)	89/689 (12.9)	5/28 (17.9)	<0.001
Psychiatric disorder	261/1958 (13.3)	73/541 (13.5)	77/673 (11.4)	109/717 (15.2)	2/27 (7.4)	0.166
Clinical presentation						
Chest pain	1326/2213 (59.9)	407/642 (63.4)	289/753 (38.4)	604/786 (76.8)	26/32 (81.2)	<0.001
Dyspnea	791/2213 (35.7)	240/642 (37.4)	326/753 (43.3)	214/786 (27.2)	11/32 (34.4)	<0.001
Killip class at admission						<0.001
1	1839/2482 (74.1)	531/717 (74.1)	567/855 (66.3)	708/873 (81.1)	33/37 (89.2)	
2	233/2482 (9.4)	75/717 (10.5)	90/855 (10.5)	67/873 (7.7)	1/37 (2.7)	
3	182/2482 (7.3)	53/717 (7.4)	77/855 (9.0)	50/873 (5.7)	2/37 (5.4)	
4	228/2482 (9.2)	58/717 (8.1)	121/855 (14.2)	48/873 (5.5)	1/37 (2.7)	
ST-segment change	1757/2146 (81.9)	509/617 (82.5)	590/724 (81.5)	633/774 (81.8)	25/37 (67.6)	0.966
Ballooning pattern†						0.110
Apical	2129/2481 (85.8)	625/717 (87.2)	714/855 (83.5)	763/872 (87.5)	27/37 (73.0)	
Midventricular	296/2481 (11.9)	78/717 (10.9)	119/855 (13.9)	91/872 (10.4)	8/37 (21.6)	
Basal	48/2481 (1.9)	12/717 (1.7)	20/855 (2.3)	14/872 (1.6)	2/37 (5.4)	
Focal	8/2481 (0.3)	2/717 (0.3)	2/855 (0.2)	4/872 (0.5)	-	
Initial LV-EF (%)	40 (33, 45)	40 (35, 50)	38 (30, 45)	40 (35, 45)	43 (31, 45)	<0.001
Follow-up LV-EF (%)	60 (55, 65)	60 (55, 64)	60 (55, 64)	60 (55, 65)	60 (56, 64)	0.400
Discharge medication						
Aspirin	1264/2196 (57.6)	393/664 (59.2)	386/741 (52.1)	467/754 (61.9)	18/37 (48.6)	<0.001
Dual antiplatelet therapy	169/1630 (10.4)	54/524 (10.3)	62/555 (11.2)	49/516 (9.5)	4/35 (11.4)	0.837
Oral anticoagulation	356/2011 (17.7)	125/617 (20.3)	127/696 (18.2)	95/661 (14.4)	9/37 (24.3)	0.029
Beta blocker	1503/2092 (71.8)	456/631 (72.3)	458/702 (65.2)	561/722 (77.7)	28/37 (75.7)	<0.001
ACE inhibitor / AT-R blocker	1532/2213 (69.2)	472/668 (70.7)	470/743 (63.3)	562/765 (73.5)	28/37 (75.7)	<0.001
Aldosterone antagonist	127/1631 (7.8)	46/524 (8.8)	46/555 (8.3)	33/517 (6.4)	2/35 (5.7)	0.468
Diuretic	538/1591 (33.8)	171/522 (32.8)	209/533 (39.2)	153/504 (30.4)	5/32 (15.6)	0.002
Statin	1147/2187 (52.4)	359/658 (54.6)	336/739 (45.5)	429/753 (57.0)	23/37 (62.2)	<0.001

Data are presented as number (percentage) of patients and median (interquartile range). Numbers in bold type indicate a significant difference.
* defined as body mass index ≥30 kg/m²
† One patient exhibited isolated right ventricular ballooning.
ACE Angiotensin converting enzyme; *AT-R* Angiotensin receptor; *LV-EF* left ventricular ejection fraction

Table 2 In-hospital course and long-term outcome

Variable	All patients (*n*=2482)	No Trigger (*n*=717)	Physical Trigger (*n*=855)	Broken Heart (*n*=873)	Happy Heart (*n*=37)	*p*
In-hospital complication*	477/2482 (19.2)	135/717 (18.8)	232/855 (27.1)	107/873 (12.3)	3/37 (8.1)	<0.001
In-Hospital death	77/2481 (3.1)	24/717 (3.3)	42/855 (4.9)	11/798 (1.4)	0/37 (0)	<0.001
Pulmonary edema	198/2482 (8.0)	59/717 (8.2)	84/855 (9.8)	53/873 (6.3)	2/37 (5.4)	0.034
Cardiogenic shock	229/2482 (9.2)	59/717 (8.2)	121/855 (14.2)	48/873 (5.5)	1/37 (2.7)	<0.001
Catecholamine therapy	216/2255 (9.6)	59/671 (8.8)	114/784 (14.5)	42/763 (5.5)	1/37 (2.7)	<0.001
Mechanical circulatory support	41/2364 (1.7)	11/701 (1.6)	16/805 (2.0)	13/821 (1.6)	1/37 (2.7)	0.728
Stroke	48/2173 (2.2)	11/619 (1.8)	30/760 (3.9)	7/758 (0.9)	-	<0.001
Length of stay in hospital (days)	7 (5, 10)	7 (5, 10)	8 (5, 13)	6 (4, 8)	6 (5, 9)	<0.001
Long-term mortality	335/2274 (14.7)	94/651 (14.4)	170/788 (21.6)	70/798 (8.8)	1/37 (2.7)	<0.001

Data are presented as number (percentage) of patients and median (interquartile range).
P-values were calculated for the comparison between happy and broken heart syndrome. Numbers in bold type indicate a significant difference.
*Death, cardiogenic shock, pulmonary edema, or stroke

Table 3 Predictors for in-hospital complications

Variable	Univariate		Multivariable	
	Odds ratio (95% CI)	*p*	Odds ratio (95% CI)	*p*
Age, years	1.02 (1.01–1.03)	<0.001	-	-
Male sex	2.36 (1.80–3.09)	<0.001	2.00 (1.30–3.07)	0.002
Hypertension	1.00 (0.80–1.23)	0.965		
Diabetes mellitus	1.74 (1.38–2.20)	<0.001	1.56 (1.09–2.24)	0.016
Hypercholesterolemia	0.93 (0.75–1.15)	0.495		
Current smoking	1.00 (0.77–1.30)	0.982		
Obesity	1.09 (0.82–1.45)	0.539		
Coronary artery disease	1.09 (0.76–1.57)	0.634		
Atrial fibrillation	2.64 (2.05–3.41)	<0.001	2.00 (1.35–2.98)	<0.001
Malignancy	1.36 (1.01–1.81)	0.039	-	-
Pulmonary disease	1.62 (1.23–2.11)	<0.001	-	-
Neurologic disease	2.02 (1.56–2.63)	<0.001	2.06 (1.40–3.04)	<0.001
Psychiatric disorder	1.21 (0.88–1.67)	0.232		
Chest pain	0.33 (0.26–0.41)	<0.001	0.45 (0.33–0.63)	<0.001
Dyspnea	4.06 (3.25–5.07)	<0.001	2.85 (2.07–3.94)	<0.001
ST-segment change	1.30 (0.97–1.76)	0.081		
Apical ballooning	1.48 (1.08–2.02)	0.015	-	-
Initial LV-EF	0.92 (0.91–0.93)	<0.001	0.92 (0.91–0.94)	<0.001
Physical Trigger	2.11 (1.73–2.59)	<0.001	-	-

Predictors for in-hospital complications in logistic regression analysis. Significant predictors are presented in bold type. The multivariable model included only significant predictors in univariable analysis.
CI confidence interval; *LV-EF* left ventricular ejection fraction

Table 4 Predictors for long term mortality

Variable	Univariate		Multivariable	
	Hazard ratio (95% CI)	*p*	Hazard ratio (95% CI)	*p*
Age, years	1.06 (1.05–1.08)	<0.001	1.08 (1.06–1.11)	<0.001
Male sex	2.24 (1.69–2.95)	<0.001	-	-
Hypertension	1.35 (1.05–1.73)	0.020	-	-
Diabetes mellitus	2.22 (1.77–2.79)	<0.001	2.67 (1.71–4.16)	<0.001
Hypercholesterolemia	1.01 (0.80–1.27)	0.944		
Current smoking	0.78 (0.58–1.05)	0.100		
Obesity	0.82 (0.60–1.14)	0.242		
Coronary artery disease	1.55 (1.07–2.23)	0.019	-	-
Atrial fibrillation	2.41 (1.87–3.11)	<0.001	-	-
Malignancy	2.40 (1.84–3.13)	<0.001	2.50 (1.50–4.17)	<0.001
Pulmonary disease	2.16 (1.65–2.82)	<0.001	-	-
Neurologic disease	2.26 (1.76–2.90)	<0.001	2.08 (1.33–3.24)	0.001
Psychiatric disorder	1.03 (0.72–1.48)	0.851		
Chest pain	0.40 (0.31–0.51)	<0.001	-	-
Dyspnea	2.05 (1.60–2.61)	<0.001	-	-
Killip class at admission	1.69 (1.56–1.84)	<0.001	1.43 (1.19–1.72)	<0.001
ST-segment change	1.31 (0.92–1.88)	0.136		
Apical ballooning	1.66 (1.18–2.33)	0.004	-	-
Initial LV-EF	0.95 (0.94–0.96)	<0.001	-	-
Follow-up LV-EF	0.95 (0.94–0.97)	<0.001	-	-
Aspirin	0.66 (0.52–0.83)	<0.001	-	-
Dual antiplatelet therapy	1.12 (0.76–1.63)	0.568		
Oral anticoagulation	1.32 (0.97–1.80)	0.072		
Beta blocker	0.64 (0.50–0.82)	<0.001	-	-
ACE inhibitor / AT-R blocker	0.53 (0.42–0.67)	<0.001	0.54 (0.36–0.83)	0.005
Aldosterone antagonist	1.38 (0.82–2.30)	0.222		
Diuretic	1.59 (1.19–2.14)	0.002	-	-
Statin	0.87 (0.68–1.10)	0.233		
Physical trigger	2.36 (1.90–2.93)	<0.001	1.80 (1.16–2.80)	0.009

Predictors for long-term mortality Cox regression analysis. Significant predictors are presented in bold type. The multivariable model included only significant predictors in univariable analysis.
CI confidence interval; *LV-EF* left ventricular ejection fraction

Vorträge

Diagnostic accuracy of amyloid scintigraphy for the histopathological diagnosis of cardiac transthyretin amyloidosis–a retrospective Austrian multicenter study

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Introduction: Previous studies indicated that amyloid scintigraphy in combination with free light chain (FLC) assessment yields an excellent diagnostic accuracy for cardiac transthyretin (ATTR) amyloidosis [1]. As a consequence, the diagnosis of ATTR amyloidosis is increasingly made without the actual gold-standard method endomyocardial biopsy (EMB). Whether this leads to misdiagnosis in real-world practice is currently under-investigated. We aimed to describe the diagnostic accuracy of amyloid scintigraphy in a real world setting performing a multicenter retrospective study.

Methods: Seven tertiary care centers throughout Austria agreed to participate in the study and performed a systematic retrospective medical records search from 2017 to 2020 after ethical approval was obtained. Patients were included in case of available results of amyloid scintigraphy, FLC assessment and EMB, respectively. Amyloid scintigraphy was performed using a 99 m-technetium-labelled tracer. Histological analysis was performed using immunohistochemistry. The number of submitted subjects with complete data per center ranged from 2–46. The patient number increased with years, with 15 patients investigated in 2017 and 32 in 2020.

Results: We enrolled 101 patients (21 % women) with a mean age of 73 ± 9 years and median NT-proBNP of 2694 (IQR 1601–5239) pg/ml (Table 1). An abnormal Perugini Score (ie. grade II or III) was present in 57 patients (56 %) and FLC assessment was overall indicative of monoclonal protein in 60 patients (59 %). Among patients with abnormal Perugini Score, 29 had FLC assessment indicative of monoclonal protein. The most common histopathological diagnoses were ATTR in 60 patients (59 %) and cardiac light chain (AL) amyloidosis in 20 patients (20 %). One further patient was diagnosed with concomitant AL and ATTR amyloidosis. Further diagnoses included ApoA4 ($n=2$) and AA amyloidosis ($n=1$), while cardiac amyloidosis was ruled out in 17 patients (17 %). ATTR was diagnosed in 54 patients with Perugini Score II or III compared with 6 patients with Perugini less than II, yielding a sensitivity of abnormal Perugini score for ATTR amyloidosis of 90 %. Among patients with abnormal Perugini Score ($n=57$), ATTR was diagnosed in 55 patients, and AL amyloidosis in 3 (one had concomitant ATTR and AL), yielding a positive predictive value (PPV) of abnormal Perugini Score of 97 % (Table 2). Two AL patients had Perugini Score of II and one had Perugini Score of III. When excluding patients with monoclonal gammopathy, the PPV of abnormal Perugini Score was 100 %.

Table 1 Baseline characteristics in the whole cohort and stratified by Perugini Score

All ($n=101$)			Perugini Score
0/I ($n=44$)			II/III ($n=57$)
Age, years	72.5 ± 8.9	68.8 ± 9.9*	75.4 ± 6.9*
Women, n (%)	21 (21 %)	15 (34 %)*	6 (11 %)*
NT-proBNP, pg/ml	2694 (1601–5239)	3037 (1363–5296)	2578 (1624–5183)
eGFR MDRD, ml/min/1.73 m²	59 ± 19	58 ± 18	61 ± 19
Endomyocardial biopsy diagnosis			
ATTR#	60 (59.4 %)	6 (14 %)	54 (95 %)
AL	20 (19.8 %)	18 (41 %)	2 (3 %)
ATTR + AL	1 (1.0 %)	0	1 (2 %)
ApoA4	2 (2.0 %)	2 (4 %)	0
AA	1 (1.0 %9	1 (2 %)	0
..negative	17 (16.8 %)	17 (39 %)	0
Amyloid scintigraphy			
Scintigraphy			
Tracer			
DPD	86 (85 %)	34 (77 %)	52 (91 %)
HDP	5 (6 %)	3 (7 %)	3 (5 %)
PYP	9 (9 %)	7 (16 %)	2 (4 %)
Perugini Score			
0	31 (31 %)	31 (71 %)**	0**
I	13 (13 %)	13 (30 %)	0
II	16 (16 %)	0	16 (28 %)
III	41 (40 %)	0	41 (72 %)
Haematology			
Serum FLC ratio	1.50 (0.79–1.99)	1.38	1.64
Monoclonal protein	60 (59 %)	(1.68–2.44)	(1.00–1.98)
..Monoclonal band	46 (46 %)	31 (71 %)*	29 (51 %)*
serum	46 (46 %)	29 (66 %)**	17 (30 %)**
urine	26 (26 %)	28 (65 %)*	18 (33 %)*
Abnormal FLC ratio	51 (51 %)	14 (34 %)	12 (23 %)
		26 (59 %)*	25 (44 %)*

2 patients with hereditary transthyretin amyloidosis
* $P<0.05$; ** $P<0.001$, for group comparison between Perugini Score 0/I vs II/III
Abbreviations: *AA* Amyloid A; *AL* amyloid light chain; *ApoA4* Apolipoprotein A4; *ATTR* amyloid transthyretin; *DPD* diphosphono-1,2-propanodicarboxylicacid; *eGFR* estimated glomerular filtration rate; *FLC* free light chain; *HDP* hydroxydiphosphonate; *MDRD* Modification of Diet in Renal Disease formula, *NT-proBNP* N-terminal prohormone of brain natriuretic peptide; *PYP* pyrophosphate

Conclusion: Our data confirm a PPV of abnormal amyloid scintigraphy of 100 % for cardiac ATTR amyloidosis when monoclonal gammopathy was excluded. However, among patients with monoclonal gammopathy, one of ten patients with abnormal scintigraphy has AL amyloidosis as the underlying condi-

Table 2 Diagnostic accuracy of abnormal amyloid scintigraphy (Perugini Score II or III) for the histopathological diagnosis derived from endomyocardial biopsy ($n = 101$)

0		Perugini Score				Sensitivity	Specificity	PPV	NPV
		I	II	III					
			abnormal						
EMB diagnosis	ATTR	1	5	14	40	90.2 %	92.7 %	96.5 %	86.4 %
		6		54					
	ATTR+AL	0	0	1	0				
	AL	14	4	1	1				
		18		2					
	ApoA4	2	0	0	0				
	AA	1	0	0	0				
	Negative	13	4	0	0				
Sums per column		31	13	16	41				
	44		57						

All three patients with AL and abnormal Perugini Score had monoclonal band in serum.
Abbreviations: *ATTR* transthyretin amyloidosis; *AL* light-chain amyloidosis; *EMB* endomyocardial biopsy; *PPV* positive predictive value; *NPV* negative predictive value

tion. Our data underscore that tissue biopsy and histopathological analysis should be performed in every patient with suspected amyloidosis and monoclonal gammopathy even in case of Perugini Score II or III.

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Invasive hemodynamic assessment prior to transcatheter tricuspid valve repair–impact of patient selection and procedural success on right ventricular remodeling and outcome

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Introduction: Severe tricuspid regurgitation (TR) is a common condition promoting right heart failure and is associated with a poor long-term prognosis. Transcatheter tricuspid valve repair (TTVR) emerged as a low-risk alternative to surgical repair techniques, however patient selection remains controversial. Aim: We therefore aimed to investigate the impact of preprocedural invasive hemodynamic assessment and procedural success on right ventricular (RV) remodeling and outcome.

Methods: All patients undergoing TTVR with a TR reduction ≥2 grades or with a TR reduction of one grade without precapillary/combined pulmonary hypertension (PH, mean pulmonary artery pressure (mPAP) ≥25 mm Hg, mean pulmonary artery Wedge pressure ≤15 mm Hg, pulmonary vascular resistance ≥3 Wood units) were assigned to the responder group. All other patients were classified as non-responders.

Results: A total of 107 patients were enrolled, 75 were classified as responders and 32 as non-responders. We observed evidence of significant RV reverse remodeling in responders with a decrease in RV diameters (–2.9 mm, $p = 0.001$) at a mean follow-up of 229 days (± SD) after TTVR. RV function improved in responders (fractional area change (FAC) + 5.7 %, $p < 0.001$, RV free wall strain +3.9 %, $p = 0.006$), but interestingly further deteriorated in non-responders (FAC –4.5 %, $p = 0.003$, RV free wall strain –3.9 %, $p = 0.007$). Non-responders had more persistent symptoms compared to responders (NYHA ≥3, 72 % vs. 11 % at follow-up). Subsequently, non-response was associated with a poor long-term prognosis in terms of death, heart failure (HF) hospitalization and re-intervention after 2 years (freedom of death, HF hospitalization and reintervention at 2 years: 16 % vs. 78 %, log-rank: $p < 0.001$).

Conclusion: Hemodynamic assessment before TTVR and procedural success are significant factors for patient prognosis. The hemodynamic profiling prior to intervention is an important component in patient selection for TTVR. The window for edge-to-edge TTVR might be limited, but timely intervention is an important factor for better outcome and successful right ventricular reverse remodeling.

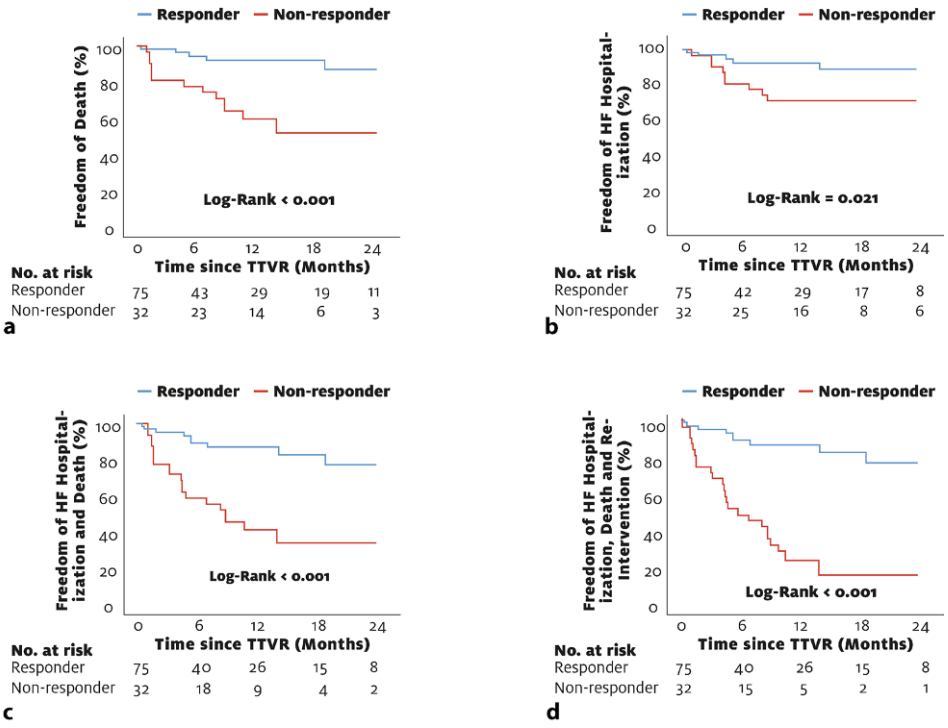

Fig. 1 Invasive hemodynamic assessment prior to transcatheter tricuspid valve repair–impact of patient selection and procedural success on right ventricular remodeling and outcome

Long-term outcomes after surgical repair of supravalvular aortic stenosis in pediatric patients–30 years single center outcome

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Introduction: Supravalvular aortic stenosis (SVAS) can be focal with a distinct stenosis localization or with a diffuse narrowing of the ascending aorta. Surgical techniques consist of the single patch repair technique (Mc Goon repair), the pantaloon- or Y-shaped patch, which extends into two aortic valve sinuses (Doty technique), and the 3-patch technique, where three separate triangular patches are inserted in the three sinuses (Brom technique).

Methods: A retrospective chart review of all patients aged below 18 years at time of surgery, who had undergone surgery for SVAS from May 1985 until April 2020 was conducted. Mortality was cross-checked with the national health insurance database and could be provided for all, but one patient, who was transferred only for surgery from a foreign country.

Results: From May 1985 until April 2020 20 repairs of SVAS were performed in 19 patients (63.2 % male; 52.6 % Williams Beuren syndrome; familial SVAS (Eisenberg syndrome) in 1 patient). The corrective surgeries consisted of a single patch repair in 4 cases, a pantaloon-shaped patch in 8 cases and a 3-patch technique in 8 cases. Median age at time of surgery was 1.3 years (IQR 0.8–2.8 years). In 10 cases concomitant patch plasty of the pulmonary arteries was performed and in 2 cases concomitant aortic valve repair was performed. There were no early deaths. Two patients required postoperative extracorporeal membrane oxygenation (ECMO) support and in 5 cases delayed sternal closure (including the two ECMO patients) was necessary. One late death occurred in a 6 months old Williams Beuren patient after re-operation for coarctation of the aorta 15 months after correction of the SVAS with a pantaloon-shaped patch. Kaplan-Meier estimated survival was 94.7 % ± 5.1 % at 30 years. Re-operation (one pantaloon-patch repair, two Ross procedures) for re-SVAS occurred in three cases and freedom from re-operation for re-SVAS was 93.3 % ± 6.4 % at 5 years, 86.2 % ± 9.1 % at 10 years and 75.4 % ± 12.8 % at 30 years.

Conclusion: Early outcomes are excellent as no early deaths occurred, but perioperative management regarding anesthesia and cardiopulmonary bypass weaning is complicated due to higher than usual pressures are often required to ensure myocardial perfusion via the thick-walled coronary arteries and ostial stenoses. In this complex patient cohort close follow-up is warranted, though long-term survival rand re-operation rates are good.

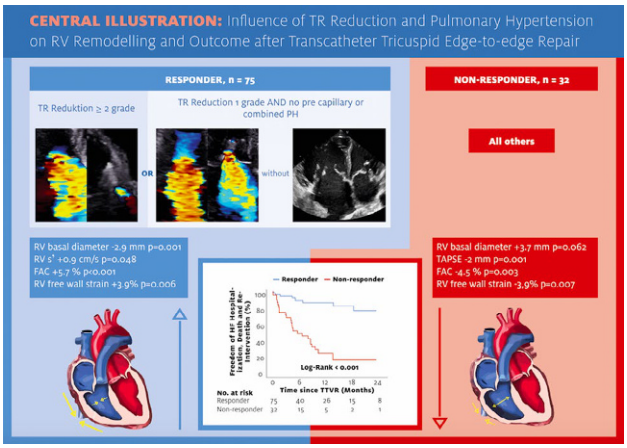

Fig. 2 Invasive hemodynamic assessment prior to transcatheter tricuspid valve repair–impact of patient selection and procedural success on right ventricular remodeling and outcome

A long-term single center experience of chronic thromboembolic pulmonary hypertension

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Introduction: Chronic thromboembolic pulmonary hypertension (CTEPH) is characterized by organized thrombus material within the pulmonary arteries, leading to an increase of mean pulmonary arterial pressure (mPAP) and consecutive right heart failure. Here we summarize our experience with CTEPH as a national reference center.

Methods: We prospectively collected data of 501 patients who were diagnosed with CTEPH at the Vienna General Hospital between 01/1993 and 06/2018. Diagnosis of CTEPH was based on right heart catheterization with hemodynamic assessment, complemented by characteristic findings by pulmonary angiography and computed tomography. The day of right heart catheterization was defined as diagnosis and enrollment date. Prevalence of general cardiovascular risk factors was assessed using definitions according to international guidelines. Established risk factors for CTEPH (e. g. history of pulmonary embolism, inflammatory bowel disease, splenectomy etc.) were also recorded. Mortality data was obtained from the Austrian Registry of Death. All-cause mortality was defined as the primary endpoint. Date of censoring was 31.12.2020. Outcome analysis was performed using multivariable Cox regression.

Results: At diagnosis, age of CTEPH patients was 60 [48, 71] years; 53.1 % patients were male. mPAP was 48 [39, 55] mm Hg. proBNP was 1353 [378, 3008] pg/ml. Cardiovascular risk factors were frequent, with diabetes mellitus, arterial hypertension and hyperlipidemia being present in 33.0 %, 61.0 % and 57.5 %, respectively. 78.7 % of patients had a history of pulmonary embolism; 43.5 % had prior deep vein thrombosis. Pulmonary endarterectomy (PEA) was performed in 52.5 % of patients. Balloon pulmonary angioplasty (BPA) was offered since mid 2014 and was performed in 18.8 % of patients since then. Over a total follow-up period of 28.3 years, 205 patients (40.9 %) died. Median survival was 13.7 years. Using multivariable Cox regression, we found that age (adjusted HR per year 1.047, 95 % CI 1.021–1.076, $p < 0.001$), coronary artery disease (adjusted HR 2.344, 95 % CI 1.307–3.854, $p = 0.003$) and proBNP (adjusted HR per standard deviation 1.266, 95 % CI 1.094–1.466, $p = 0.002$) independently predicted all-cause mortality after adjustment for other demographic risk factors. Serum creatinine did not predict outcome. Pulmonary endarterectomy was associated with better survival (adjusted HR 0.347, 95 % CI 0.184–0.654, $p = 0.001$). In patients undergoing BPA, survival improved with an increasing number of interventions (HR per BPA session 0.748, 95 % CI 0.647–0.865, $p < 0.0001$).

Conclusion: In our single center experience, we can present evidence from a large CTEPH cohort. Cardiovascular risk factors are common, with coronary artery disease conferring particularly poor outcome. Mechanical treatments were beneficial, but the impact of medical treatments deserves further analyses.

Electroanatomic mapping system guided His Bundle Pacemaker Implantation: Experience of the His Bundle Registry Graz

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Introduction: Patients with bradyarrhythmia in need for pacemaker implantation and ventricular pacing may suffer from pacing-induced heart failure due to unphysiological pacing by the right ventricular lead. His bundle pacing allows to overcome this common issue with a more physiologic approach but real-life procedural data using this technology is scarce.

Methods: We report a single centre experience of the first 44 consecutive patients being implanted with a His-bundle-based pacemaker 09/2020–11/2021 per 3D-mapping guided implantation due to different types of bradyarrhythmia, or for cardiac resynchronisation therapy in heart failure combined with a left-ventricular lead (HOT-CRT) ± a right ventricular defibrillator lead. The positioning of the His-bundle-lead was done by identifying the His-bundle-location with a 3D electroanatomic mapping system via an introducing sheath that is provided with electrodes at its tip.

Results: Mean age was 70 [16;86] years, 12/44 (27 %) patients were female, mean baseline LVEF was 44 ± 18 %. Baseline ECG was captured before implantation: QRS width was 123 ± 33 ms, with typical LBBB in 14/44 (32 %), typical RBBB in 4/44 (9 %), alternating BBB in one patient (2 %) and either no BBB or ventricular escape rhythm in 25/44 (57 %). Indications for implantation were AV-block grade II-III in 19/44 (43 %), primary prophylactic ICD indication in HFrEF in 13/44 (30 %), atrial fibrillation with bradycardic conduction in 7/44 patients (16 %), sick-sinus-syndrome in 4/44 (9 %) and secondary prophylactic ICD indication in one patient (2 %). In 41/44 (93 %) a primary device was implanted, in 3/44 (7 %) a pre-existent device was upgraded with a HB lead. Therefore, 14 dual-chamber-pacemaker, 5 single-chamber-pacemaker, 8 single-chamber CRT-P, 11 dual-chamber CRT-P, 4 single-chamber CRT-D, 2 dual-chamber CRT-D were implanted. In 50 patients his bundle pacing was attempted, while in 6/50 (12 %) patients outside of this analysis the attempt was not successful, these patients were consecutively implanted with a non-HBP-device and therefor excluded from the further analysis. In the 44 patients included in this analysis with a primary successful pacing at the his-position, 4/44 (9 %) his-bundle-leads dislocated within the first 48 h, leading to a secondary success rate of 91 %. There were two post-procedural pneumothorax that needed drainage, no major procedure-related complications occurred.

Conclusion: Median skin-to skin procedure time was 109 ± 50 min in his-bundle-device-implantation. The paced QRS width at the post-implantation follow up was 115 ± 32 ms with a change in QRS width of –10 ms (+72; –92 ms). When excluding the secondary lead dislocations and including only the successful HB paced QRS complexes, the paced QRS width was 105 ± 30 ms and the change in QRS width was –12 ± 42 ms. The mean his-bundle threshold was 1.2 ± 1 V over 0.5 ms (0.5;1.5 ms). The proportion of ventricular pacing was 69 ± 38 %. Electroanatomic-guided His bundle pacing is feasible, with high implantation success rate and electric impact, both regarding QRS width and pacing threshold.

Barriers and facilitators for employing digital technologies in cardiac rehabilitation: A cross-sectional online survey among healthcare professionals in Austria

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Introduction: Cardiovascular diseases remain the leading cause of death and the main contributor to loss of healthy life expectancy globally. Much could be achieved through risk factor modification in primary and secondary prevention. However, this requires patients to adopt sustained heart-healthy behaviours, which continues to prove challenging [1]. Advances in digital technologies have opened new and promising avenues for offering patients support with lifestyle modification, including mHealth apps, teleconsultation, telemonitoring, and telerehabilitation [2]. While the scientific evidence-base for the effectiveness of these digital modalities is continuously growing, their adoption and spread in clinical practice depends on a host of hindering and facilitating factors relating to: the technology itself, its value proposition to users, characteristics, attitudes and experiences of user groups (patients, their caregivers and healthcare professionals), and organisational and wider system factors [3]. A thorough understanding of these factors is required, so that barriers may be addressed and facilitators may be leveraged early on in the design of digital technology interventions and in their implementation in clinical practice. The aim of this study was to explore barriers and facilitators for employing digital technologies in the secondary prevention of cardiovascular disease, from the perspective of healthcare professionals in cardiac rehabilitation (CR).

Methods: An online survey questionnaire was developed based on scientific literature and input from CR experts. The questionnaire underwent thorough piloting, including cognitive debriefing interviews, with 8 representatives from different healthcare professions in CR. The survey was deployed via the LimeSurvey© platform during November 2021–February 2022 across Austria. All healthcare professionals working in patient-facing roles in CR phase I (acute care), phase II (4–6 weeks inpatient or outpatient programme), phase III (6–12 months outpatient programme), and phase IV (patients' self-directed lifelong heart-healthy lifestyle) as well as healthcare professionals working in home-based long-term disease management services for cardiac patients were invited to participate. Email invitations with open link to the online survey were sent to medical and nursing directors of all 13 inpatient cardiac rehabilitation centres, 21 outpatient cardiac rehabilitation providers, and 3 long-term disease management services for cardiac patients in Austria; and to the executives of all relevant professional societies in Austria (cardiology, dietetics, nursing, nutritional science, occupational therapy, physiotherapy, psychology, social work and sports science). Recipients were asked to forward the survey invitation via email to all staff/members of their organisation. Research ethics approval was obtained from the University of Salzburg (EK-GZ 21/2021).

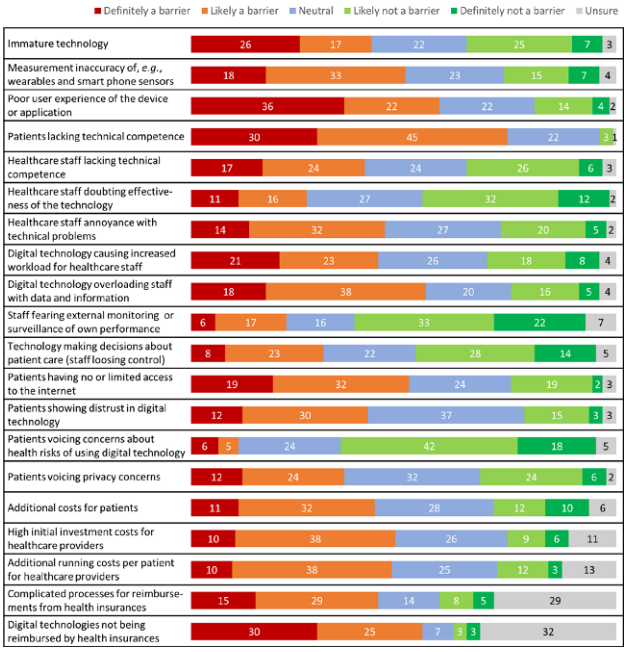

Fig. 1 Barriers and facilitators for employing digital technologies in cardiac rehabilitation: A cross-sectional online survey among healthcare professionals in Austria Barriers to employing digital technologies in cardiac rehabilitation and in the long-term disease management of cardiac patients. Respondents ($N = 125$) rated each potential barrier on a 5-point Likert scale. Shown are percentages for each response category

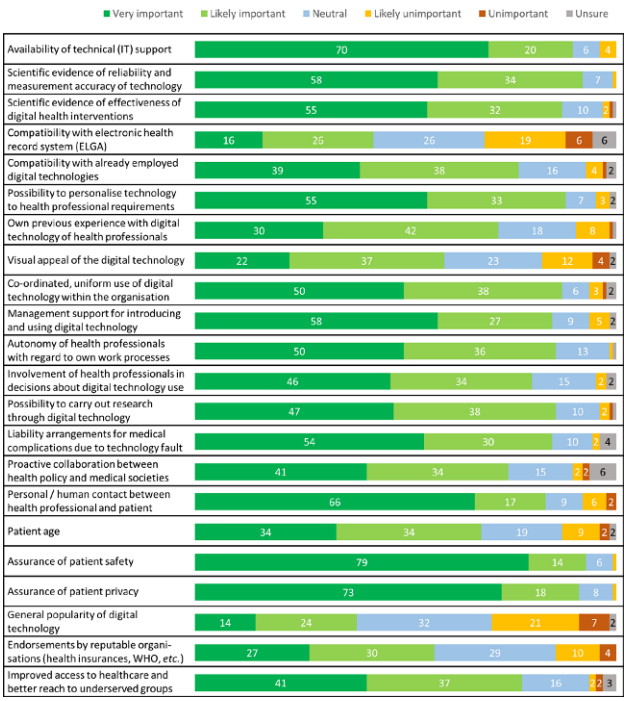

Fig. 2 Barriers and facilitators for employing digital technologies in cardiac rehabilitation: A cross-sectional online survey among healthcare professionals in Austria Influencing factors in the decision to use digital technologies. Respondents ($N = 125$) rated each potential facilitator on a 5-point Likert scale. Shown are percentages for each response category

Results: The survey recruited a convenience sample of 125 healthcare professionals (64 % female, median [IQR] age 40 [30, 49] years) representing medicine (20 %), nursing (32 %), sports science (17 %), physiotherapy (10 %), psychology/psychotherapy (10 %), dietetics (8 %), and others. Respondents worked in CR phases I (22 %), II (64 %), III (36 %) and IV (26 %), and in long-term disease management services for cardiac patients (6 %). General willingness for employing digital technologies in the care of cardiac patients was high (median [IQR] 2 [1, 2] on a 5-point Likert scale from 1 ['very willing'] to 5 ['very unwilling']), but only 65 (52 %) respondents reported that they currently used digital technologies, including activity trackers (15 %), step counters (18 %), smart watches (17 %), watches that display heart rate measurement from the wrist (26 %) or from a chest strap (49 %), online information (18 %), and apps (32 %). The top-3 rated barriers (response 'definitely a barrier') for employing digital technologies were poor user experience of devices/applications, patients lacking technical competence, and lack of reimbursement from health insurances (Fig. 1). The top-3 rated influencing factors (facilitators, response 'very important') in the decision to use digital technology were the assurance of patient safety of digital technologies, assurance of patient privacy of digital technologies, and availability of technical support (Fig. 2).

Conclusion: This study provides a current snapshot of barriers and facilitators for employing digital technologies in CR from the perspective of healthcare professionals in Austria. Within the acknowledged limitation of an open online survey design (recruitment of a self-selected convenience sample with likely prior interest in digital technology applications), these findings offer insights for developers, researchers, and adopters of digital technologies. Prominent barriers and facilitators highlighted in this study should be considered for the development and implementation of digital technologies. This will support the acceptability of digital technologies to healthcare professionals and contribute to a successful adoption and spread of digital technology interventions into clinical practice.

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High energy vs. pacemaker lead extraction using advanced extraction techniques

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Introduction: With growing incidence of cardiac implantable electric devices (CIEDs) in the last years, transcatheter lead extraction (TLE) becomes increasingly important in the treatment of device complications. However, limited data is available about outcome of TLE in patients with vs. without high energy leads in the last decades.

Methods: This is an analysis of consecutive patients undergoing TLE at a high-volume TLE centre from 2001–2021, using the stepwise approach. Baseline characteristics, procedural details and outcome of patients with high energy lead (ICD group) vs. without high energy lead (no-ICD group) were compared. Furthermore, the validity of various risk scores (SAFeTY TLE and MB scores) was evaluated.

Results: Out of 667 procedures undergoing TLE, 991 leads were extracted in 393 procedures (58.9 %) in the ICD group and 439 leads in 274 procedures (41.1 %) in the no-ICD group. ICD patients were significantly younger (median 67 vs. 74 years) and were significantly less often female (18.1 vs. 27.7 %, $p < 0.005$ for both). Advanced extraction tools were used significantly more often in the ICD group (73.2 % vs. 37.5 %, $p < 0.001$), but there were no significant differences in the successful removal (98.5 % vs. 99.6 %) and any complications (4.7 % vs. 3.1 %) between both groups ($p > 0.2$ for both). The SAFeTY risk score and the MB score significantly correlated with a complex procedure and the occurrence of complications, with higher ROC-AUC values with the MB score.

Conclusion: Using the stepwise approach, overall procedural success was high and complication rate was low in a high-volume centre. In patients with a high energy lead, the TLE procedure was more complex, but outcome was comparable to remaining patients.

Impact of different triggers on complications and outcome in patients with Takotsubo Syndrome: Results from the international, multicenter GEIST registry

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Introduction: Takotsubo syndrome (TS) is a form of acute heart failure related to a typical pattern of transient left ventricular (LV) contraction abnormalities. The association with a preceding stressful event, either of physical or emotional nature, is a characteristic feature of TS. The aim of the present study was to

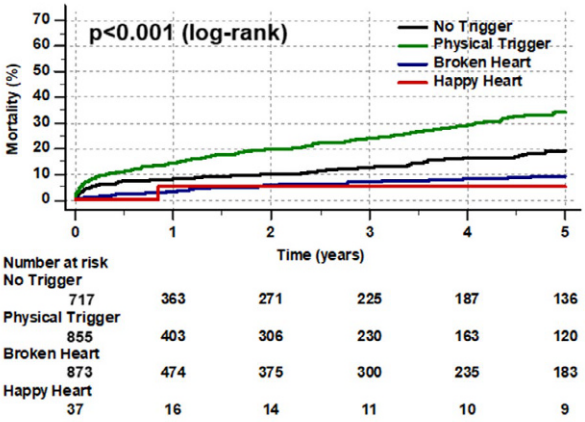

Fig. 1 Impact of different triggers on complications and outcome in patients with Takotsubo Syndrome: Results from the international, multicenter GEIST registry Kaplan-Meier Plot

analyze the prognostic impact of different triggering events preceding TS in the GErman-Italian-Spanish Takotsubo (GEIST) registry.

Methods: The international, multicenter GEIST registry includes 2492 cases with confirmed TS from 49 participating study centers in Germany, Italy and Spain. Patients with available information regarding stressful triggers ($n=2482$; 99.6 %) were included in the present study. The preceding stressors were classified as "physical" ($n=855$ patients; 34.4 %), "emotional" ($n=910$ patients; 36.7 %) or "no identifiable trigger" ($n=717$ patients; 28.9 %) according to the nature of the episodes. Emotional triggers were further categorized as 'broken hearts' ($n=873$; 95.9 %) in case of negative and 'happy hearts' ($n=37$; 4.1 %) in case of positive emotional events. The primary study endpoints were in-hospital complications (defined as death, pulmonary edema, cardiogenic shock, and stroke) and long-term mortality.

Results: Baseline characteristics revealed distinct clinical phenotypes depending on the stressful triggers. Patients with physical triggers were significantly older, more frequently male and had a higher prevalence of cardiovascular risk factors and severe comorbidities compared to emotionally triggered TS. Furthermore, the clinical presentation was more frequently characterized by dyspnea rather than chest pain in case of physical triggers and the Killip-class on admission was significantly worse, consistent with a more severely impaired LV function. These aspects translated into higher rates of in-hospital complications (27.1 % vs. 12.1 %; $p<0.01$) and long-term mortality (21.6 % vs. 8.5 %; $p<0.01$) after physical compared to emotional triggers. The risk profile of patients without identifiable triggers was roughly between those of patients with physically and emotionally triggered TS, which is also reflected in intermediate rates of in-hospital complications (18.8 %) and long-term mortality (14.4 %). The comparison between 'happy' and 'broken heart syndrome' showed numerically lower event rates in 'happy hearts' without reaching statistical significance (in-hospital complications: 8.1 % vs. 12.3 %, $p=0.45$; long-term mortality: 2.7 % vs. 8.8 %, $p=0.20$). Multivariable regression analyses revealed that physical triggers were not independently associated with the in-hospital course, but they emerged as valuable markers for long-term mortality (HR 1.80; 95 % CI 1.16–2.80; $p<0.01$).

Conclusion: The different nature of preceding stressful triggers in patients with TS reveals distinct clinical phenotypes and risk profiles. Physically triggered TS is associated with a particularly increased risk for adverse outcome and emerged as an independent predictor of long-term mortality. Therefore, a systematic differentiation of patients with TS according to their underlying triggers could be the basis for an individualized management with improved outcome.

The presence of cardiogenic shock is not associated with poor long-term survival in patients undergoing percutaneous coronary intervention during acute coronary syndrome

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Introduction: The development of cardiogenic shock (CS) mirrors a common and dreaded complication during the acute phase of acute coronary syndrome (ACS). While a strong association of CS with in-hospital mortality is well established, less attention has been paid on its prognostic impact on patient outcome from a long-term perspective. Thus, evidence-based data on the individual risk for fatal cardiovascular events in CS patients is needed in order to provide tailored secondary prevention measures in the era of personalized medicine.

Methods: To evaluate the prognostic impact of CS on long-term survival, 1173 patients presenting with ACS who underwent percutaneous coronary intervention (PCI) at Vienna General Hospital, Austria, between 1997 and 2009 were enrolled. Individuals were screened for the presence of CS at the time of PCI-defined as signs of hemodynamic instability, the development of fatal cardiac arrythmias and cardiac arrest. Patients were followed prospectively until the primary study endpoint (cardiovascular mortality) was reached.

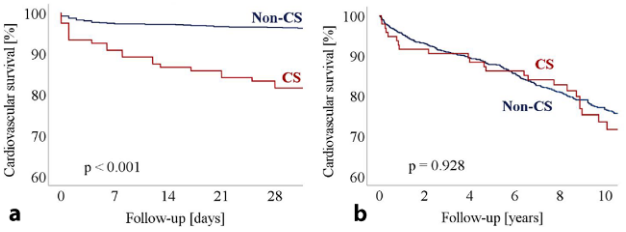

Fig. 1 The presence of cardiogenic shock is not associated with poor long-term survival in patients undergoing percutaneous coronary intervention during acute coronary syndrome Accumulated cardiovascular survival comparing hemodynamically stable patients to patients with cardiogenic shock, (**a**) during the acute event, (**b**) after survival of the acute event

	Crude HR (95% CI)	p-value	Adjusted HR (95% CI)	p-value
In-hospital mortality				
Cardiogenic shock	6.20 (3.77 – 10.19)	< 0.001	7.45 (4.51 – 12.33)	< 0.001
Cardiovascular mortality after hospital discharge				
Cardiogenic shock	1.02 (0.67 – 1.57)	0.928	1.18 (0.77 – 1.81)	0.455

Fig. 2 The presence of cardiogenic shock is not associated with poor long-term survival in patients undergoing percutaneous coronary intervention during acute coronary syndrome Cox proportional hazard model of crude and adjusted effects of cardiogenic shock on in-hospital mortality and cardiovascular long-term mortality after hospital discharge. The multivariate model was adjusted for age and sex

Results: Within the entire study population ($n=1173$), a total of 122 (10.4 %) individuals developed CS during the initial phase of ACS. As expected, the in-hospital mortality of CS individuals was significantly higher compared to non-CS patients (non-CS: 3.7 % vs. CS: 21.3 %; $p<0.001$; see Fig. 1). Notably, after survival of the initial phase, there was no association of CS on long-term mortality after a median follow-up time of 9 years (non-CS: 23.5 % vs. CS: 24.0 %; $p=0.923$). We observed balanced rates of cardiovascular deaths between both groups with an adjusted hazard ratio (HR) of 1.18 (95 % CI: 0.77–1.81; $p=0.455$; see Table 1). CS patients ≥55 years ($p=0.021$), individuals presenting with severely impaired left ventricular function (LVF; $p=0.048$) and chronic kidney disease (CKD; $p=0.013$) after ACS had an increased risk of experiencing a fatal CV event during long-term follow-up.

Conclusion: In line with pre-existing evidence, patients presenting with CS during ACS showed significantly increased in-hospital mortality rates compared to non-CS patients. The present investigation extends, however, currently available evidence that, if CS individuals survived the acute phase of ACS, rates for fatal cardiovascular events were similar to those observed in patients free of CS. Considering an individualized secondary prevention after ACS complicated by CS, patients over 55 years that present with impaired LVF and CKD during the acute phase seem to be at increased risk for fatal events from a long-term perspective and could therefore potentially benefit from intensified follow-up measures.

Innate reverse remodeling reveals novel treatment option for heart failure

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Introduction: Heart failure represents a severe global socio-economic health burden. Contractile cardiomyocytes are replaced by dysfunctional scar tissue. Subsequent remodeling of the myocardium results in change of ventricular geometry and impairment of cardiac function. Current treatment strategies provide symptomatic relieve and at best stop of disease progression. However, there are no treatment options available to regenerate failing myocardium via reverse remodeling. The right (RV) and the left ventricle (LV) differ markedly in their anatomy, function and capability of reverse remodeling. The RV is able to reverse remodel due to a preserved anti-fibrotic mechanism necessary for physiological postnatal adaptation. We aimed to (a) identify the conserved mechanisms of innate reverse remodeling of the RV and thus (b) reveal therapeutic strategies for reverse remodeling of the LV.

Methods: LV and RV heart failure were induced using absorbable sutures in a murine transaortic constriction (TAC) or pulmonary artery banding (PAB) procedure. Sutures were absorbed after 2 weeks, mimicking afterload relieve. RV and LV function and mass were evaluated weekly via transthoracic echocardiography. Cardiomyocyte size, myocardial thickness and myocardial fibrosis were analyzed in histological sections. 1 week after afterload relieve, RNA sequencing was performed to determine genes involved in the reverse remodeling of the RV. Identified genes were analyzed via siRNA knock down in functional cell culture assays. Overexpression and knockdown of identified genes was performed in vivo. Adaption of the RV postpartum is analyzed via µCT, histological sections and qPCR.

Results: In a murine model of reversible heart failure, we observed reverse remodeling of the RV without fibrosis in contrary to LV. RNA sequencing of the regenerating RV revealed the undescribed gene KIAA0408 as possible underlying cause. In vitro an anti-fibrotic effect of KIAA0408 via the JNK/ELK-1/SRF axis was fund. In contrast to the LV, RV myocardial mass decreased from day 1–day 3 postpartum upon afterload relief. In line, cardiomyocyte size decreased in the RV on day 3 postpartum. No signs of fibrosis were observed in the same time period. In the adaption of the heart postpartum increased levels of KIAA0408 could be observed in the physiological reverse remodeling RV. Therapeutic application of KIAA0408 reduced fibrosis and heart failure.

Conclusion: Our data suggest a conserved postnatal mechanism behind the regenerative capacity of the RV. We reveal the undescribed gene KIAA 0408 as potential anti-fibrotic agent to treat heart failure.

Hippo/YAP/TAZ mediates angiogenic response and exosome release upon SWT

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Introduction: Shockwave Therapy (SWT) has been shown to induce tissue heart regeneration via (a) the release of angiogenic exosomes and (b) stimulating innate immune receptor TLR3. Hippo/YAP/TAZ is a crucial mechanosensing pathway mediating cardiac regeneration by stimulating the TLR-IFN pathway via exosome release. We therefore hypothesized that the mechanical stimulation of SWT causes the release of TLR3-activating exosomes by activating the Hippo/YAP/TAZ pathway.

Methods: In order to investigate the detailed underlying mechanisms, human umbilical vein endothelial cells were stimulated with 300 impulses at a frequency of 3 Hz and an energy flux density of 0.1mJ/mm2. Four hours thereafter, mRNA expression of YAP/TAZ target genes (ANKRD1, CYR61) was measured and the nuclear localization of YAP/TAZ was examined by immunofluorescence. A wound healing assay, a tube formation assay and proliferation were analyzed upon SWT in the presence of Hippo/YAP/TAZ stimulation or inhibition. The cell culture supernatant was collected. The release of Extracellular Vesicles (EVs) was characterized by flow cytometry to detect bigger EVs using annexin V (Anx5) as a marker of phosphatidylserine (PS) expressing EVs. Furthermore, EVs were analyzed by a bead-based flow cytometry assay to detect smaller EVs by using CD63-coupled magnetic beads. The particle concentration was measured by nanoparticle tracking analysis.

Results: SWT of HUVECs resulted in a higher concentration of Anx5+ EVs (12,675 ±2863 vs.8650 ±1614 EVs/µl) in the culture supernatant as compared to the untreated control. This observation was confirmed by a higher percentage of EV-decorated beads after SWT. This was accompanied by higher mRNA expression of YAP/TAZ target genes ANKRD1 ($p=0.0005$, respectively) and CYR61 ($p=0.0006$, respectively). Immunofluorescence staining showed a nuclear translocation of YAP/TAZ

upon SWT compared to untreated controls. These effects were abolished upon pharmacological inhibition of YAP/TAZ nuclear translocation.

Conclusion: SWT activates Hippo/YAP/TAZ with concomitant downstream signaling. Hippo/YAP/TAZ activation upon SWT induces exosome release. The Hippo/YAP/TAZ pathway plays a crucial role in the mechanotransduction of SWT and represents a regenerative approach for ischemic heart disease.

Pharmacologic masking of the glycocalyx reduces binding of SARS-CoV-2 spike protein

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Introduction: Corona viruses are able to bind to ACE2 receptors, which are highly expressed in endothelial lung cells, arterial and vein endothelial cells and smooth arterial muscle cells. The binding is mediated via the corona spike protein. Besides direct binding to the receptor, different glycosaminoglycans of the glycocalyx play an important role during the attachment of the virus to the host. Especially, heparin sulfate was identified as major cofactor for binding interactions. Calcium dobesilate (commercial name Doxium), was hypothesized to inhibit or minimize the interaction potential of the corona spike protein with the cellular glycocalyx due to its interaction with the charged chains. Therefore we tested the capability of Doxium to modify the interaction of the coronavirus SARS-CoV-2 spike protein with host cells.

Methods: To determine the effect of Doxium on this general adhesion mechanism, we used human pulmonary microvascular endothelial cells (HPMEC), human umbilical vein endothelial cells (HUVEC), human umbilical vein smooth muscle cells (HUVSMC) and human tracheal epithelial cells (HTEpiC), stimulated with different concentrations of corona spike protein and Doxium. In order to detect inflammatory processes, the supernatant of the stimulated cells was analyzed via ELISA, with the targets PAI, uPA and IL-6. To test the direct in vitro binding of the spike protein to HPMEC, HUVEC, HUVSMC and HTEpiC, we used a His-Tag corona spike protein for short term binding analysis (1–4 h). Binding was quantified by confocal microscopy (LSM700). To identify if the binding process of the spike protein is altered by the lack of certain glycosaminoglycans, different enzymes, (chondroitinase, heparinase and hyaluronidase) were used for enzymatic digestion of specific residues. We hypothesize that the spike protein binding to the glycocalyx is minimized when certain glycosaminoglycans are missing.

Results: The microscopy showed that the corona spike protein was internalized by the cells and is located in/around the nucleus. When cells were incubated with hyaluronidase, there was a significant reduction of attached spike protein. This could be also seen when cells were treated with chrondroitinase but not with heparinase. In the microscopy data we observed a significantly lower fluorescence signal of the spike protein of cells which were treated with Doxium. The effect was observed for HUVEC, HUVSMC and HTEpiC but not in HPMEC. In addition, we invastigated that there is no significant increase of inflammation factors in the cell supernatant of HPMEC, HUVEC, HUVSMC and HTEpiC when treated with concentrations of spike protein that were used in microscopy experiments. Therefore, the spike protein did not induce an inflammation process.

Conclusion: The corona spike protein is able to bind to a cells surface via glycocalyx. This ability can be minimized by enzymatic digestion of the glycocalyx or by the use of Doxium, which we hypothesize leads to a sterical hindrance of spike protein binding. Therefore, glycosaminoglycans play an important role during the attachment and internalization of the virus. By observing a significant reduction of attached spike protein to the cells, we showed that Doxium can minimize the ability of the spike protein to enter the cell via the glycocalyx.

Identification of gene expression signatures for phenotype-specific drug targeting of myocardial fibrosis

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Introduction: Myocardial fibrosis (MF) contributes to the progression to heart failure (HF) and is generated by different pathological mechanisms. We have designed two types of MF by using translational animal models: volume and pressure-overload induced reactive interstitial diffuse MF and replacement diffuse fibrosis by application of cardiotoxic agents. The aim of our postprocessing analyses was to compare the global transcriptomics signatures of animal models of different MF for targeted search for potential treatment approaches.

Methods: Domestic pigs (Sus scrofa) were treated with either doxorubicin (DOX, $n=5$) or a liposomal encapsulation of doxorubicin-citrate complex (Myocet®, MYO, $n=5$) in a human dose to generate cardiotoxicity-induced MF. For pressure overload-induced MF, we used our porcine artificial isthmus stenosis with stepwise developing myocardial hypertrophy and final fibrosis (Hyper, $n=3$). Volume-overload MF was observed in adverse remodeling of the enlarged left ventricle after extensive anterior myocardial infarction (RemoLV, $n=3$). Sham interventions served as controls (Control, $n=3$). Myocardial samples from the anterior wall of groups DOX, MYO, Hyper and Control, and from the non-ischemic remodeled posterior wall of animals in group RemoLV were stored in RNAlater, followed by mRNA isolation and RNA-sequencing for differential gene expression (DGE) analysis. Gene expression profile of each MF model was compared with the LINCS chemical perturbagen signature in the iLINCS database to search for potential drug candidates.

Results: RNA-seq analysis revealed a clear distinction between the transcriptome of different MF models, although the most prominent pathway was TGF-β signaling which was dysregulated across all treatment groups. Application of anticancer drugs activated the TNF-alpha and adrenergic signaling pathways in cardiomyocytes. Several significantly overexpressed genes in DOX and MYO group, such as TNNT1 (coding skeletal TroponinT), ADRB1 (adrenoreceptor beta1), CASP3 (apoptosis signaling), FOS (cell transformation) or KNE3 (potassium ion and voltage-gated channel gene) were either not significantly regulated or downregulated in RemoLV and Hyper groups. Induction of pro-fibrotic processes by pressure- or volume-overload exhibited activation of FoxO pathway components. Drug prediction model confirmed the antifibrotic effect of ACE inhibitors, ARBs and the neprilysin inhibitor (Fig. 1). A significant upregulation of adrenergic signaling and FoxO signaling resulted in the identification of potential new drug candidates (Fig. 2), such as Thiostrepton, that targets the FOXM1-regulated Angiotensin-converting enzyme (ACE) switch. Analysis of the FOXM-1-associated genes revealed significant regulation

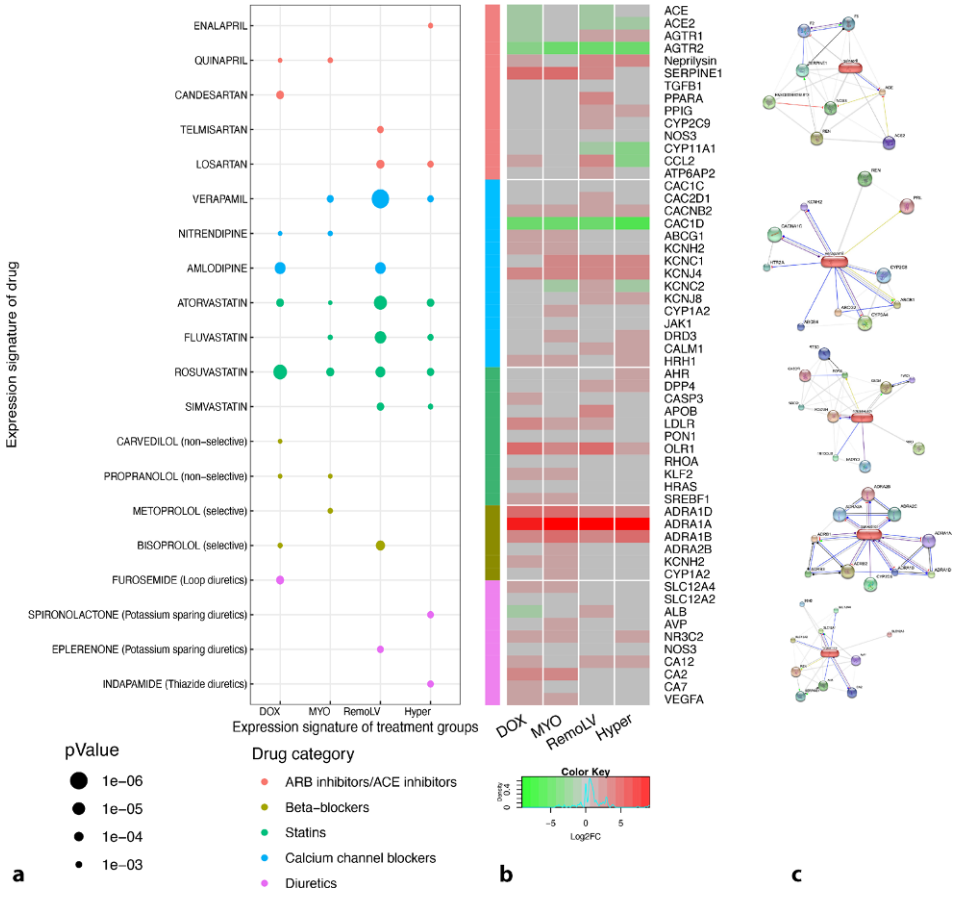

Fig. 1 Identification of gene expression signatures for phenotype-specific drug targeting of myocardial fibrosis Drug prediction based on the gene expression signature data sets

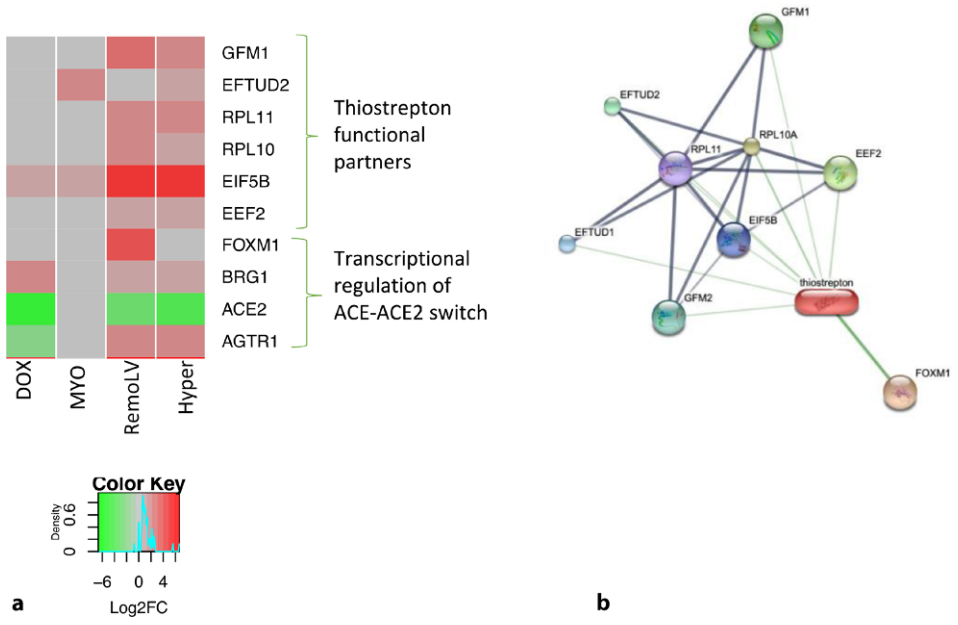

Fig. 2 Identification of gene expression signatures for phenotype-specific drug targeting of myocardial fibrosis Gene expression of Thiostrepton functional interaction partners involved in Angiotensin converting enzyme switch

of FOXM1 interaction partners (GFM, EFTUD2, RPL11, RPL10, EIF5B, EEF2) in RemoLV and Hyper groups.

Conclusion: Our study identified different molecular pathways involved in the development of distinct MF in a porcine model leading to HF. A new antifibrotic drug Thiostrepton might exhibit beneficial effect in reduction of cardiac fibrosis via ACE-regulation especially in pressure and volume overload-induced MF model.

Proteomic profiling reveals a critical role of the complement system in stent thrombosis

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Introduction: Stent thrombosis (ST) is a severe complication after primary percutaneous coronary intervention (pPCI) and associated with significant morbidity and mortality. Apart from procedure- and lesion-related parameters and patient-related factors. However, the underlying molecular and cellular mechanisms of ST are still not fully understood. We aimed to perform in-depth proteomic analysis of ST to understand its pathogenesis.

Methods: We recruited 77 patients suffering from ST after pPCI for myocardial infarction (MI). As controls, we included matched patients suffering from native vessel acute myocardial infarction (NT, $n=154$). Five cases of acute ST (within 24 h) and six cases of NT thrombi aspirated from the culprit site were subjected to shotgun proteomic analysis. Gene-set analysis was employed to screen for pathways differing between ST and NT. Soluble complement factor (C)5a was measured from both coronary culprit site plasma and femoral plasma as in-patient control. All-cause mortality was assessed using Kaplan-Meier, ROC analysis and multivariable Cox regression.

Results: 9 patients presented with acute ST (<24 h, 11.7 %), 18 patients with subacute ST (24 h to 30 days, 23.4 %), 11 patients with late ST (30 days to 1 year, 14.3 %) and 39 patients with very late ST (>1 year, 50.6 %). ST was associated with increased all-cause mortality compared to NT (mean survival 129 vs. 109 months, log-rank $p=0.032$). Using proteomics, we identified a total of 2438 proteins to be expressed in both ST and NT thrombi. Gene set analysis revealed the complement system to be highly active in acute ST compared to NT. Specifically, we found factors of both the classical (complement factor [C]1q, C1s) and alternative pathway (complement factor B) to be increased in ST, along with higher levels of C2, C3, C4a, C4b, C5, C8a and C9. Employing ELISA, we found C5a levels to be increased at the culprit site of ST, but not of NT, patients. ROC analysis yielded a culprit site C5a level of 14,604.59 ng/ml to predict all-cause mortality (ROC AUC 0.76 [0.62, 0.90], $p<0.0001$; sensitivity 72.2 %, specificity 74.3 %). Using this cut-off, C5a levels independently all-cause mortality in ST (adjusted HR 4.102, 95 % CI 1.293–13.009, $p=0.017$) but not in NT patients (adjusted HR 0.645, 95 % CI 0.256–1.622, $p=0.351$).

Conclusion: This hypothesis-generating study highlights a crucial role of the complement system in the pathogenesis of acute ST. Further studies are required to validate these findings in a larger cohort.

Featured Poster Session: Beste Poster 1:

Impact of myocardial injury after coronary artery bypass grafting on long-term prognosis

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Introduction: The most appropriate definition of perioperative myocardial infarction (pMI) after coronary artery bypass grafting (CABG) and its impact on clinically relevant long-term events is controversial. We aimed to (i) analyse the incidence of pMI depending on various current definitions in a 'real-life' setting of CABG surgery and (ii) determine the long-term prognosis of patients with pMI depending on current definitions.

Methods: A consecutive cohort of 2829 coronary artery disease patients undergoing CABG from two tertiary university centers with the presence of serial perioperative cardiac biomarker measurements (cardiac troponin and creatine kinase-myocardial band) were retrospectively analysed. The incidence and prognostic impact of pMI were assessed according to (i) the 4th Universal Definition of Myocardial Infarction (4UD), (ii) the definition of the Society for Cardiovascular Angiography and Interventions (SCAI), and (iii) the Academic Research Consortium (ARC). The primary endpoint of this study was a composite of myocardial infarction, all-cause death, and repeat revascularization; secondary endpoints were mortality at 30 days and during 5-year follow-up.

Results: There was a significant difference in the occurrence of pMI (49.5 % SCAI vs. 2.9 % 4UD vs. 2.6 % ARC). The 4th Universal Definition of Myocardial Infarction and ARC criteria remained strong independent predictors of all-cause mortality at 30 days [4UD: odds ratio (OR) 12.18; 95 % confidence interval (CI) 5.00–29.67; P: 0.001; ARC: OR 13.16; 95 % CI 5.41–32.00; P: 0.001] and 5 years [4UD: hazard ratio (HR) 2.13; 95 % CI 1.19–3.81; $P=0.011$; ARC: HR 2.23; 95 % CI 1.21–4.09; $P=0.010$]. Moreover, the occurrence of new perioperative electrocardiographic changes was prognostic of both primary and secondary endpoints.

Conclusion: Incidence and prognosis of pMI differ markedly depending on the underlying definition of myocardial infarction for patients undergoing CABG. Isolated biomarker release-based definitions (such as troponin) were not associated with pMI relevant to prognosis. Additional signs of ischemia detected by new electrocardiographic abnormalities, regional wall motion abnormalities, or coronary angiography should result in rapid action in everyday clinical practice.

Eligibility for HFpEF/HFmrEF outcome trials and mortality risk in a cohort of decompensated heart failure patients

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Introduction: Heart failure with preserved (HFpEF) and mildly reduced ejection fraction (HFmrEF) account for more than half of heart failure decompensations. Probably as a consequence to increasingly selective eligibility criteria, randomized controlled trials partly reported low numbers of included patients per participating centre. Moreover, previous studies indicated that trial populations may differ from real-world populations in terms of baseline characteristics and outcomes [1]. We aimed to test the hypothesis that patients eligible for trial participation differ significantly from non-eligible patients with regard to mortality risk.

Methods: We performed a systematic retrospective medical records review of all patients presenting in the internal emergency unit of a tertiary care centre between August 2018 and July 2019 ($n=32{,}028$). In accordance with international heart failure guidelines, we identified 554 patients with decompensated heart failure and a left ventricular ejection fraction (LVEF) above 40 %. We excluded 146 patients who fulfilled general trial exclusion criteria (dementia, nursing home residency, palliative care setting). For the remaining patients, we serially applied major in- and exclusion criteria of CHARM-Preserved, I-PRESERVE, TOPCAT, PARAGON-HF and EMPEROR-Preserved, respectively. All-cause mortality data were collected from the national death registry.

Results: The selected cohort ($n=407$) included 212 females (52 %), mean age was 78 ± 10 years. Median (interquartile range) NT-proBNP was 3092 (1554–6948) pg/ml and mean eGFR was 49 ± 20 ml/min/m^2. During median follow-up of 20 months, death was reported in 119 patients (29 %). When applying in- and exclusion criteria, eligibility rates decreased from 92 % (only inclusion criteria) to 81 % (both inclusion and exclusion criteria) for CHARM-Preserved, from 83–45 % for I-PRESERVE, from 69–40 % for TOPCAT, from 73–34 % for PARAGON-HF, and from 76–55 % for EMPEROR-Preserved. Mortality rates among eligible patients decreased from 28 % (only inclusion criteria) to 25 % (both inclusion and exclusion criteria) for CHARM-Preserved, from 29–17 % for I-PRESERVE, from 25–18 % for TOPCAT, from 30–18 % for PARAGON-HF, and from 30–22 % for EMPEROR-Preserved. Non-eligibility was significantly associated with an increased mortality risk in CHARM-Preserved (HR 1.98 [95 % CI 1.34–2.94]), I-PRESERVE (HR 2.70 [1.79–4.07]), TOPCAT (HR 2.24 [1.48–3.39]), PARAGON-HF (HR 2.24 [1.43–3.50]), and EMPEROR-Preserved (HR 1.87 [1.30–2.69]) (Figures).

Conclusion: Out data indicate that previous HFpEF/HFmrEF outcome trials may have systematically excluded patients at the highest mortality risk. Generalization of trial results, but also of post-hoc analyses performed in trial cohorts, should be performed with caution.

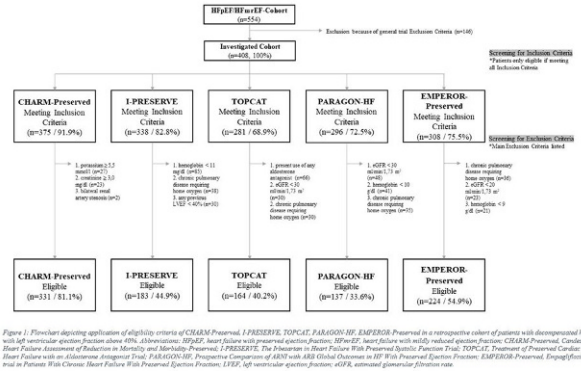

Figure 1: Flowchart depicting application of eligibility criteria of CHARM-Preserved, I-PRESERVE, TOPCAT, PARAGON-HF, EMPEROR-Preserved in a retrospective cohort of patients with decompensated heart failure with left ventricular ejection fraction above 40%. Abbreviations: HFpEF, heart failure with preserved ejection fraction; HFmrEF, heart failure with mildly reduced ejection fraction; CHARM-Preserved, Candesartan in Heart Failure Assessment of Reduction in Mortality and Morbidity-Preserved; I-PRESERVE, The Irbesartan in Heart Failure With Preserved Systolic Function Trial; TOPCAT, Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist Trial; PARAGON-HF, Prospective Comparison of ARNI with ARB Global Outcomes in HF With Preserved Ejection Fraction; EMPEROR-Preserved, Empagliflozin outcome trial in Patients With Chronic Heart Failure With Preserved Ejection Fraction; LVEF, left ventricular ejection fraction; eGFR, estimated glomerular filtration rate.

Fig. 1 Eligibility for HFpEF/HFmrEF outcome trials and mortality risk in a cohort of decompensated heart failure patients Flowchart depicting eligility rates for HFpEF/HFmrEF trials

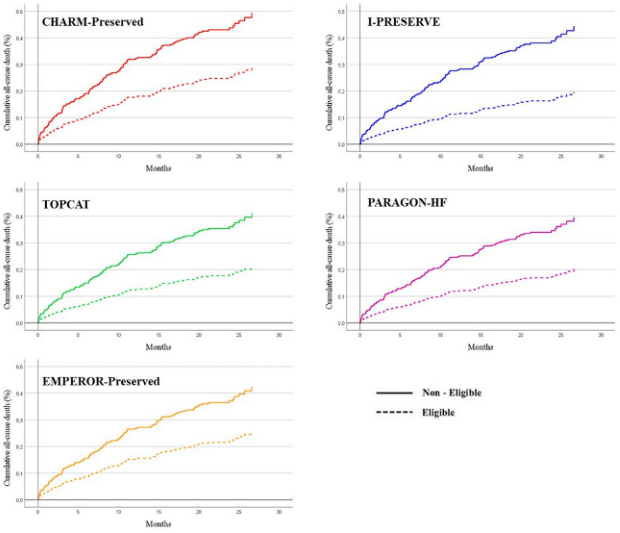

Figure 2: Estimates of cumulative incidence of all-cause death stratified by trial eligibility. Abbreviations: CHARM-Preserved, Candesartan in Heart Failure Assessment of Reduction in Mortality and Morbidity-Preserved; I-PRESERVE, The Irbesartan in Heart Failure With Preserved Systolic Function Trial; TOPCAT, Treatment of Preserved Cardiac Function Heart Failure with an Aldosterone Antagonist Trial; PARAGON-HF, Prospective Comparison of ARNI with ARB Global Outcomes in HF With Preserved Ejection Fraction; EMPEROR-Preserved, Empagliflozin outcome trial in Patients With Chronic Heart Failure With Preserved Ejection Fraction.

Fig. 2 Eligibility for HFpEF/HFmrEF outcome trials and mortality risk in a cohort of decompensated heart failure patients Estimates of cumulated incidence of all-cause death stratified by trial eligibility

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Standardized measurement of abdominal muscle by computed tomography: Association with cardiometabolic risk in the Framingham Heart Study

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Introduction: Muscle mass on computed tomography (CT) has been linked with cardiorespiratory fitness in population-based studies and mortality in patients with chronic disease. However, no standardized method has been established and the role of total abdominal muscle mass (TAM) in a primary prevention setting is unknown. The aim of the study was to establish a standardized method for TAM quantification on CT and assess its association with cardiometabolic risk.

Methods: We included 3016 Framingham Heart Study participants free of cardiovascular disease (CVD) who underwent abdominal CT between 2002 and 2005. On a single CT slice at the level of L3/L4 we segmented (1) TAM-Area, (2) TAM-Index (=TAM-Area/height2) and, (3) TAM-Fraction (=TAM-Area/total cross-sectional CT area). The association of these muscle mass measures with prevalent and incident cardiometabolic risk factors, as well as CVD events during a follow-up of 11.0 ± 2.7 years, are reported.

Results: In this community-based sample (49 % women, mean age: 50.0 ± 10.0 years) all muscle quantity measures were significantly associated with prevalent and incident cardiometabolic risk factors and CVD events in age and sex-adjusted models. However, only TAM-Fraction remained significantly associated with key outcomes (e. g. adj. OR 0.68 [0.55, 0.84] and HR 0.73 [0.57, 0.92] for incident hypertension and CVD events, respectively) after adjustment for body mass index and waist circumference. Moreover, the directionality of association in that less muscle was associated with higher risk was only seen for TAM-F (e. g.: TAM-Fraction: adj. OR: 0.56[0.36–0.89] for incident diabetes versus TAM-Area: adj.OR 1.26 [0.79–2.01] and TAM-Index: 1.09 [0.75–1.58]).

Conclusion: These data suggest that total abdominal muscle expressed as the fraction of total cross-sectional body area at L3/L4 is a novel body composition marker of cardiometabolic risk in a primary prevention setting that has the potential to improve risk stratification beyond traditional measures of obesity.

Hairdresser evaluation to improve diagnostic management in hypertension in primary care–Friseurinitiative Ottakring „Keine Therapie ohne Diagnose"

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Introduction: Arterial hypertension (HTN) is the most important preventable cardiovascular risk factor for overall mortality worldwide [1]. In Austria, 1.6 millions of people above 15 years suffer from HTN [2]. Only 41 % of patients in Austria have their HTN controlled [3]. In Europe, average blood pressure (BP) values are obvious above the target values. Despite widespread medical antihypertensive treatment options on the European market only 38.8 % of patients on OMT reach the target values. Whereas 72.7 % of 7642 patients ≥50 years suffer HTN in 12 European countries [1]. HTN is a well-known "silent killer", because of its asymptomatic nature in the early stage of the disease. However, long-term issues are atherosclerosis and arteriosclerosis leading to end organ damage with severe sec-

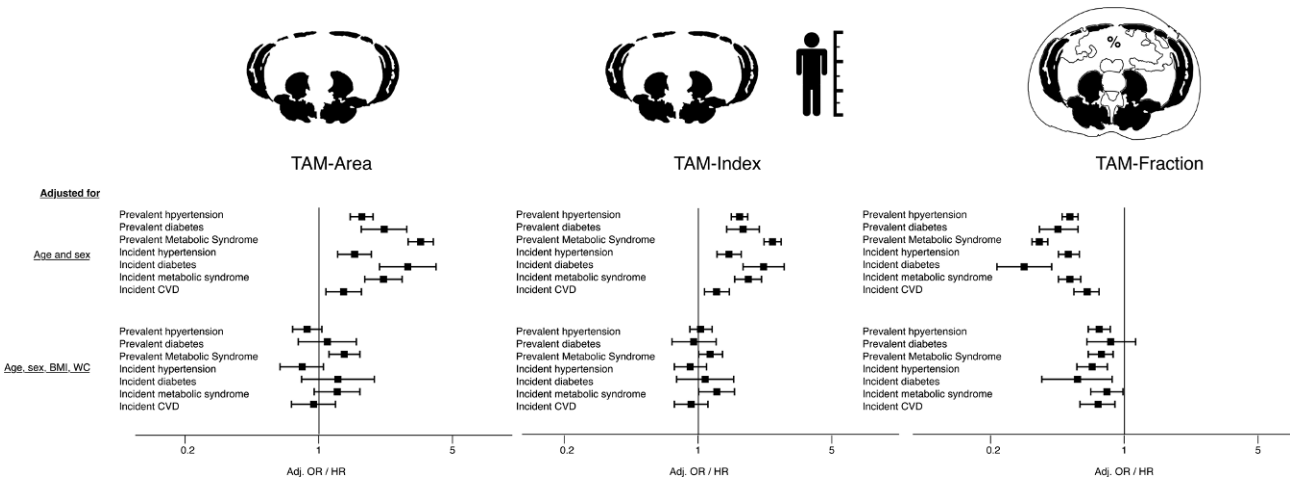

Fig. 1 Standardized measurement of abdominal muscle by computed tomography: Association with cardiometabolic risk in the Framingham Heart Study Association of total abdominal muscle area, index, and fraction (TAM-Area, TAM-Index, and TAM-Fraction) with prevalent and incident cardiometabolic risk factors and cardiovascular disease (CVD) events (Odds and hazard ratios are given per 1-SD increase of TAM-Area, TAM-Index, and TAM-Fraction respectively)

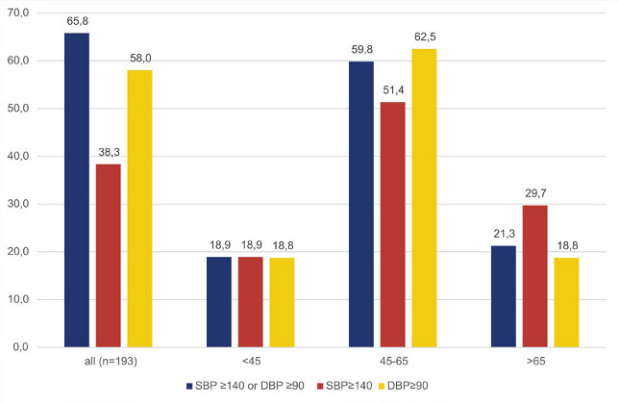

Fig. 1 Hairdresser evaluation to improve diagnostic management in hypertension in primary care–Friseurinitiative Ottakring „Keine Therapie ohne Diagnose“ Blood Pressure and Age

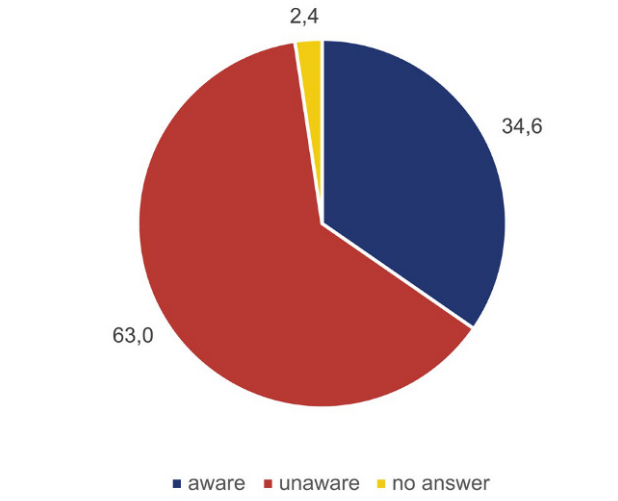

Fig. 2 Hairdresser evaluation to improve diagnostic management in hypertension in primary care–Friseurinitiative Ottakring „Keine Therapie ohne Diagnose“ Awareness

ondary consequences such as stroke, coronary artery disease, heart failure, renal insufficiency and peripheral artery occlusive disease [4]. Living in socioeconomic well-being states suffering affluenza, there is an urgent need for Disease Management Programs (DMP).

Methods: The Friseurinitiative Ottakring “Keine Therapie ohne Diagnose” represents a trial to diagnose hypertension in a non-medical setting at a very early stage, raise awareness for hypertension in affected people and avoid secondary diseases. For a non-medical setting hairdresser accomplish all criteria for an optimal blood pressure (BP) measurement. The staff received expert training on how to measure blood pressure in a guideline compliant way. All members of the study received a questionnaire about their demographic data and cardiovascular risk factors.

Results: 193 people participated in this study, 56.5 % female and 43.5 % male persons. The mean age was 54 ±15.1 years. In the first automatic measured office blood pressure (AOBP) measurement the mean systolic BP was 137.1 ±17.8, the mean diastolic BP was 91.6 ±11.2. 65.8 % had a SBP ≥140 mm Hg or DBP ≥90 mm Hg whereof 74.8 % have no treatment at all. 63 % are unaware of their elevated BP values. 28 % are already diagnosed with HTN whereof 18.5 % [10.4; 30.9] have normotensive values in the current measurement. 20.2 % are taking medication against HTN, and 62.2 % didn’t take their BP medication on the day of recruitment.

Conclusion: In the study, we could confirm that screening for HTN in an unconventional setting is effective to diagnose HTN and raise awareness. An implementation of such a cost-effective and feasible disease management program in Austria might therefore reduce the burden of preventable cardiovascular events associated with HTN. It requires urgent need for action. An implementation of a cost-effective and feasible disease management program might therefore reduce the burden of preventable cardiovascular events associated with HTN.

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Quantification of systolic anterior motion of the mitral valve to identify left ventricular outflow tract obstruction in patients with hypertrophic cardiomyopathy

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Introduction: In patients with hypertrophic cardiomyopathy (HCM), left ventricular outflow tract obstruction (LVOTO) is associated with an increased risk of heart failure and death [1]. It is typically caused by dynamic systolic anterior motion (SAM) of the mitral valve leaflet [2]. SAM can be categorized by echocardiography but diagnostic accuracy is limited by high inter-observer variability. We aimed to investigate the accuracy of echocardiographic parameters quantifying systolic motion of the mitral valve leaflets to identify LVOTO in patients with HCM.

Methods: We present a cross-sectional analysis of the HyperCard Registry, a prospective single-center cohort study enrolling consecutive patients with suspected or confirmed HCM. For the present analysis, patients with confirmed HCM and a valid standardized transthoracic echocardiographic were included. LVOT gradients were measured at rest and during Valsalva maneuver using continuous wave Doppler. In patients with clinical suspicion of dynamic LVOTO, further provocation maneuvers were conducted. LVOTO was defined as a maximal peak LVOT gradient ≥30 mm Hg. Parameters quantifying systolic motion of the mitral valve were measured in parasternal and apical views, both at early and late systole, by an investiga-

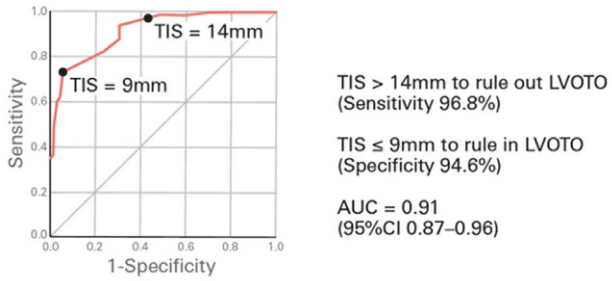

Fig. 1 Quantification of systolic anterior motion of the mitral valve to identify left ventricular outflow tract obstruction in patients with hypertrophic cardiomyopathy Receiver-operating characteristic curve analysis depicting the diagnostic accuracy of the distance from mitral valve tip to ventricular septum at late-systole (TISls) measured in apical three chamber view to identify the presence of left ventricular outflow tract obstruction in patients with hypertrophic cardiomyopathy (AUC area under the curve)

tor blinded to individual patient characteristics. SAM was visually assessed and categorized into grade 0 (no SAM), I (leaflet motion towards LVOT), II (late systolic septal contact), and III (early systolic septal contact).

Results: We analyzed 142 patients (59 ±13 years, 42 % women). LVOTO was present in 68 (48 %) patients of whom 30 (21 % of all) exhibited LVOTO at rest, and 38 (27 % of all) had LVOTO only during provocation maneuvers (ie. dynamic LVOTO). SAM was present in 86 patients (60 %) and had a sensivitiy, specificity and PPV of LVOTO of 91 %, 69 % and 73 %, respectively (Table 1). The late-systolic distance between mitral leaflet tip and anterior septum (TISls) measured in apical 3-chamber view was best associated with the degree of SAM (F = 123, $P < 0.001$), and with peak LVOT gradient (at rest: Pearson r = –0.817; during Valsalva maneuver: r = –0.816, both $P < 0.001$). In ROC analyses (Fig. 1), the AUC of TISls for identification of LVOTO and dynamic LVOTO were 0.914 (95 % CI 0.868–0.959) and 0.857 (0.786–0.927), respectively. TISls ≤14 mm had a 97 % sensitivity for LVOTO and of 94 % for dynamic LVOTO. TISls ≤9 mm showed specificity and PPV of 95 % and 92 % for LVOTO, and 94 % and 83 % for dynamic LVOTO, respectively.

Conclusion: Quantification of SAM by TISls showed high diagnostic accuracy in identifying HCM patients with LVOTO. Prospective studies are needed to assess the incremental benefit of this novel parameter in the diagnostic work-up of HCM patients.

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Table 1 Quantification of systolic anterior motion of the mitral valve to identify left ventricular outflow tract obstruction in patients with hypertrophic cardiomyopathy Diagnostic accuracy of SAM and of TIS cut-offs for identification of left ventricular outflow tract obstruction

SAM							
	no LVOTO	LVOTO	Sum	Sensitivity	Specificity	PPV	NPV
No SAM	51 (69 %)	6 (9 %)	57	91.2 %	68.9 %	72.9 %	89.5 %
SAM	23 (31 %)	62 (91 %)	85				
Sum	74	68	142				
TISls distance							
	no LVOTO	LVOTO					
>9 mm	70 (95 %)	17 (27 %)	87	73.0 %	94.6 %	92 %	80.5 %
≤9 mm	4 (5 %)	46 (73 %)	50				
Sum	74	63	137				
>14 mm	42 (57 %)	2 (3 %)	44	96.8 %	56.8 %	65.6 %	95.4 %
≤14 mm	32 (43 %)	61 (97 %)	93				
Sum	74	63	137				

SAM systolic anterior motion of the mitral valve; *LVOTO* left ventricular outflow tract obstruction; *PPV* positive predictive value; *NPV* negative predictive value; *TISls* late sytolic mitral leaflet tip to septum distance

Detection of sympathetic denervation defects in Fabry disease by hybrid [11C]meta-hydroxyephedrine positron emission tomography and cardiac magnetic resonance

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Introduction: Cardiac involvement, characterized by glycosphingolipid accumulation, resulting in left ventricular hypertrophy (LVH) and myocardial fibrosis, occurs frequently in patients with Fabry disease. Consequently, lowered T1-relaxation times, and late gadolinium enhancement (LGE) are typical features in cardiac magnetic resonance (CMR) examinations. However, there are still gaps in knowledge about the biochemical and structural mechanisms triggered by glycosphingolipid accumulation in affected cells and organs. This study aimed to non-invasively investigate the sympathetic innervation in patients with Fabry disease, using hybrid cardiac positron emission tomography (PET)/magnetic resonance (MR) with [11C] meta-hydroxyephedrine ([11C]mHED).

Methods: Patients with different stages of Fabry disease (no LVH, LVH without LGE, and patients with LVH and LGE) were prospectively enrolled to undergo a standardized CMR protocol on a 1.5T MRI, followed by 3T hybrid PET/MR with [11C]mHED.

Results: Sixteen patients (10 females, six males) with either no evidence of cardiac involvement ($n=5$), evidence of LVH ($n=5$), or evidence for LVH and fibrosis by LGE ($n=6$) were included, of whom a total of 224 myocardial segments were analyzed. Compared to patients without LVH, patients with LVH or LVH and LGE had lower median T1 relaxation times (ms) at 1.5T (1007 vs. 918 vs. 941, $p=0.025$). Myocardial denervation was prevalent only in the group of patients with fibrosis, where there was a total of 16 denervated segments ([11C]mHED retention <7 %/min). Furthermore, overall [11C]mHED retention was lower in segments of patients with LGE compared to both other groups ($p<0.001$). The respective area of denervation exceeded the area of LGE (from 24 % vs. 36 % to 4 % vs. 32 %). Furthermore, we found correlations of reduced sympathetic innervation with left ventricular mass ($p=0.034$, rs = –0.57) and with an impaired left ventricular function, measured by global longitudinal strain (GLS) ($p=0.023$, rs = –0.6).

Conclusion: Sympathetic denervation, accompanied by impaired GLS, is present in patients with advanced stages of Fabry disease showing fibrosis in CMR. As there may be a higher risk for malignant arrhythmia, these patients should be monitored closely and may be considered for specific forms of antiarrhythmic therapy.

Mitral annular disjunction in patients undergoing cardio-pulmonary resuscitation–A retrospective MRI study

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Introduction: Mitral annular disjunction (MAD), representing the defective attachment of the mitral annulus to the ventricular myocardium, has recently been linked to malignant arrhythmias. However, its significance in patients requiring cardio-pulmonary resuscitation (CPR) remains largely unknown. This retrospective analysis investigates the prevalence and significance of MAD, as assessed by cardiac magnetic resonance imaging (CMR), in out-of-hospital cardiac arrest (OHCA) patients.

Methods: Eighty-six patients with OHCA and a CMR scan 5 days after CPR (interquartile range (IQR): 49 days before–9 days after) were consecutively enrolled. MAD was defined as disjunction-extent ≥1 mm in CMR long-axis cine-images. Medical records were screened for laboratory parameters, comorbidities and history of arrhythmic events.

Results: In 34 patients (40 %), no underlying cause for OHCA was found during hospitalization despite profound diagnostics. Unknown-cause CPR-patients showed a higher prevalence of MAD compared to patients with a definite cause of cardiac arrest (56 % vs. 10 %, $p<0.001$) and had a MAD-extent of 6.3 mm (interquartile range: 4.4–10.3); moreover, these patients

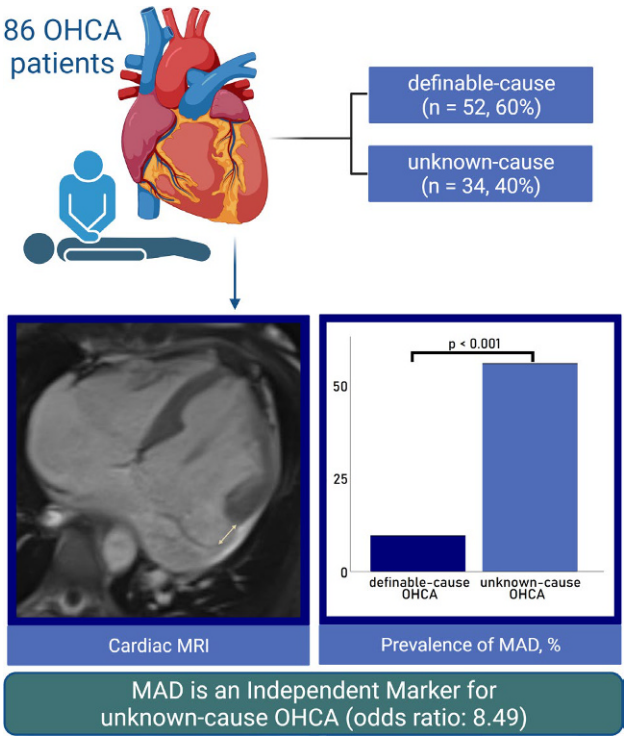

Fig. 1 Mitral annular disjunction in patients undergoing cardio-pulmonary resuscitation–A retrospective MRI study In a cohort of out-of-hospital cardiac arrest (OHCA) patients, mitral annular disjunction (MAD) was significantly more common in unknown-cause OHCA patients

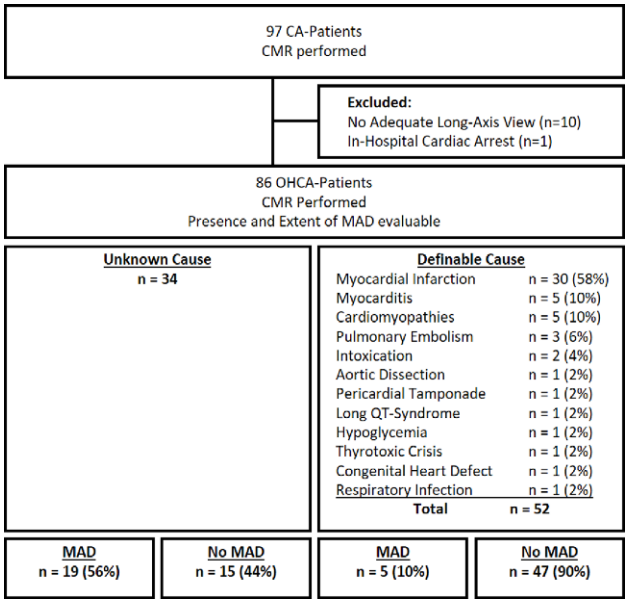

Fig. 2 Mitral annular disjunction in patients undergoing cardio-pulmonary resuscitation–A retrospective MRI study Flowchart of patient in- and exclusion, displaying the particular causes for cardiac arrest

were significantly younger (43 years vs. 61 years, $p < 0.001$), more often female (74 % vs. 21 %, $p < 0.001$) and showed less comorbidities (hypertension, hypercholesterolemia, coronary artery disease (CAD), all $p < 0.005$). By logistic regression analysis, presence of MAD remained significantly associated to OHCA of unclear cause (odds ratio: 8.49, 95 % confidence interval: 2.37–30.41, $p = 0.001$) after adjustment for age and presence of hypertension and hypercholesterolemia.

Conclusion: MAD is present in more than 50 % of patients with OHCA and no definitive aetiology. Presence of MAD remains independently associated to OHCA without identifiable trigger. Further research is needed to understand the exact role of MAD in OHCA patients.

Pregnancy in women with bioprosthetic valves: Re-examining the risks

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Introduction: For women of childbearing age with severe valve disease, valve replacement choice is complex. Bioprosthetic valves (BPV) are typically considered a good option because they are associated with lower rates of complications during pregnancy when compared to mechanical valves. However, structural valve deterioration limits the lifespan of BPV and necessitates reoperation. Information on pregnancy outcomes in women with BPV is based on older studies that do not always discriminate between different BPV types, valve position, or valve function. Therefore, we sought to assess pregnancy outcomes in a large contemporary cohort of women with BPV and examine differences in outcomes according to valve position and valve function.

Methods: Pregnancy outcomes in women with BPV were collected as part of a larger prospective study on pregnancy outcomes in women with heart disease. The first antenatal echocardiograms were used to identify the presence of SVD. BPV were defined according to their position as either left-sided (aortic and/or mitral) or right-sided (pulmonary or tricuspid). BPV in aortic position were subclassified according to the type of the aortic prosthesis: pulmonary autograft (after Ross operation) or bio-

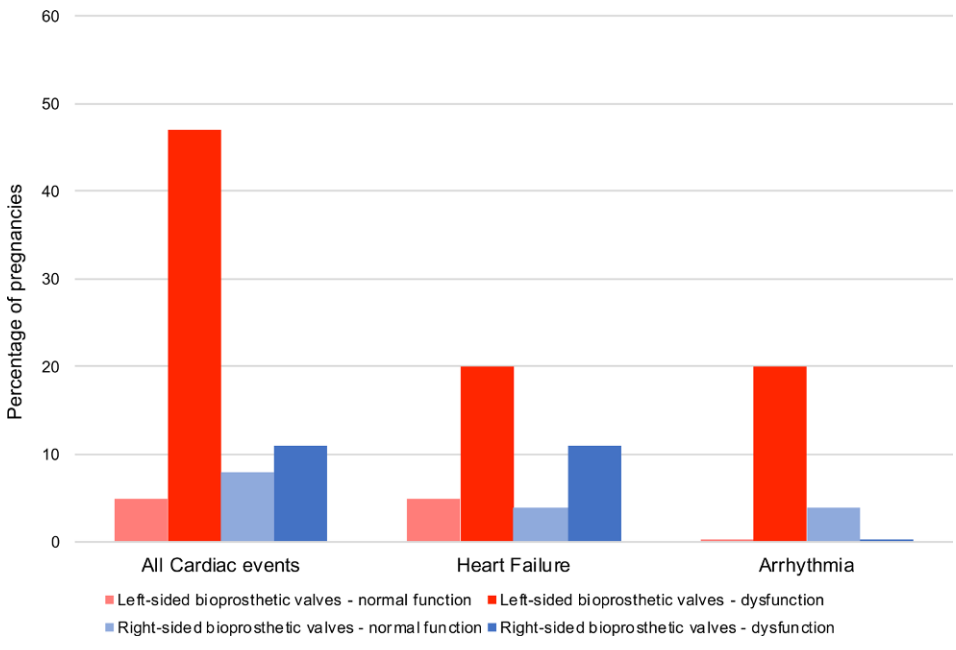

Fig. 1 Pregnancy in women with bioprosthetic valves: Re-examining the risks Cardiac events in left-sided and right-sided bioprosthetic valves with normal function and with structural valve dysfunction

prosthesis (pericardial and porcine xenografts and homografts). Women who had a Ross operation were included in the group of right-sided BPV as the bioprosthetic valve was in the pulmonary position. The time (years) between the most recent valve replacement surgery and the index pregnancy was recorded. Adverse maternal cardiac events (CE) included cardiac death, cardiac arrest, sustained arrhythmia requiring treatment, heart failure, cardiac thromboembolism, and stroke or transient ischemic attack. Maternal outcomes were examined in women with: (a) left-sided and right-sided BPV, and (b) normal valve function and SVD. Predictors of CE were determined using logistic regression.

Results: Overall, 125 pregnancies in 102 women with BPV were included. Thirty-four pregnancies (27 %) occurred in women with left-sided BPV, among whom 17 had aortic valves, 9 had bioprosthetic mitral valves, and 8 had both aortic and mitral valves. Women with right-sided BPV (73 %, $N=91$) primarily had pulmonary valves ($N=86$); five women had tricuspid valves. Twenty pregnancies occurred in 16 women after a Ross operation, with a pulmonary autograft in the aortic position and a BPV in the pulmonary position. SVD was present in 27 % (34/125) of the pregnancies. SVD was more common in women with left-sided (44 %, $n=15/34$) compared to right-sided BPV (21 %, $n=19/91$, $p=0.009$). Notably, only one woman (5 %, $N=1/20$) with a Ross operation had dysfunction of the autograft in the aortic position, whereas 40 % ($N=10/25$) of the aortic BPV were dysfunctional at the first antenatal visit. The time between pregnancy and valve replacement surgery was similar in left-sided (5 ±3 years) and in right-sided BPV (6 ±6 years, $p=0.23$). CE occurred in 13 % (16/125) of pregnancies. In women with left-sided BPV, CE were more common in women with SVD vs. normal functioning BPV (47 % vs. 5 %, $p=0.011$). In contrast, in pregnancies with right-sided BPV, there was no difference in CE rates in those with and without SVD (11 % vs. 8 %, $p=0.67$). Left-sided SVD was an important determinant of CE ($p=0.007$).

Conclusion: In this study, a large number of women followed in our tertiary CardioObstetric clinics had dysfunctional BPV at the time of the first antenatal visit. The risk for adverse maternal cardiac and fetal events was substantially increased in women with SVD of any left-sided BPV, whereas this association was not seen in women with right-sided SVD. This new information needs to be incorporated into decision-making and highlights that the right prosthesis choice for young women with significant left-sided valvular lesions remains difficult. Counseling about the reduced longevity of left-sided bioprosthetic valves and its implications on pregnancy outcomes needs to be included in the discussion.

Featured Poster Session: Beste Poster 2:

Prevalence and mechanisms of cardiac implantable electronic device induced tricuspid regurgitation: A prospective cross sectional echocardiographic study

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Introduction: Cardiac implantable electronic device (CIED) therapy can lead to primary (implantation-related, lead-related) or secondary (pacing related) CIED-induced tricuspid regurgitation (TR) associated with increased morbidity and mortality. The role of the position of the right ventricular (RV) lead in the development of CIED-induced TR is unclear. However, unfavorable RV lead position as well as placement of more than one RV lead may play a major role in the development of novel TR as well as the worsening of preexisting TR. In this prospective cross sectional echocardiographic study we aimed to investigate the prevalence as well as the mechanisms of TR in patients with CIED ([1–3]; Fig. 1).

Methods: Consecutive patients with a history of CIED implantation with at least one RV lead who underwent echocardiography for any cause at our tertiary center were included in this prospective study. Comprehensive transthoracic echocardiographic (TTE) examination was performed according to current guidelines. In addition, a subcostal 2D en-face view of the tricuspid valve (TV) by an approximately 90° counter-clockwise rotation of the transducer from a standard subcostal 4-chamber view was obtained and the position of the RV lead in the tricuspid valve plane (postero-septal/anter-oseptal/antero-posterior commissure, or central position) was determined whenever feasible (Fig. 1).

Results: A total of 70 patients were included in this interim analysis. In this predominantly male (77 %) cohort the median age was 74 years (interquartile range: 62, 79). Indications for CIED were complete heart block, diseases of the sinus node, and cardiomyopathies of different causes. Devices included pacemakers (PM) (36/70, 51 %), implantable cardioverter-defibrillators (ICD) (18/70, 26 %), cardiac resynchronization therapy (CRT) (2/70, 3 %) as well as CRT-defibrillators (14/70, 20 %). Period of time from implantation to inclusion into the study spanned from few days to 33 years. Five patients (7 %) had received two or more RV leads. Fifty-two patients (74 %) showed no or only mild TR. In 15 patients (21 %) moderate TR could be found, while 3 patients (4 %) presented with severe TR. By obtaining the subcostal 2D en-face view of the TV, exact RV lead position in respect to the TV plane could be determined in 47/70 patients (67 %). A postero-septal passage through the TV could be seen in 25/47 (53 %) of cases. Thirty-six percent (17/47) of RV leads passed the TV in a central position. An antero-posterior trajectory could be observed three times, while in only two patients the RV lead was found in an antero-septal position. Failure to determine lead position from the subcostal 2D

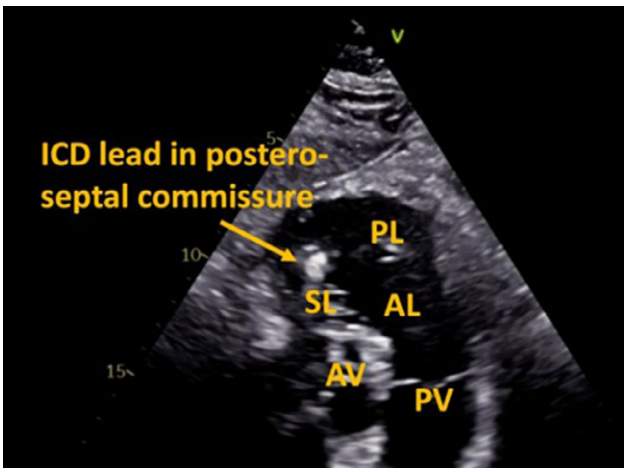

Fig. 1 Prevalence and mechanisms of cardiac implantable electronic device induced tricuspid regurgitation: A prospective cross sectional echocardiographic study

en-face view in 23/70 (33 %) patients was mostly due to inferior image quality.

Conclusion: In the subgroups of patients with postero-septal vs. non-postero-septal RV lead position 28 % (7/25) vs. 32 % (7/22) showed moderate or greater than moderate TR. Interim statistical analysis showed no significant difference in TI severity between the two subgroups ($p = 0.95$). CONCLUSION At least moderate TR was present in 25 % of patients with CIED in this prospective cross sectional study. Exact description of the lead position in the TV plane was possible in 67 % through a subcostal en-face view of the TV. Our data suggests that lead position in the TV plane does not have an influence on severity of TR.

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Neurofilaments in heart failure–depicting the brain-heart axis

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Introduction: Recent data indicate that the prevalence of cognitive impairment is exceedingly high in patients with heart failure with reduced ejection fraction (HFrEF). Experimental studies have fueled theoretical concerns about neurocognitive side effects of the angiotensin receptor–neprilysin inhibitor (ARNi) sacubitril/valsartan as neprilysin is not only involved in the degradation of vasoactive peptides, but also in the degradation of the amyloid-β (Aβ) peptide in the brain. However, to date, no study could demonstrate cognition- and dementia-related adverse effects following neprilysin inhibition. Recent advances in blood-based tests made it feasible to precisely measure the concentration of neurofilament light chain in plasma (sNfL). sNfl has been found to be altered in patients with neurodegenerative disease, making it a potential biomarker for screening and early detection of cognitive impairment.This study investigated the association of sNfL levels with the severity of disease and prognosis in patients with HFrEF and explored the response of sNfL concentrations to the initiation of ARNi to potentially unmask subclinical effects on cognition-associated pathophysiologic pathways.

Methods: Stable HFrEF patients were prospectively enrolled and clinically followed-up. Laboratory markers including NT-proBNP were assessed. Soluble NfL levels were measured for patients with available plasma samples and a follow-up longer than 2-years by a single-molecule array assay (SIMOA Quanterix, MA, USA). The association of sNfL with heart failure (HF) severity and outcome was assessed. The discriminatory power of sNFL as a biomarker was assessed using Harrell's C-statistic and compared to NT-proBNP. In a subset of 47 patients, sNfL levels were determined before the initiation of ARNi (baseline), and 3 ±2 months (short-term) and 12 ±3 months (long-term) after. sNfL levels were compared between different timepoints.

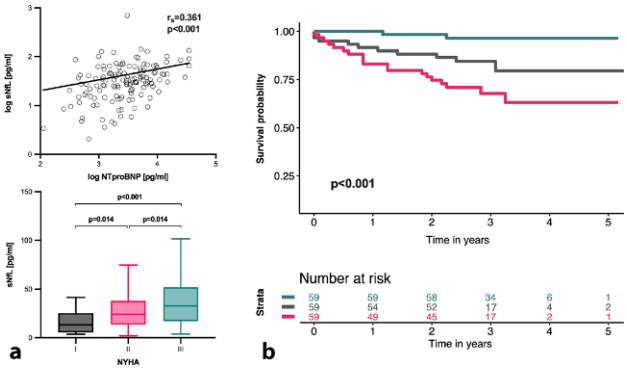

Fig. 1 Neurofilaments in heart failure–depicting the brain-heart axis Relationship of soluble neurofilament light chain (sNfL) with heart failure severity and prognosis. a Scatter plot with linear regression analysis and the Spearman rho coefficient for sNfL and N-terminal pro B-type natriuretic peptide (NT-proBNP) as well as group comparisons between New York Heart Association (NYHA) classes as shown by Tukey-boxplots. b Association of sNfL tertiles with all-cause mortality applying Kaplan-Meier analysis. Comparison was calculated by the log-rank test

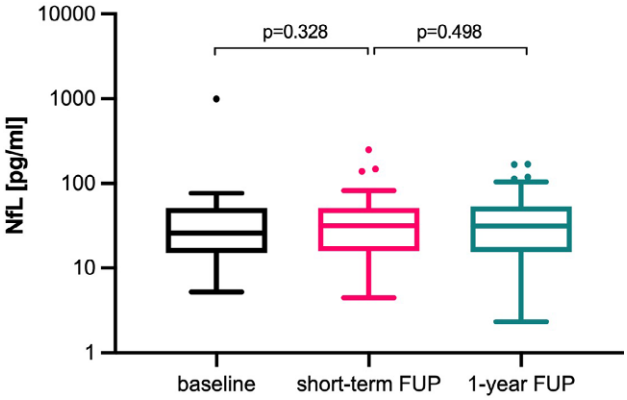

Fig. 2 Neurofilaments in heart failure–depicting the brain-heart axis Baseline, short-term and long-term follow-up (FUP) changes in soluble neurofilament light chain (sNfL) levels after initiation of the angiotensin receptor–neprilysin inhibitor sacubitril/valsartan are shown as Tukey-boxplots. Comparison was calculated by the Wilcoxon signed-rank test

Results: A total of 177 HFrEF patients were included into the study. Median age was 61 years (IQR: 51–72), 75 % were male and median NT-proBNP was 2729 pg/ml (IQR: 1240–5660). Median sNfL levels were 26.2 pg/ml (IQR: 14.1–43.8). sNfL concentration was significantly associated with HF severity reflected by NT-proBNP (rs = 0.361, $p < 0.001$) and NYHA class ($p < 0.001$) (Fig. 1a). A total of 31 (17.5 %) patients died during a median follow-up time of 2.8 years (IQR: 2.3–3.3). Increased sNfL concentration was indicative for worse overall survival even after adjustment for age, sex, kidney function and NT-proBNP [adj. HR for ln[sNfL]: 1.78 (95 % CI: 1.13–2.80, $p = 0.012$)]. Kaplan-Meier analysis illustrates the impact of sNfL levels on outcome

graphically (Fig. 1b). In terms of discriminatory accuracy, sNfl was comparable with NT-proBNP (C-index: 0.75 [CI: 0.67–0.83] vs. 0.71 [CI: 0.63–0.80], $p=0.299$). Regarding the effect of ARNi therapy no significant change in sNfL was observed at short-term and long-term follow-up (26.0 pg/ml [IQR: 15.1–51.4] vs. 31.5 pg/ml [IQR: 15.8–51.3]; vs. 31.3 pg/ml [IQR: 15.5–54.0], $p>0.05$ for all) (Fig. 2).

Conclusion: This study suggests that sNFL is a high performing marker to predict outcome in patients with HFrEF independent of NT-proBNP. NEP inhibition by ARNi does not seem to reasonably influence sNfL levels. The nature of sNfL release is uncertain. As sNfL is assumed to be specific for axonal injury, sNfL increase in HFrEF may be driven by hypoperfusion due to fixed peripheral vasoconstriction characteristic for more severe disease states. Whether sNfL levels are able to aid screening and early therapy of neurocognitive decline in HFrEF has to be elucidated in further studies.

Two-dimensional speckle-tracking echocardiography in Tafamidis-treated patients with transthyretin amyloid cardiomyopathy: A glimmer of hope for viable therapy monitoring?

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Introduction: Treatment with Tafamidis in patients with transthyretin amyloid cardiomyopathy (ATTR-CM) has been shown to have beneficial effects on the left ventricle (LV), as assessed by cardiac magnetic resonance (CMR) imaging. However, in clinical practice, the availability of CMR is limited. Therefore, we aimed to determine Tafamidis-induced changes using advanced transthoracic echocardiography (TTE) and to identify imaging parameters for specific therapy monitoring.

Methods: ATTR-CM patients underwent serial TTE with two-dimensional (2D) speckle-tracking imaging.

Results: Patients receiving Tafamidis 61 mg ($n=62$) or 20 mg ($n=21$) once daily (QD) showed stable measurements at follow-up [61 mg: 8.5 months, 20 mg: 7.0 months] in LV global longitudinal strain (GLS) (61 mg: –11.75 % vs. –11.58 %, $p=0.534$; 20 mg: –10.61 % vs. –10.12 %, $p=0.309$), right ventricular (RV) GLS (61 mg: –14.18 % vs. –13.72 %, $p=0.377$; 20 mg: –14.53 % vs. –13.99 %, $p=0.452$), and left atrial (LA) reservoir strain (LASr; 61 mg: 8.80 % vs. 9.42 %, $p=0.283$; 20 mg: 8.23 % vs. 8.67 %, $p=0.589$), whereas treatment-naïve ATTR-CM patients ($n=54$) had clear signs of disease progression at the end of the observation period [10.5 months; LV-GLS: –11.71 % vs. –10.59 %, $p=0.001$; RV-GLS: –14.36 % vs. –12.99 %, $p=0.038$; LASr: 10.67 % vs. 8.41 %, $p=0.005$]. Between-group comparison at follow-up revealed beneficial effects of Tafamidis 61 mg on LASr ($p=0.003$) and LV strain (IVS: $p=0.006$, LV-GLS: $p=0.030$) resulting in clinical benefits (six-minute walk distance (6-MWD): $p=0.006$, NT-proBNP: $p=<0.001$), while patients treated with Tafamidis 20 mg QD showed positive effects on LASr ($p=0.039$) but no differences in LV strain (IVS: $p=0.068$, LV-GLS: $p=0.274$) and clinical status (6-MWD: $p=0.124$, NT-proBNP: $p=0.053$) compared to the natural course.

Conclusion: Treatment with Tafamidis 61 mg in ATTR-CM patients delays the increase in IVS thickness and deterioration of LA and LV longitudinal function, resulting in significant clinical benefits compared with natural history. Serial TTE with 2D speckle tracking imaging may be appropriate for disease-specific therapy monitoring.

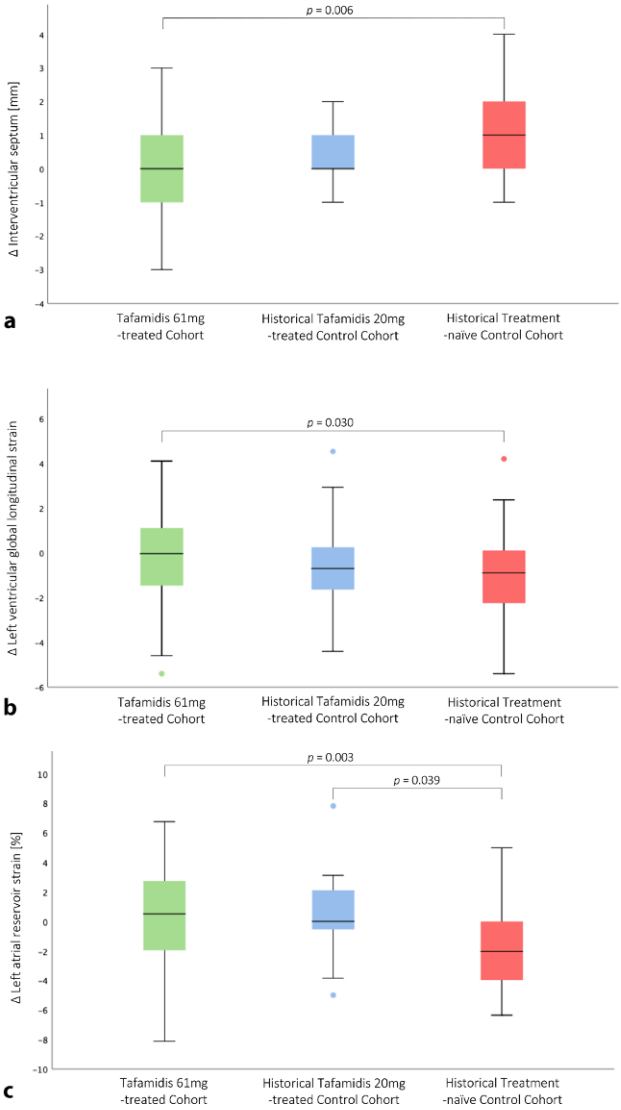

Fig. 1 Two-dimensional speckle-tracking echocardiography in Tafamidis-treated patients with transthyretin amyloid cardiomyopathy: A glimmer of hope for viable therapy monitoring? Longitudinal changes in echocardiographic parameters in patients with transthyretin amyloid cardiomyopathy (ATTR-CM)

	Tafamidis 61mg*-treated Cohort n=62‡			Historical Tafamidis 20mg*-treated Control Cohort n=21§			Historical Treatment-naïve Control Cohort n=54			Δ61mg vs. ΔNaïve	Δ20mg vs. ΔNaïve	Δ61mg vs. Δ20mg
Characteristic	*Baseline*	*Follow-up*	*p*	*Baseline*	*Follow-up*	*p*	*Baseline*	*Follow-up*	*p*	*p*	*p*	*p*
Clinical parameters												
Body-mass index (kg/m²), mean (SD)	25.7 (2.5)	25.5 (3.5)	0.104	25.6 (2.7)	25.0 (3.5)	0.084	26.2 (3.0)	25.8 (3.6)	0.250	0.641	0.698	0.287
NYHA functional class ≥ III, n (%)	33 (53.2)	21 (33.9)	**<0.001**	8 (38.1)	7 (33.3)	0.055	25 (46.3)	31 (57.4)	**<0.001**	**<0.001**	0.057	0.157
6-min walk distance (m), mean (SD)	381.2 (118.9)	386.3 (137.8)	0.764	408.4 (121.0)	397.3 (141.1)	0.486	388.1 (148.9)	336.4 (141.2)	**0.002**	**0.006**	0.124	0.479
Laboratory parameters												
Hemoglobin (g/dL), mean (SD)	13.7 (1.6)	13.4 (1.8)	0.050	13.6 (1.3)	13.5 (1.7)	0.659	12.8 (1.7)	12.5 (1.9)	0.137	0.720	0.675	0.510
Creatinine (mg/dL), mean (SD)	1.44 (1.06)	1.43 (0.84)	0.822	1.38 (0.41)	1.25 (0.37)	**0.016**	1.44 (0.79)	1.65 (1.20)	**0.016**	**0.020**	**0.019**	0.187
eGFR (mL/min/1.73m²), mean (SD)	53.8 (19.8)	53.0 (18.9)	0.566	53.9 (18.9)	59.9 (20.0)	**0.009**	53.4 (21.6)	48.5 (20.4)	**0.005**	0.061	**0.001**	**0.014**
Troponin T (ng/L), mean (SD)	66.9 (68.0)	69.7 (61.4)	0.204	73.3 (26.4)	75.4 (45.3)	0.983	86.2 (91.5)	100.1 (99.8)	**0.006**	**0.038**	0.225	0.685
NT-proBNP (pg/mL), median (IQR)	2633 (1445-5185)	2244 (1353-4784)	0.488	2740 (1355-4419)	2533 (1271-3975)	0.614	2798 (1803-4634)	3422 (2060-9065)	**<0.001**	**<0.001**	0.053	0.741
Echocardiographic parameters												
Left heart parameters												
LVEDD (mm), mean (SD)	42.4 (6.7)	41.4 (6.9)	0.227	40.0 (6.3)	39.8 (6.6)	0.841	41.6 (6.6)	41.6 (7.2)	0.918	0.358	0.838	0.575
IVS (mm), mean (SD)	19.6 (4.2)	20.0 (4.4)	0.067	21.3 (3.8)	22.0 (3.5)	**0.004**	18.8 (3.7)	19.9 (3.7)	**<0.001**	**0.006**	0.068	0.411
LVEF (%), mean (SD)	48.7 (8.6)	48.3 (10.6)	0.716	46.8 (8.5)	45.7 (10.6)	0.530	47.6 (8.6)	45.4 (10.8)	0.075	0.272	0.558	0.776
LV-GLS (-%), mean (SD)	11.75 (3.16)	11.58 (3.14)	0.534	10.61 (2.76)	10.12 (2.50)	0.309	11.71 (3.17)	10.59 (3.00)	**0.001**	**0.030**	0.274	0.568
E/A ratio, mean (SD)	1.9 (1.1)	2.1 (1.2)	0.227	1.9 (0.6)	2.1 (0.5)	0.090	1.8 (0.8)	2.0 (0.9)	0.228	0.965	0.684	0.687
E/e´ ratio, mean (SD)	14.9 (6.8)	14.9 (6.4)	0.986	15.7 (6.7)	15.7 (5.5)	0.975	17.8 (7.2)	18.4 (9.4)	0.796	0.811	0.815	0.974
LA length (mm), mean (SD)	60.9 (8.3)	60.2 (7.3)	0.429	63.7 (5.4)	63.0 (5.8)	0.652	63.0 (6.4)	64.2 (6.1)	0.209	0.143	0.294	0.988
LAVI (mL/m²), mean (SD)	38.4 (12.4)	37.8 (11.1)	0.577	40.7 (12.0)	40.6 (12.1)	0.932	38.1 (14.8)	39.9 (13.9)	0.257	0.192	0.299	0.785
LASr (%), mean (SD)	8.80 (3.41)	9.42 (4.83)	0.283	8.23 (2.91)	8.67 (3.82)	0.589	10.67 (6.19)	8.41 (4.78)	**0.005**	**0.003**	**0.039**	0.861
LAScd (-%), mean (SD)	5.25 (2.11)	5.53 (2.75)	0.534	4.69 (2.27)	5.30 (2.92)	0.272	6.22 (3.54)	4.75 (2.43)	**0.007**	**0.012**	**0.018**	0.707
LASct (-%), mean (SD)	3.55 (2.30)	3.89 (2.55)	0.173	3.55 (2.12)	3.37 (2.21)	0.720	4.45 (3.43)	3.66 (3.12)	**0.037**	**0.009**	0.338	0.311
Right heart parameters												
RVEDD (mm), mean (SD)	35.4 (5.8)	34.8 (4.8)	0.414	32.7 (7.1)	34.3 (6.0)	0.193	34.7 (5.3)	36.4 (7.0)	0.067	**0.048**	0.951	0.125
TAPSE (mm), mean (SD)	14.6 (3.9)	14.3 (3.8)	0.152	14.5 (3.6)	13.9 (4.3)	0.083	14.6 (4.3)	13.3 (3.8)	**<0.001**	**0.023**	0.184	0.612
RV-GLS (-%), mean (SD)	14.18 (4.86)	13.72 (5.07)	0.377	14.53 (6.22)	13.99 (5.73)	0.452	14.36 (5.37)	12.99 (5.21)	**0.038**	0.269	0.440	0.939
RA length (mm), mean (SD)	59.5 (8.9)	59.5 (7.5)	0.957	59.4 (6.8)	61.9 (6.5)	0.065	61.8 (7.5)	62.7 (8.2)	0.279	0.450	0.276	0.137
TR velocity (m/s), mean (SD)	3.0 (0.5)	3.0 (0.4)	0.663	2.8 (0.4)	2.9 (0.3)	0.479	2.9 (0.5)	3.0 (0.6)	0.667	0.540	0.836	0.471
sPAP (mmHg), mean (SD)	48.7 (13.3)	46.7 (11.4)	0.145	44.3 (7.7)	44.2 (8.3)	0.644	45.7 (11.0)	47.7 (13.6)	0.292	0.077	0.465	0.553

NYHA indicates New York Heart Association; eGFR, estimated glomerular filtration rate; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; LVEDD, Left ventricular end-diastolic diameter; IVS, Intraventricular septum; LVEF, Left ventricular ejection fraction; LV-GLS, Left ventricular global longitudinal strain; LA, Left atrium; LAVI, Left atrial volume index; LASr, Left atrial reservoir strain; LAScd, Left atrial

Fig. 2 Two-dimensional speckle-tracking echocardiography in Tafamidis-treated patients with transthyretin amyloid cardiomyopathy: A glimmer of hope for viable therapy monitoring? Comparison of baseline and follow-up characteristics

Temporal trends and outcomes in ST-segment elevation myocardial infarction: A cardiac magnetic resonance imaging study over the course of 15 years

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Introduction: Development of evidence-based treatments in ST-elevation myocardial infarction (STEMI) patients during the last 30 years have been associated with improved outcome; however, there are data suggesting a plateauing since around 2008. Moreover, contemporary data are very scarce regarding the temporal trends of infarct outcomes. This study sought to describe the temporal trends in infarct severity at myocardial tissue level over the course of 15 years by means of cardiac magnetic resonance imaging (MRI).

Methods: This study analyzed STEMI patients treated with percutaneous coronary intervention (PCI) at the University Clinic for Cardiology at Medical University of Innsbruck, Austria who underwent a cardiac MRI between 2005–2021. The 15-year study period was divided into sequential 2-years blocks. Infarct characteristics were measured using MRI at 3 days [IQR 2–5] after PCI.

Results: A total of 844 STEMI patients (17 % female) with a median age of 57 (interquartile range [IQR]: 51–66) years were included. The rate of evidence-based treatments was high for aspirin (99 %), P2Y12i (99 %), beta-blockers (91 %), ACEi/ATi (92 %) and statins (100 %) and did not change significantly over the study period ($p > 0.05$) with the exception for ACEi/ATi ($p = 0.03$) and prasugrel ($p < 0.001$) which increased and clopidogrel which decreased during the study course ($p < 0.001$). TIMI risk score did not change over the study period ($p = 0.43$). Overall median infarct size was 16 [9–25]% and did not change ($p = 0.39$) significantly. MVO, a marker of severe reperfusion injury, was also comparable ($p = 0.16$). Accordingly, LV ejection fraction remained virtually unchanged ($p = 0.23$).

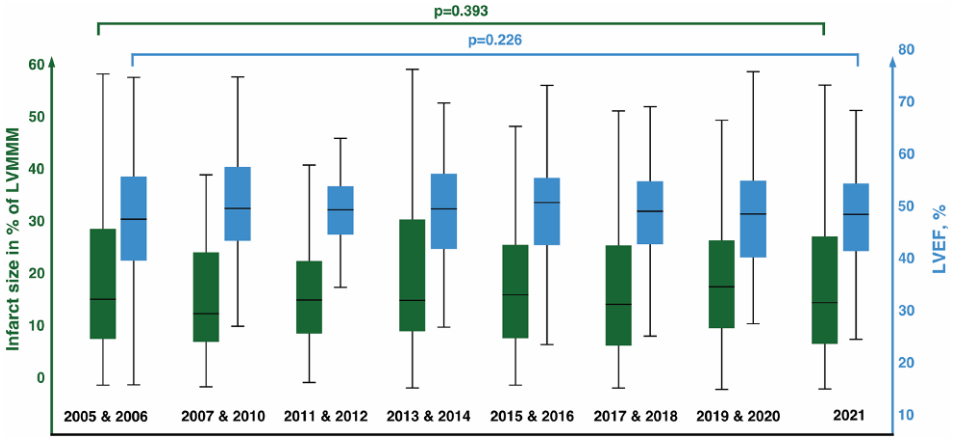

Fig. 1 Temporal trends and outcomes in ST-segment elevation myocardial infarction: A cardiac magnetic resonance imaging study over the course of 15 years

Conclusion: Although further implementation of evidence-based treatments was seen also during the last 15 years, there has been no effect on infarct size, reperfusion injury and LV ejection fraction for patients who undergo primary PCI due to STEMI. Novel treatment strategies are needed to address this unmet therapeutic need.

Implementierung von Conduction System Pacing (HIS Bundle Pacing, Left Bundle Branch Pacing) unter Verwendung eines elektroanatomischen 3D-Mapping Systems

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Einleitung: Die Möglichkeiten zur Herzschrittmacher-Therapie stellen sich heute vielfältig dar: neben transvenösen Ein- und Zweikammerschrittmachern (2KSM), sondenlosen Devices und Systemen zur klassischen kardialen Resynchronisationstherapie (CRT) steht seit wenigen Jahren die Option zur Stimulation des spezifischen Reizleitungssystems zur Verfügung (Conduction System Pacing (CSP): HIS Bündel Stimulation (HBP), Linksschenkelstimulation (LBBP)), welche in Studien bei Patienten mit einem Pacing-Anteil >20 % eine signifikante Überlegenheit zur rechtsventrikulären (RV) Stimulation gezeigt hat (Mortalität jedweder Ursache, Herzinsuffizienz-bedingte Hospitalisierung, [1]). Bei aufwändigeren Prozeduren können lange Fluoroskopiezeiten und eine erhebliche Strahlenbelastung für den Patienten und das implantierende Team entstehen. Im Sinne der Strahlenschutz-Leitlinie ALARA (As Low As Reasonably Achievable) sollten möglichst Maßnahmen ergriffen werden, um diesem Prinzip gerecht zu werden. CSP ist eine hybride Implantationstechnik, die herkömmliche Schrittmacher-Implantation mit Aspekten der Elektrophysiologie (EP) vereint, sodass der Schritt zu Verwendung eines elektroanatomischen 3D-Mapping-Systems (EAMS) hierfür naheliegend scheint. An unserer Abteilung werden oben genannte Formen von CSP unter Verwendung eines EAMS seit 03/2020 erfolgreich durchgeführt. Wir berichten über die Implementierung von CSP und klinische sowie prozedurbezogene Daten anhand unserer ersten 73 durchgeführten Prozeduren.

Methoden: Die Eingriffe erfolgen in tiefer Sedierung und Lokalanästhesie, mit transvenösen Zugängen über die V cephalica, axillaris oder subclavia. Mittels EP-Katheter wird ein 3D-Map (EnSite NavX, Abbott) der relevanten Strukturen angefertigt (Venae cavae, rechtes Atrium, Coronarsinus, interventrikuläres Septum (IVS)). Meist wird nach einer fixen Reihenfolge (HBP → LBBP → CRT) vorgegangen. Nach Markierung von Positionen mit typischen HIS-Signalen im 3D-Map wird eine CSP-geeignete Schleuse (fixe Kurve: C315 HIS, Medtronic; Selectra 3D, Biotronik; SSPC Delivery Catheter, Boston Scientific; steu-

Abb. 2 Implementierung von Conduction System Pacing (HIS Bundle Pacing, Left Bundle Branch Pacing) unter Verwendung eines elektroanatomischen 3D-Mapping Systems Zusammenfassende Grafik der erhobenen Prozedur-bezogenen Daten mit Indikationen für und angewendeten Formen von CSP, OP-Dauer in Minuten, linksventrikuläre Aktivierungszeit bei LBBP-Prozeduren, Strahlendosis und -zeit, QRS Breite im Verlauf (Präoperativ, unter RV-Stimulation über die Backup-Sonde, CSP unmittelbar postoperativ, CSP zum 3-Monats-FU und 12-Monats-FU), QRS-Breite unter HBP und LBBP präop/postop sowie Breite der stimulierten Kammerkomplexe pro Patienten im zeitlichen Verlauf

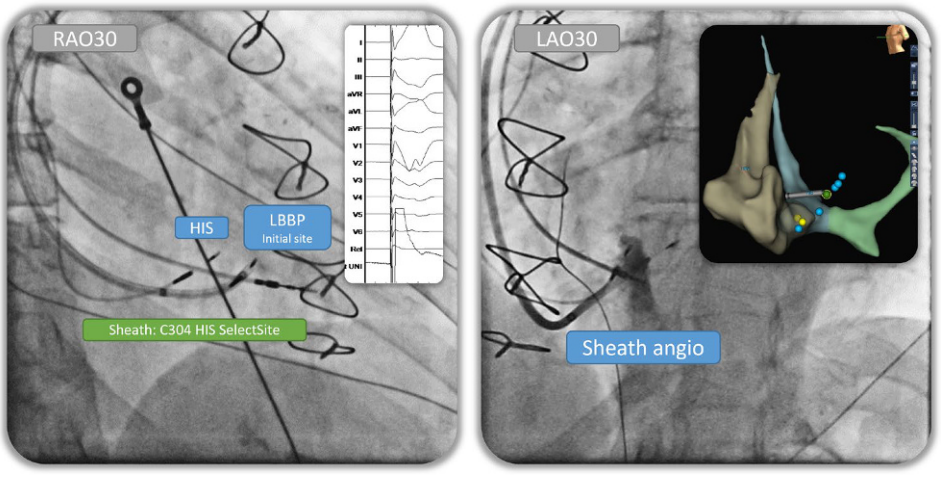

Abb. 1 Implementierung von Conduction System Pacing (HIS Bundle Pacing, Left Bundle Branch Pacing) unter Verwendung eines elektroanatomischen 3D-Mapping Systems links: Position der HIS-Sonde und initiale Position für die Linksschenkelsonde unter Verwendung einer steuerbaren Schleuse; rechts: sowohl die Schleusen-Angiographie nach Sondenfixierung als auch das 3D-Map zeigen die tief im interventrikulären Septum verschraubte Linksschenkelsonde

erbar: Agilis HIS Pro, Abbott; C304 bzw C304HIS, Medtronic) mit Schrittmachersonde eingebracht und unter 3D-Darstellung entweder dort verschraubt (selektives und nicht-selektives HBP) oder für LBBP an das proximale rechtsseitige IVS gebracht und dort nach EP-Kriterien tief in das IVS (Deep Septal Pacing, Left Septal Pacing) bzw bis an den Linksschenkel (selektives und nichtselektives LBBP) eingeschraubt. Als erfolgreich gilt, wenn die geforderten Kriterien für CSP und Klinik (zB LSB-Korrektur) erfüllt sind. Dokumentation der Sondenlage mittels Schleusen-Angiographie (Abb. 1). Erfasst werden demographische Daten, Indikation und Art des CSP, Prozedur-bezogene (OP-Dauer, Fluoroskopie-Dauer und -Dosis, Typ des Aggregates, linksventrikuläre Aktivierungszeit (LVAT)) und klinische Parameter (QRS-Dauer prä- und postoperativ sowie bei RV-Stimulation und im Follow Up (FU) nach 3 und 12 Monaten).

Resultate: Bei 67 von 73 Patienten (91 %) war CSP erfolgreich (HBP 25 Pat. = 37 %, LBBP 42 Pat. = 63 %, davon LOT-CRT 10 %). Indikationen waren AV-Blockierungen (46 %), CRT (43 %), pace&ablate (19 %), schrittmacherinduzierte Kardiomyopathie (PICM, 12 %) und frustrane CRT-Anlage im Vorfeld (10 %). Die 3D-Darstellung war einfach durchführbar und trug in hohem Ausmaß zum anatomischen Verständnis bei. Die OP-Dauer betrug im Schnitt 137 ± 43 min, die Fluoroskopiedauer lag bei 11 ± 8 min, die mediane Fluoroskopie-Dosis bei 519 µGym^2, wobei hier Patienten mit hochgradig dilatiertem Ventrikel und diffuser Leitungsverzögerung (IVCD) teils deutlich darüber lagen. Verglichen zu einer internationalen, multizentrischen CSP-Experten-Studie ohne 3D konnte hier eine längere Prozedurdauer (vgl. 105 ± 54 min) sowie deutlich reduzierte Fluoroskopie-Zeit (vgl. 19 ± 15 min) verzeichnet werden [1]. 22 Patienten erhielten eine RV-Backup-Sonde (33 %; meist HBP); 64 % der Patienten erhielten einen 2K-Schrittmacher, 36 % ein CRT-Aggregat. Verglichen zum Ausgangs-QRS präoperativ kam es unter RV-Stimulation (Backup-Sonde) zu einer signifikanten QRS-Verbreiterung und unter CSP postoperativ und zum 3-Monates-FU zu einer signifikanten QRS-Schmälerung (Abb. 2). Bei LBBP konnten exzellente Reizschwellen erzielt werden, die um 0,75 mV@0,4 ms lagen und im Verlauf stabil blieben. Unter HBP kam es bei 4 Patienten (16 % des HBP) 3–12 Monate postoperativ zu einem Anstieg der Reizschwelle auf den 2- bis 3-fachen Ausgangswert.

Schlussfolgerungen: Die Stimulation des Reizleitungssystems (CSP) ist eine moderne und aufstrebende Methode zur physiologischen Herzschrittmachertherapie, die in Studien – insbesondere ab einem Pacing-Anteil >20 % – verglichen zu RV-Pacing einen signifikanten Mortalitäts-Benefit bietet und Herzinsuffizient-bedingte Hospitalisierungen signifikant reduziert. Die Implantation unter Verwendung eines 3D-Mapping-Systems ist einfach anwendbar, mit moderatem zeitlichem Mehraufwand verbunden und hilft in hohem Ausmaß dem anatomischen Verständnis sowie Fluoroskopie-Dauer und -Dosis auf ein notwendiges Minimum zu reduzieren. Die Beherrschung mehrerer Arten von CSP (HBP, LBBP …) erhöht die Chancen auf eine erfolgreiche Prozedur deutlich. Fortgeschrittene Kardiomyopathien mit diffuse Erregungsleitungsstörung können unserer Erfahrung nach zu sehr aufwändigen Prozeduren führen, um ein für den Patienten günstiges Ergebnis zu erzielen (zB LOT-CRT).

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Impaired outcome after CABG in women

Pölzl L[1], Thielmann M[2], Cymorek S[2], Nägele F[1], Hirsch J[1], Graber M[1], Sappler N[3], Eder J[1], Staggl S[3], Theurl F[3], Abfalterer H[1], Reinstadler SJ[3], Holfeld J[1], Griesmacher A[4], Ulmer H[5], Grimm M[1], Bauer A[3], Ruttmann E[1], Bonaros N[1], Gollmann-Tepeköylü C[1]

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Introduction: Female sex is protective against coronary artery disease (CAD). However, CAD is still underdiagnosed in women, and current data on their outcome after CABG remains controversial. The aim of this study was to (a) determine the impact of sex on outcome after CABG surgery and (b) identify responsible factors.

Methods: Data from 2829 patients (18.1 % female) undergoing CABG procedure was analyzed retrospectively. Study population was gathered from 2008–2018 from two centers (Innsbruck, Austria: $n = 1885$; Essen, Germany: $n = 944$). The composite primary outcome was myocardial infarction, all-cause mortality, and repeat revascularization (MACE) at 30 days and 5 years. The secondary outcome was all-cause mortality at 30 days and 5 years. Kaplan–Meier estimates were used to plot survival curves for MACE and all-cause mortality, which were compared in log-rank tests. Outcomes of groups were subjected to logistic regression analysis for the 30-day results and Cox proportional hazards model analysis for the five-year results.

Results: Preoperative data showed significant differences between men and women, reflected in a higher EURO score II in women (1.34 vs. 2.28; $p < 0001$). Within the first 30 days, MACE rates (2.5 % vs. 4.9 %; $p = 0.005$) and all-cause mortality (1.5 % vs. 3.3 %; $p = 0.004$) were twice as high in female patients. These findings were supported by results of regression models (MACE OR 1.960, CI 1.215–3.160, $p = 0.006$; all-cause mortality OR 2.300, CI 1.275–4.151, $p = 0.006$). In the 5-year follow up an increased risk for long-term MACE was observed in women (HR 1.271, CI 1.008–1.601; $p = 0.042$). Prognostic relevance of the female gender was not significant after adjustment in the regression models.

Conclusion: Female gender is associated with higher rates of MACE and all-cause mortality upon CABG procedure. The underlying cause might be worse preoperative characteristics.

Postersitzung 1 – Akutes Koronarsyndrom

1-1

Impact of Copeptin plasma level in differentiation of Type 1 and Type 2 myocardial infarctions

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Introduction: Since the release of the "Fourth Universal Definition of Myocardial Infarction" consensus document and its classification for myocardial infarction (MI), distinguishing between the different subtypes, particularly type 1 MI (T1MI) from type 2 MI (T2MI), has been of great diagnostic and therapeutic importance. This study aimed to investigate whether copeptin, a stress hormone produced in the hypothalamus and a surrogate marker for vasopressin, helps to differentiate between T1MI and T2MI in addition to cardiac troponin.

Methods: In a retrospective analysis, 1271 unselected consecutive patients presenting with symptoms suggestive of cardiac ischemia between 2011 and 2017, were evaluated. Patients diagnosed with ST-elevation MI were excluded. All Non-ST-ACS patients underwent clinical assessment, including cardiac troponin I (cTnI) and copeptin measurements. Afterwards, troponin-positive patients were further classified into T1MI and T2MI (including those with myocardial injury) using clinical assessment and coronary imaging.

Results: Out of all remaining patients 1007 (86.7 %) had no troponin elevation; whereas 153 (13.3 %) subjects showed increased cTnI levels and were diagnosed as having an MI, of which 78 (51 %) were classified as T1MI and 76 (49 %) as T2MI, respectively. The Mann-Whitney-U test revealed a significant difference in the copeptin plasma concentration between patients with or without a cTnI-elevation (12.34 pmol/L vs 5.24 pmol/L, $p < 0.001$), as well as between T1MI or T2MI patients (8.11 pmol/l vs 21.38 pmol/l, $p = 0.001$). The calculated area under the curve (AUC) for using copeptin in differentiating between both MI types was 0.66 (CI: 0.57; 0.74). A multivariable regression analysis revealed that higher concentrations of Copeptin and CRP as well as a higher heart rate at admission and a lower frequency of smoking habits remained significantly associated with T2MI.

Conclusion: Copeptin has only a moderate ability for differentiating T1MI from T2MI.

1-2

Primär-PCI in Österreich seit 2005: Charakteristika, prozedurale Trends und Outcome von 19.054 Patienten

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Einleitung: Nach der flächendeckenden Einführung der Primär-PCI haben die Fortschritte der adjuvanten pharmakologischen Therapie und der Interventionstechniken in randomisierten Studien zu einer weiteren Verbesserung des Outcomes nach ST-Hebungsinfarkt geführt. Ziel der vorliegenden Analyse war es die zeitlichen Veränderungen des Managements sowie des Outcomes nach Primär-PCI in Österreich zu beschreiben.

Methoden: Für die vorliegende Analyse wurden die Daten von 19.054 Patienten, die zwischen 2005 und 2018 eine Primär-PCI erhielten und prospektiv im österreichischen Akut-PCI Register erfasst wurden, berücksichtigt. Die Patienten wurden nach dem Einschlusszeitraum in eine frühe (2005–2011) und eine späte Gruppe (2012–2018) eingeteilt und verglichen. Zudem wurde für die am stärksten variierenden Parameter der jährliche Trend analysiert.

Resultate: Die Verteilung des Alters, des Geschlechts sowie der Hauptrisikofaktoren war ebenso wie die Häufigkeit an Eingriffen im kardiogenen Schock in beiden Gruppen gleich. Demgegenüber nahm die Rate der reanimierten Patienten mit Primär-PCI im zeitlichen Verlauf zu (8 % vs. 11 %). Patienten basierte sowie extramurale Therapieverzögerungen zeigten keinen Unterschied, während in der door-to-balloon Zeit eine Verbesserung von im Median 52 (IQR 34–80) auf 44 (IQR 30–67) min erreicht wurde. Eine vollständige Verschiebung von bare-metal zu drug eluting Stents konnte ebenso wie eine stetig steigende Rate an radialen PCI's dokumentiert werden. Die Rate an GPIIb/IIIa Inhibitoren sowie die Vortherapie mit P2Y12

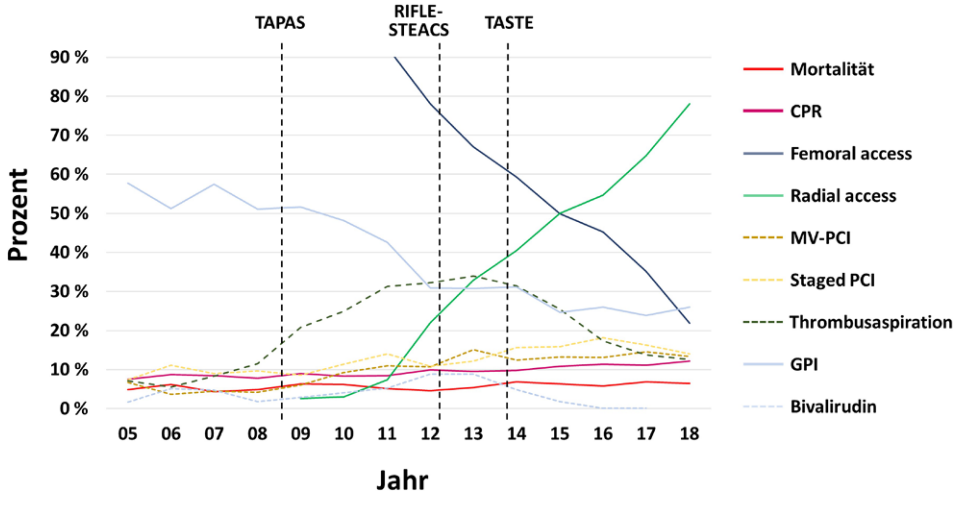

Abb. 1 | 1-2 Outcome und Trends der interventionellen und adjuvanten Medikamentösen Therpie bei Primär-PCI in Österreich

Inhibitoren und die Häufigkeit von Thrombusaspirationen nahmen im zeitlichen Verlauf ab, wohingegen die Anwedung von Ticagrelor oder Parasugrel anstieg. Mechanische Unterstützungssysteme kamen bei insgesamt rückläufiger Anwendung nur vereinzelt zum Einsatz. Die Krankenhausmortalität blieb im zeitlichen Verlauf unverändert nieder (6.0 % vs. 6.5 %).

Schlussfolgerungen: Zusammenfassend spiegelt die Infarktversorgung mittels Primär-PCI in Österreich in hohem Maß die Dynamik der Evidenz zu den angewendeten Devices und der adjuvanten medikamentösen Therapie wider. Die Sterblichkeit im akuten Myokardinfarkt bleibt trotz komplexer werdender Patienten nieder und vergleichbar mit anderen Ländern.

1-3

The impact of the antidepressive therapy on bleeding and ischemic events in patients with Takotsubo Syndrome

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Introduction: Previous studies showed that antidepressive therapy, especially selective serotonin reuptake inhibitors (SSRI), may inhibit platelet activity. However, data analysing the effect of antidepressive therapy in patients with a history of acute coronary syndrome on P2Y12-inhibitors present conflicting evidence with regard to MACE and bleeding events. This study aims to evaluate mortality, bleeding, and ischemic events in patients with Takotsubo Syndrome (TTS) on antidepressive therapy in combination with P2Y12-inhibitors.

Methods: We conducted a single-centre, retrospective study of TTS patients, from September 2006–December 2020. Altogether 207 patients were included in the study. The primary endpoints were bleeding, ischemic events, all-cause and cardiovascular mortality within one year after discharge in TTS patients on P2Y12-inhibitors therapy. Moreover, we analysed if there is a difference in the outcome of antidepressive therapy irrespective of the concomitant antiplatelet therapy. Antidepressive therapy as a predictor of adverse events was analysed using binomial logistic regression, and time-to-event analyses were performed with Kaplan-Meier estimators.

Results: Forty-three (21 %) patients were on antidepressive therapy. There was no difference in demographic characteristics between TTS patients with and without antidepressive therapy. However, patients with no antidepressive therapy received statistically significantly more often P2Y12-inhibitors therapy at discharge. In general, antidepressive therapy was not associated with bleeding, ischemic event, all-cause or cardiovascular mortality, respectively. Furthermore, the analysis of TTS patients on P2Y12-inhibitors showed that antidepressive therapy was not associated with any of the primary endpoints in the first year after discharge for the TTS event.

Conclusion: The results of our study demonstrate that unlike in ACS patients, antidepressive therapy on top of P2Y12-inhibitors in TTS patients is not associated with the ischemic or bleeding event and mortality in the first year after discharge.

1-4

Geschlechtsassoziierte Einflussfaktoren auf das Kurzzeit-Outcome nach PPCI

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Einleitung: Frauen weisen eine höhere Kurzzeit-Mortalität und -Komplikationsrate nach primärer PCI (PPCI) auf als Männer, wobei es dafür unterschiedliche Hypothesen gibt. Ziel dieser Studie ist zu evaluieren, ob es weiterhin Unterschiede des Kurzzeit-Outcome in unserer täglichen klinischen Praxis gibt und geschlechtsassoziierte Einflussfaktoren zu beschreiben.

Methoden: Für die gegenwärtige unizentrische Analyse wurden Patient:innen, die konsekutiv zwischen 01/2012 und 12/2020 eine PPCI an unserem Institut erhielten, analysiert. Primäre Endpunkte waren intra-hospitale Mortalität, sowie die kombinierten Endpunkte MACE (Tod, Myokardinfarkt und Schlaganfall) und NACE (MACE und nicht assoziierte Blutungen). In einer multivariaten Regressionsanalyse wurde der Einfluss relevanter geschlechtsassoziierter Begleitfaktoren auf das klinische Outcome beschrieben.

Resultate: Insgesamt wurden 916 Patient:innen eingeschlossen, wobei 228 (25 %) Frauen und 688 (75 %) Männer waren. Die intra-hospitale Mortalität war bei Frauen höher als bei Männern (9,5 % vs. 5 %; $p=0{,}02$), wodurch MACE (9,9 vs. 6,4 %; $p=0{,}09$) und NACE (12 % vs. 7,3 %; $p=0{,}03$) bei Frauen ebenfalls häufiger beobachtet wurden. Nach Elimination relevanter Störgrößen zeigte sich lediglich ein Trend hinsichtlich einer Mortalitätssteigerung durch das weibliche Geschlecht (OR 1,6; SE 0,6). Während vor allem das Alter (OR 1,1; SE 0,02; $p<0{,}001$), das Vorliegen eines kardiogenen Schocks (OR 11,7; SE 4,1; $p<0{,}001$), als auch die Gabe von P2Y12-Inhibitoren (OR 0,3; SE 0,1; $p<0{,}001$) einen signifikanten Einfluss auf die Mortalität hatte, spielte das Vorliegen von Blutungskomplikationen lediglich bei Männern eine Rolle (OR 0,6; SE 0,8 vs. OR 4,8; SE 3,8; $p<0{,}05$). Betrachtet man die Entwicklung der Mortalität über den gesamten Beobachtungszeitraum (≤2016 vs. ≥2017), so zeigt sich im früheren Zeitraum kein signifikanter Unterschied zwischen Frauen und Männern (≤2016: 8,2 % vs. 5,7 %; $p=0{,}32$), jedoch ein deutlicher Unterschied in der Mortalität zwischen den Geschlechtern in den letzten Jahren (≥2017: 11,2 % vs. 4,1 %; $p=0{,}01$).

Schlussfolgerungen: Trotz überarbeiteter Therapiekonzepte und Guidelines ist in unserem Kollektiv die Kurzzeit-Mortalität von Frauen nach PPCI weiterhin höher als bei Männern und hat in den letzten Jahren weiter zugenommen.

1-5

Left ventricular thrombus in a patient with acute anterior wall infarction due to spontaneous coronary artery dissection (SCAD)–A case report

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Introduction: Spontaneous coronary artery dissection (SCAD) is a more common cause of acute coronary syndrome (ACS) and sudden cardiac death than previously believed. It is assumed to be the cause of ACS in up to 35 % of myocardial infarction in women under 50 years. The main risk factors differ significantly from ACS of atherosclerotic etiology including female sex, pregnancy, arteriopathies such as fibromuscular dysplasia and mechanical or emotional stressors.

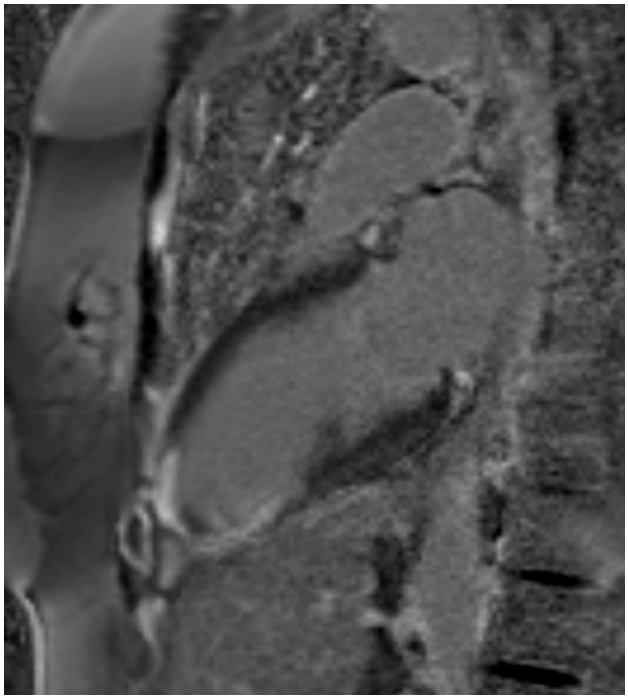

Fig. 2 | 1-5 Cardiac magnetic resonance imaging showing apical scar and apical thrombus

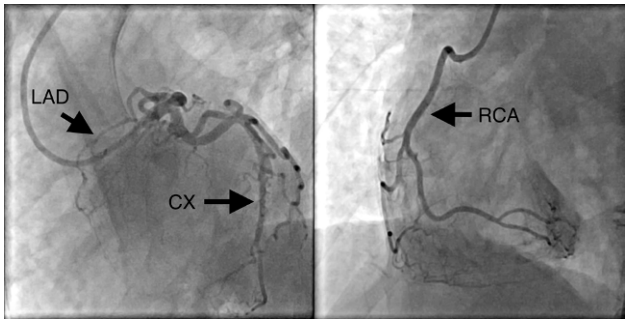

Fig. 1 | 1-5 Coronary angiogram showing SCAD type 2B of the LAD with long diffuse stenosis

Methods: Case Presentation: A 43-year-old woman with no significant medical history complained about sudden onset of chest pain and palpitations. Upon arrival of the emergency physician the electrocardiogram showed ST segment elevations in the area of the anterior wall. During the transport to our hospital the patient experienced three episodes of ventricular fibrillation with successful defibrillation. At the time of the admission to our intensive care unit the patient was hemodynamic stable and breathing spontaneously.

Results: Coronary angiography revealed a dissection from the mid-to-distal left anterior descending artery (SCAD type 2B), a conservative approach was chosen. The patient was observed on the intensive care unit and monitoring showed no more rhythmological abnormalities. First diagnosis of Graves' disease with hyperthyroidism as potential contributing SCAD-trigger mechanism was made and a therapy with thyreostatic drugs was applied. Follow-up echocardiography and cardiac magnetic resonance imaging showed a mildly reduced left ventricular ejection fraction (EF 50 %) due to apical akinesia and a mural apical thrombus (3 ×1 cm). Treatment with an ace-inhibitor and a beta-blocker was established and the initial therapy with aspirin was switched to anticoagulation with phenprocoumon. She was discharged one week after the initial event in good general condition and equipped with a life-vest. A follow-up appointment was set to reevaluate the thrombus and the need of oral anticoagulation.

Conclusion: This case underlines the therapeutic challenges of optimal medical therapy of conservatively managed SCAD. There is an ongoing debate about the role of single and dual antiplatelet regimen. Anticoagulation is usually avoided as it may enhance intramural bleeding. However, the presence of a left ventricular thrombus with the risk of embolic complications justifies the use after benefit-risk assessment.

1-6

Impact of body size indices on platelet function in acute coronary syndrome patients under dual anti-platelet therapy after coronary stenting

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Introduction: Patients undergoing acute percutaneous coronary intervention (PCI) receive a dual anti-platelet therapy for secondary prevention. Still, some patients develop ischemia while others bleed. Dosing of therapy may be based on body size indices.

Methods: We correlated ADP- and SFLLRN-mediated platelet aggregation with the body mass index, body surface area, lean body mass and blood volume, respectively, obtained from 220 patients (121 patients received prasugrel and 99 ticagrelor).

Results: Fifty-six patients had BMI values of >30 kg/m2. There were no correlations between aggregation responses and any of the body size indices in patients on therapy in the recommended fixed dosages

Conclusion: The body size indices we studied in our cohort, do not appear to have any relevance for individualised therapy dosage.

Postersitzung 2 – COVID-19 und Herz

2-1

Left ventricular global longitudinal strain is associated with more severe COVID-19 even in patients without a patient history of cardiac disease

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Introduction: The SARS-CoV-2 pandemic of 2020 has not only claimed the lives of millions, but has also put an immense strain on the healthcare system and the global economy. Even though the virus mostly affects the respiratory system, some patients show signs of cardiac involvement. However, we do not know, if the virus has more subtle effects on the heart even in patients without evident cardiac involvement and if these effects may persist even after recovery from COVID-19. The aim of this study was to detect echocardiographic abnormalities after recovery from COVID-19 and especially assess subclinical myocardial dysfunction by measuring left and right ventricular strain and assessing diastolic function in these patients.

Methods: Patients were included after a verified infection with the SARS-CoV-2 virus, after discharge from the hospital. Baseline information including clinical history, vital signs and symptoms were assessed. In addition, we measured laboratory parameters and a transthoracic echocardiography exam was performed in every patient. Left ventricular (LV) global longitudinal strain (LV-GLS) was measured in an apical long axis-, four- and two-chamber view. Right ventricular (RV) longitudinal strain (RV-LS) was measured in an RV-optimized four-chamber view in the free lateral wall of the RV. In addition, standard 2D and Doppler measurements were performed in each patient to describe cardiac dimensions as well as systolic, diastolic and valvular function. Assessed parameters were compared between two groups, which were divided by median length of hospital stay. Results were adjusted for patients with previously diagnosed cardiac and renal disease.

Results: Data from 150 patients were analyzed in this study. Echocardiographic abnormalities were found in 46.7 % of patients after a median time of 6 months from the date of discharge after COVID-19 hospitalization (Table 1). We found that patients with longer hospitalization duration–indicating a more severe course of disease–more commonly presented with diastolic dysfunction (more pronounced LV hypertrophy, higher E/e' ratio, larger left atrial size). These patients also showed signs of systolic dysfunction, determined by reduced LV-GLS, even though there was no difference in LV ejection fraction, depending on the duration of their hospital stay. Patients who had more impaired LV-GLS also reported more severe dyspnea and performed worse in the six-minute walk test. Interestingly, in patients without previously diagnosed cardiovascular or renal disease, patients with higher levels of serum NT-pro BNP and more impaired LV-GLS were hospitalized longer during acute COVID-19.

Conclusion: Cardiac abnormalities are common in patients who had been hospitalized for an acute SARS-COV-2 infection. Especially in patients with severe disease, diastolic dysfunction and subtle systolic dysfunction was present. Even in patients without previously known cardiovascular or renal disease, subtle changes such as slightly higher NT-pro BNP and impaired reduced global longitudinal left ventricular strain may be associated with a more severe course of COVID-19.

Table 1 | 2-1 Overview of abnormal echocardiographic findings in patients with longer versus shorter hospital stays

	Total study population ($n=150$)	Hospital stay ≤9 days ($n=90$)	Hospital stay >9 days ($n=60$)	p-value
Any abnormal finding	70 (46.7)	39 (43.3)	31 (51.7)	0.508
LV hypertrophy	71 (48.6)	36 (40.4)	35 (61.4)	0.013
Reduced LVEF	6 (4.6)	2 (2.5)	4 (7.8)	0.159
Abnormal LV-GLS				0.025
Borderline	24 (21.6)	12 (16.7)	12 (30.8)	
Reduced	17 (15.3)	8 (11.1)	9 (23.1)	
Regional hypokinesia	4 (2.7)	0 (0.0)	4 (6.9)	0.012
Diastolic dysfunction	6 (4.3)	2 (2.3)	4 (7.5)	0.027
Enlarged atria	27 (22.0)	10 (13.3)	17 (25.4)	0.004
Reduced RVF	7 (4.7)	2 (2.2)	5 (8.3)	0.079
Reduced RV-LS	8 (17.4)	3 (6.5)	5 (10.9)	0.410
Elevated sPAP	3 (2.0)	1 (1.1)	2 (3.3)	0.341
Significant VHD	6 (4.1)	3 (3.4)	3 (5.1)	0.615
Pericardial effusion	1 (0.6)	0 (0.0)	1 (1.7)	0.406

Categorical variables are shown as numbers and percentages. Due to image quality, LV-GLS could only be measured reliably in 111 patients and RV-LS could only be measured in 46 patients. *LV* indicates left ventricle; *LVEF* left ventricular ejection fraction; *RVF* right ventricular function; *RV-LS* right ventricular longitudinal strain; *sPAP* systolic pulmonary artery pressure; *VHD* valvular heart disease

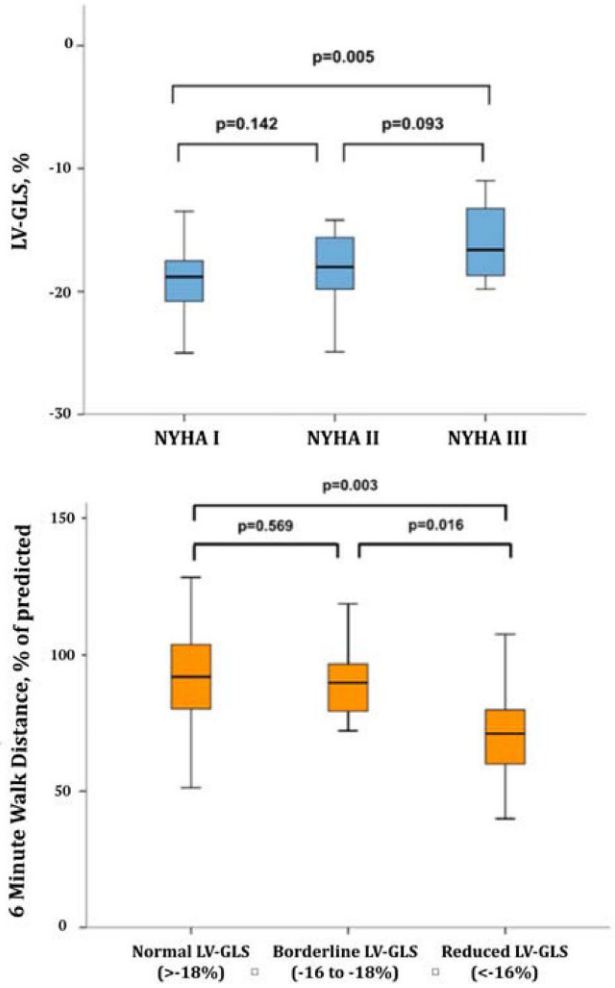

Fig. 1 | 2-1 Panel A shows the association between self reported dyspnea (New-York Heart Association Score) and left ventricular global longitudinal strain (LV-GLS) after recovery from COVID-19, Panel B shows the association between LV-GLS and results of the 6 Minute Walk Test after recovery from COVID-19

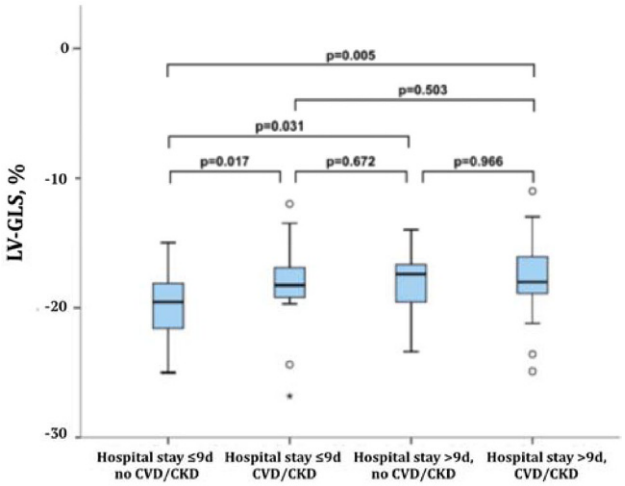

Fig. 2 | 2-1 Left ventricular global longitudinal strain (LV-GLS) after recovery from COVID-19 according to number of days of hospitalization and presence of previously diagnosed cardiovascular (CVD) or renal disease (CKD)

2-2

Association of interleukin-32 and interleukin-34 with cardiovascular disease and prognosis in COVID-19

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Introduction: Cardiovascular disease is a common risk factor in hospitalized patients with COVID-19. Interleukin-32 (IL-32) and Interleukin-34 (IL-34) have been hypothesized to contribute to cardiovascular involvement in COVID-19.

Methods: This prospective, observational study of hospitalized patients with COVID-19 was conducted from June 6th to November 26th, 2020 in a tertiary care hospital in Vienna, Austria. IL-32 and IL-34 were measured upon hospital admission. We sought to evaluate the association of both biomarkers with cardiovascular disease and to assess the prognostic impact of IL-32 and IL-34 on short-term all-cause mortality in the context of COVID-19.

Results: A total of 200 patients with COVID-19 were enrolled. The 28-day mortality rate was 13 % ($n=26$) in our study population. Patients with cardiovascular disease (history of heart failure or coronary artery disease) had a significantly higher risk of mortality (Crude HR 4.357 [95 % CI, 2.019–9.406], $P<0.001$). Levels of IL-32 and IL-34, however, did not show any significant differences between survivors and those with a fatal disease course (IL-32 median of 0.00 pg/ml [IQR, 0.0–102.60] vs 1.62 pg/ml [IQR, 0.00–35.80]; $p=0.985$ and IL-34 median of 10.58 pg/ml [IQR, 6.76–58.87] vs 9.15 pg/ml [5.75–29.00], $P=0.128$). The area under the ROC curve for IL-32 predicting 28-day mortality was 0.501 (95 % CI, 0.376–0.626) and for IL-34 0.593 (95 % CI, 0.473–0.712). The interleukins also showed no association with cardiovascular disease or traditional cardiovascular biomarkers (high-sensitive troponin I or NT-proBNP).

Conclusion: Patients with cardiovascular disease are at an increased risk of mortality in hospitalized patients with COVID-19. IL-32 and IL-34, however, neither show an association with cardiovascular disease nor do they provide additional benefit for outcome prediction in our study population.

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2-3

Cardiac and pulmonary long-term sequelae in patients after severe Covid-19 infection

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Introduction: Multiple studies have described acute effects of the Covid-19 infection on the heart, but little is known about the long-term cardiac and pulmonary effects and complications after recovery. The aim of this analysis was to deliver a comprehensive report of symptoms and possible long-term impairments after hospitalization because of Covid-19 infection as well as to try to identify predictors for Long-Covid.

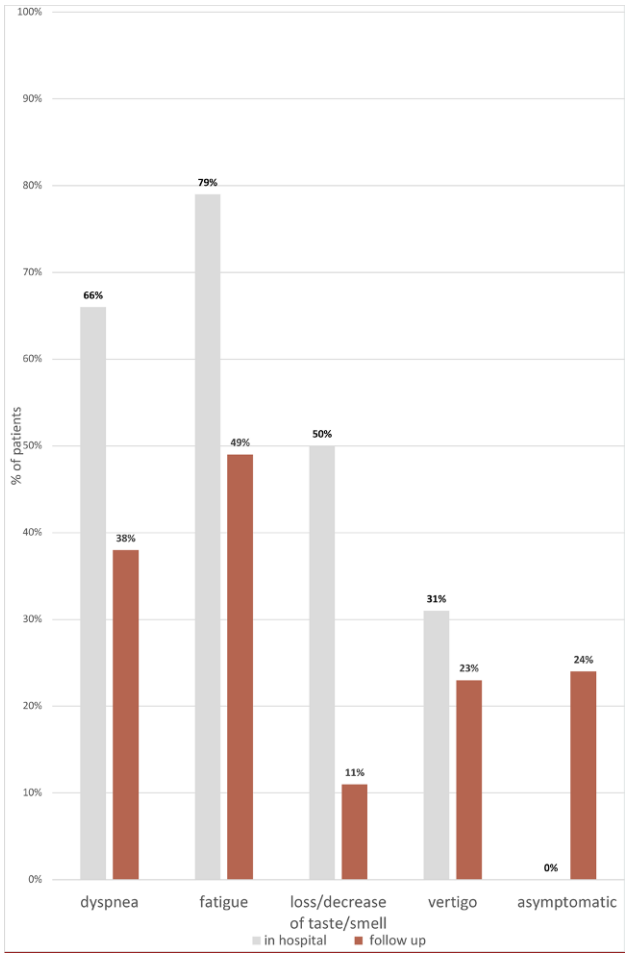

Fig. 1 | 2-3 Spectrum of Long-Covid symptoms 6 months post discharge in relation to acute phase symptoms during hospital stay

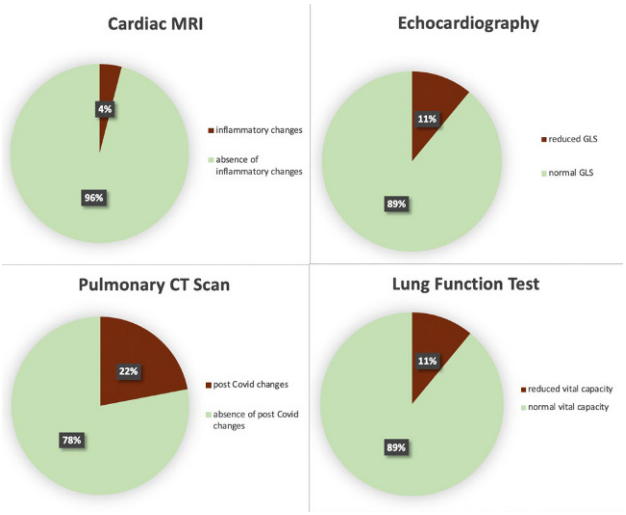

Fig. 2 | 2-3 Cardiac and pulmonary structural and functional changes 6 months post Covid-19 (*MRI* magnetic resonance imaging, *GLS* global longitudinal strain, *CT* computed tomography)

Methods: This was a prospective, multicenter registry study. Patients with verified Covid-19 infection, who were treated as in-patients at our dedicated Covid hospital (Clinic Favoriten), have been included in this study. In all patients, testing was performed approximately 6 months post discharge. During the study visit the following tests and investigations were performed: detailed patient history and clinical examination, transthoracic echocardiography, electrocardiography, cardiac magnetic resonance imaging (MRI), chest computed tomography (CT) scan, lung function test, six-minute walk test (6MWT) and a comprehensive list of laboratory parameters including cardiac bio markers such as brain natriuretic peptide (NTpro-BNP) and troponin T.

Results: Between July 2020 and October 2021, 150 patients were recruited. Sixty patients (40 %) were female and the average age was 53.5 ± 14.5 years. Of all patients, 92 % had been admitted to our general ward and 8 % had a severe course of disease, requiring admission to our intensive care unit. Six months after discharge the majority of patients still experienced symptoms and 75 % fulfilled the criteria for Long-Covid: 49 % presented with fatigue and general weakness and 38 % with exertional dyspnea, representing the two most common symptoms. Only 24 % were completely asymptomatic (Fig. 1). Echocardiography detected reduced global longitudinal strain (GLS) in 11 %. Cardiac MRI revealed pericardial effusion in 18 %. Furthermore, cardiac MRI showed signs of former peri- or myocarditis in 4 %. Pulmonary CT scans identified post-infectious residues, such as bilateral ground glass opacities and fibrosis in 22 %. Exertional dyspnea was associated with either reduced forced vital capacity measured during pulmonary function tests in 11 %, with reduced GLS and/or diastolic dysfunction, thus providing evidence for a cardiac and/or pulmonary cause. Independent predictors for Long-Covid were markers of a more severe disease course like length of in-hospital stay, admission to an intensive care unit, type of ventilation as well as higher NT-proBNP and/or troponin levels.

Conclusion: Even 6 months after recovery from Covid-19 infection, the majority of previously hospitalized patients still suffer from at least one symptom, such as chronic fatigue and/or exertional dyspnea. While there was no association between

fatigue and cardiopulmonary abnormalities, impaired lung function, reduced GLS and/or diastolic dysfunction were significantly more prevalent in patients presenting with exertional dyspnea. In summary, only mild impairments of cardiopulmonary function could be identified. However, approximately one fifth of all patients showed post infectious changes in chest CT, which do not appear to be functional, and several suspected post infectious changes in cardiac MRI such as myo- and pericarditis in a few cases as well as an accumulation of pericardial effusions.

2-4

Evolution of electrophysiology interventions in Austria 2016–2021: Effect of lockdowns during the COVID-19 pandemic

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Introduction: Catheter ablation is an established interventional therapeutic option for a variety of supraventricular and ventricular arrhythmias. Therefore, number of ablations performed annually increase annually. Additionally, success rates constantly improve in left- and right-atrial- and ventricular arrhythmias. In 2015 we initiated an Austrian Ablation Registry and analyzed the development of procedures during the COVID-19 pandemic based on input from multiple Austrian ablation centers.

Methods: The number and type of electrophysiological interventions if the Austrian Ablation Registry 2016–2021 were analyzed as monthly and weekly totals. In particular, the impact of lockdowns with imposed restrictions on elective procedures in April and November 2020 was addressed.

Results: A total of 17,030 catheter ablation records were entered in Austria between 2016 and 2021. Indications for ablation were 19.6 % AVNRT, 5.2 % WPW, 14.2 % VH flutter, 28.5 % paroxysmal and 11.7 % persistent VH fibrillation, 8.1 % atrial and AV nodal ablations, and 6.9 % ventricular tachycardia (VT). The total number of ablations increased steadily from approximately 122 per month in January 2016 to 321 in June 2021, with seasonal fluctuations. The largest absolute increases were seen in procedures for paroxysmal atrial fibrillation (AF) from 11–29 per month. During the two lockdowns due to the COVID-19 pandemic, there were significant decreases in ablations in April 2020 (251–79) and November 2020 (from 300–210). Ablations for ventricular tachycardia were not affected by these changes. However, the number of procedures per year did not decrease significantly due to national lockdowns by reason of COVID-pandemic.

Conclusion: Since 2016, a steady increase in ablation procedures has been registered in Austria. Pronounced increase was observed in left atrial catheter ablations (paroxysmal and persistent AF), but also in ablations of VT. During the COVID-19 pandemic, the 2 major lockdowns resulted in transient reductions in ablation numbers, but the total number of procedures remained stable in 2020 and 2021.

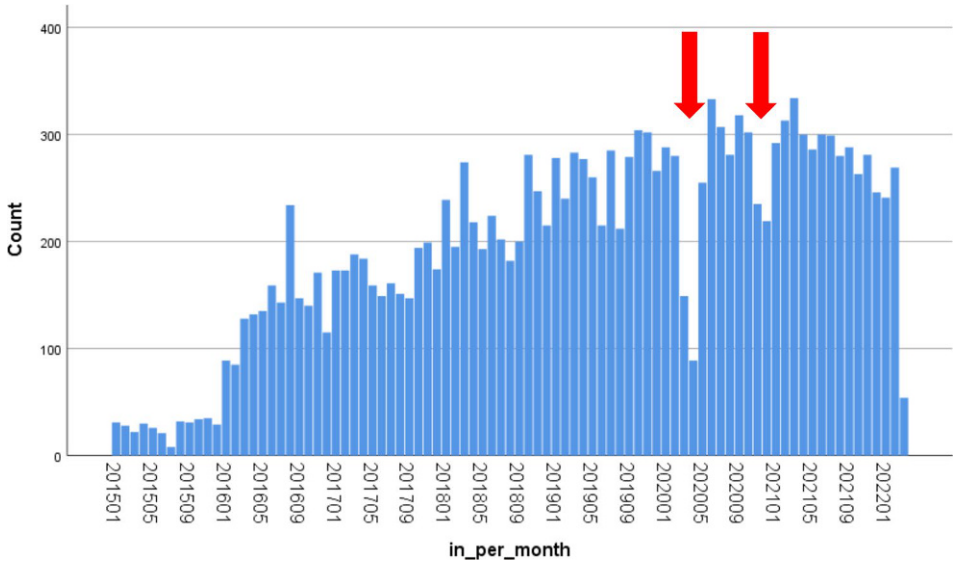

Fig. 1 | 2-4

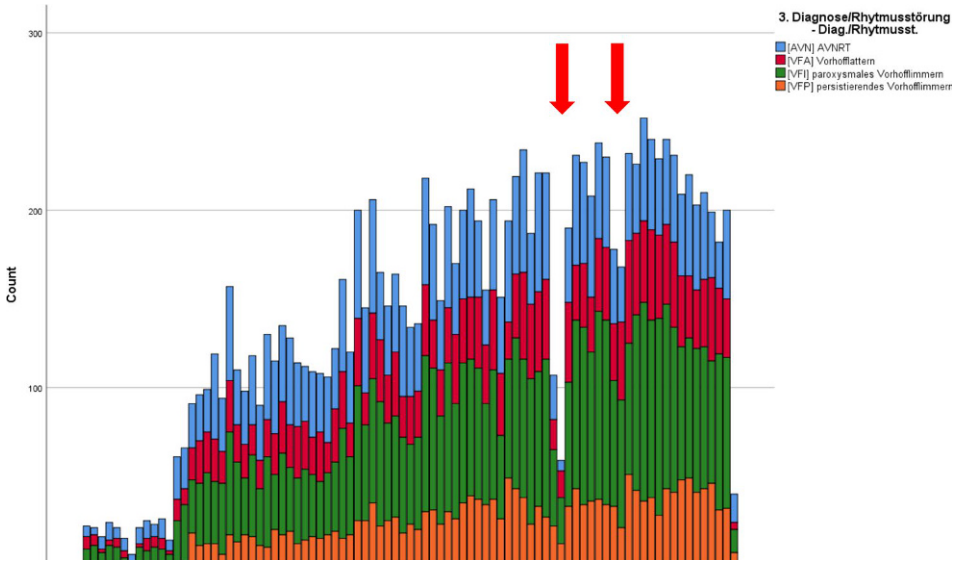

Fig. 2 | 2-4

2-5

Subjective pain perception and anxiety in patients with acute myocardial infarction during COVID-19

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Introduction: Patients often experience the acute phase of a myocardial infarction as a stressful, traumatic, and life-threatening experience, that leads to overall mental distress and severe anxiety [1]. To date, few data has been published on the association between acute myocardial infarction (AMI) related severe anxiety and myocardial infarction related perceived pain. However, previous study results suggest that AMI related anxiety might be associated with the severity of myocardial infarction related perceived pain. Not only acute cardiovascular diseases, but also threatening infectious diseases, are often associated with the development of increased anxiety, panic and phobic fears. This is particularly the case during the current COVID-19 pandemic. Studies have already shown that the COVID-19 pandemic leads not only to higher stress levels, but also to increased anxiety symptoms in the general population [2]. Thus, pre-existing severe COVID-19 related anxiety and pre-existing trait anxiety may further increase stress levels, making AMI more vulnerable to overwhelming myocardial infarction related anxiety, which might further intensify myocardial infarction related pain perception.

Methods: The aim of the study was to investigate the impact of trait anxiety and severe fear of COVID-19 on the perception of pain during the acute event of myocardial infarction. Therefore, differences in myocardial infarction related pain perception should be related not only to acute myocardial infarction related anxiety, but also to pre-existing trait anxiety and to the burden of the COVID-19 pandemic. Patients who experienced low levels of pain at the AMI were compared with those who experienced high levels of pain, regarding differences in state and trait anxiety, as well as in fears of COVID-19. For this, the visual analog scale (VAS) pain scale was used to measure remembered pain intensity in the acute phase of the AMI. Based on the VAS pain scale the participants were divided into low and high AMI related pain groups using the sample's median. The State-Trait-Anxiety Inventory (STAI) was used to assess AMI-related state anxiety as well as pre-existing trait anxiety. Furthermore, health-related questions about COVID-19 were asked to assess the severity of fear of COVID-19.

Results: 130 patients were assessed for mood and anxiety symptoms after admission to the hospital due to AMI. STAI-Trait anxiety scores of AMI patients who experienced more pain were significantly higher than those who experienced the AMI with less pain (t-Test for independent samples: $t(111) = -2.621$, $p = 0.01$). However, there were no significant differences in state anxiety scores when differences in pain were considered (t-Test for independent samples: $t(109) = -0.592$, $p = 0.56$). Furthermore, the statistical analyses revealed significant differences in fears of COVID-19. AMI patients who experienced less pain during AMI showed higher COVID-19 related anxiety scores compared to those who experienced higher pain (t-Test for independent samples: $t(111) = 2.146$, $p = 0.03$). The results indicate significant differences in pre-existing trait anxiety, as well as in fear of COVID-19 when patients experience different levels of pain during the AMI. Patients with higher pain at the time of AMI showed higher pre-existing trait anxiety, but less fear of COVID-19. There were no significantly different state anxiety scores in AMI patients.

Conclusion: In cause of the bidirectional relationship between pain and anxiety it seems even more exciting to discuss the connections in more detail. Our data shows a relationship between increased trait anxiety and increased severity of perceived pain, which is in line with other publications. Higher pain scores are associated with lower COVID-19 anxiety levels. This may support the hypothesis that patients with high COVID-19 related fear are more likely to avoid a hospital even in the acute setting and therefore classify pain as less severe. Another explanation could be that although pre-existing trait anxiety increases pain perception in principle, a severe stress situation caused by fear of COVID-19 could possibly induce so-called stress-induced analgesia. This causes pain to be dampened and one does not perceive it to the normal extent. It has already been demonstrated in borderline patients that in a state of high tension, which (borderline) patients frequently experience as well as extremely unpleasant, pain sensitivity is additionally reduced. Future studies should therefore look more

closely on the causes of differential pain perception, particularly in the COVID-19 pandemic or other pandemics.

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2-6

Tele-Covid-Tirol: Experiences of a telemedical surveillance programme in the course of the Covid-19 pandemic

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Introduction: For almost two years, the Covid-19 pandemic has posed a challenge to healthcare systems. After a brief stabilization in the summer of 2021, Austria was confronted with another, much more severe wave of disease in the fall. The telemedical care program Tele-Covid-Tirol, which had been installed during the previous Covid-19 wave, proved its worth in monitoring high-risk patients in home isolation: on the one hand, close monitoring enables early detection of deterioration of the disease, timely intensification of therapy and thus prevention of necessary intensive care stays. On the other hand, if the course of the disease is stable, unnecessary hospital admissions can be prevented and thus relieving the burden on the healthcare system.

Methods: Patient acquisition is done in collaboration with the Tyrolean Health Department, primary care physicians, or through private contacts by phone or email. Covid-19-positive high-risk patients (age >65 years and/or severe comorbidities) from the greater Innsbruck area are fitted with a Cosinuss® Home Monitoring System. The ear sensor measures SpO2, respiratory rate, body temperature and heart rate. The monitoring team (25 medical students under the supervision of 6 physicians) provides continuous monitoring of vital signs (24/7). After validation of the measurements, the collected parameters are evaluated with the help of a specially developed risk score. If a predefined risk score is exceeded, the patient is contacted by telephone. The combination of the clinical condition and the risk score determines the further course of action: (a) watchful waiting, (b) notification of the primary care physician, or (c) referral to our center for therapy optimization.

Results: The program was started in December 2020. After 6 months, the program was temporarily paused. During this first period, 48 patients (age 74.5 IQR: [60–81]; 37.5 % male) were monitored. At the end of November 2021, the program was reactivated and is still running. Since the start of the second period, 68 patients (age 73.5 IQR: [68.3–79.8]; 44.1 % male) participated in the program. Comparing the patient populations of the two periods, a significant decrease in hospitalizations (29.2 % versus 7.4 %; $p < 0.005$) was observed in the second period. 60.2 % of the patients in the second period were immunized with at least two dosages of Covid-19 vaccines before infection. Four out of five (80 %) of hospitalized patients were not vaccinated.

Conclusion: The telemedical care program Tele-Covid-Tirol can monitor a large number of high-risk patients in domestic isolation. The striking decrease in hospitalization rate in the second monitor phase is probably a consequence of the higher vaccination rate in the population. It is also possible that the currently predominant Covid-19 subtype B.1.1.529 (Omikron) is associated with a more favorable disease course. A comprehensive analysis is planned after completion of Tele-Covid-Tirol.

2-7

Longitudinal analysis of lung perfusion SPECT/CT in hospitalized patients due to Covid-19 infection

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Introduction: Acute pulmonary embolism (PE) has been reported as the most frequent complication of COVID-19 infection in the form of predominantly small vessel thrombosis that can be underestimated on CT pulmonary angiogram. Ventilation (V)-Perfusion (Q) SPECT/CT has been validated to establish PE even in the presence of pneumonia. Prophylactic use of parenteral anticoagulants during COVID-19 hospitalization is recommended. However, there are conflicting data about post-hospital discharge extended anticoagulation therapy.

Methods: All patients who underwent a Q SPECT/CT scan prior to discharge from the hospital due to severe COVID-19 disease were included in a prospective 3-month observational study. The finding of one or more segmental perfusion defects outside the area of inflammation was considered a positive finding and patients were discharged with continued anticoagulant therapy. A subsegmental perfusion defects or segmental in the area of pneumonic inflammation was considered a negative finding. Patients continued the protective dose of acetylsalicylic acid 100 mg daily. According to the decision of the attending physician, prolonged corticosteroid therapy was continued in some patients. Over a 3-month period all patients underwent a control Q SPECT CT/scan, pulmonary function tests, MSCT of the thorax, control laboratory tests (D-dimers, NTproBNP, ferritin, C-reactive protein) as well as a pulmonologist examination to evaluate symptoms.

Results: 104 patients with severe COVID-19 hospitalized for more than 14 days due to prolonged oxygen therapy, had the first (Q) SPECT/CT before discharge and the second after 3 months. Before discharge, 49 patients (47 %) had at least one segmental perfusion defect outside the area of inflammation and were discharged with continued anticoagulant therapy, mainly Rivaroxaban. For comparison, 35 patients (34 %) with subsegmental or segmental perfusion defects in the area of inflammation were treated with acetylsalicylic acid (ASS) 100 mg daily. 20 patients (19 %) had normal perfusion. Out of 78 patients (pts) who completed the study, 48 perfusion scans improved after discharge. Anticoagulation therapy, in particular Rivaroxaban, was significantly associated with improvement of perfusion defects outside the inflammation areas (OR 8.63, CI 2.86–29, $p < 0.001$). Cortisone therapy and ASS did not affect perfusion.

Conclusion: The majority of patients with severe COVID-19 infection show perfusion defects, but almost half of them present with segmental defects outside of inflammation. Treatment with Rivaroxaban leads to a significant improvement of perfusion after 3 months.

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Postersitzung 3 – Chirurgie 1

3-1

St Thomas Hospital polarizing cold cardioplegia does not have superior effects on hemodynamic parameters in an infarcted rat model

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Introduction: The use of cardioplegic solutions is indispensable during cardiac arrest in order to reduce myocardial metabolism and oxygen demand. Most commonly, hypothermic hyperkalemic cardioplegic solutions are used for open heart surgery. However, high potassium concentrations have several effects that limit left ventricular recovery, such as intracellular calcium overload resulting in the loss of contractility and increased cell death. Recently, we have shown that polarized cardiac arrest results in similar myocardial protection and improves cardiac functional recovery in a porcine model of cardiopulmonary bypass. The purpose of this study was to identify and compare the hemodynamic effects of cold St Thomas' Hospital polarizing cardioplegia (STH-Pol) in contrast to standard St Thomas' Hospital cardioplegia (STH2) in rats with chronic myocardial infarction. We hypothesize that St Thomas' Hospital polarizing cardioplegia shows superior protection on left ventricular hemodynamic recovery as compared to standard STH2 cardioplegia.

Methods: Permanent myocardial infarction was induced by permanent occlusion of the left anterior descending artery LAD on Sprague-Dawley rats (593 ± 65 g, day of sacrifice). Six weeks post-MI, after echocardiography assessment, the animals were sacrificed, and hemodynamic parameters were measured in an erythrocyte-perfused isolated heart model (STH2, control group: $n=5$ or STH-Pol, study group: $n=4$). Fifteen minutes of Langendorff mode and 30 min of Working-heart mode were followed by cardiac arrest with the two types cardioplegia (was applied three times every 20 min (t1 = 0, t2 = 20, t3 = 40)). STH-Pol, consisting of esmolol, adenosine and magnesium, was mixed with erythrocyte-buffer shortly prior to administration (1:4). After ischemia, the hearts were started with a hot shot with warm erythrocyte-buffer. Hemodynamic parameters were measured every five minutes in Langendorff mode and Working-heart mode. Finally, pump function was examined and tissue samples were taken for analysis of troponin-T and high-energy phosphates. Results will be given as % of preischemic baseline value.

Results: The use of STH-Pol instead of STH2 did not yield any significant differences in hemodynamic recovery (%) across the parameters of left atrial flow (LAF: 40.87 ± 13.22 vs. 53.24 ± 11.27), coronary flow (CF: 58 ± 14.36 vs. 76.21 ± 9) and cardiac output (CO: 42.82 ± 13.36 vs. 51.83 ± 11.76). Furthermore, we have not been able to identify superior effects of STH-Pol on stroke volume (SV: 46.55 ± 13.91 vs. 52.66 ± 11.33) recovery. Moreover, heart rate was comparable in both groups (92.07 ± 2.02 vs. 99.35 ± 1.72), which indicates swift reversal of negative chronotropic effects of esmolol.

Conclusion: Polarizing cardioplegic arrest does not show superior effects on hemodynamic parameters of left ventricular recovery after ischemia in chronically infarcted rat hearts as compared to depolarizing cardioplegic arrest.

3-2

Long-term outcomes after surgical repair of subvalvular aortic stenosis in pediatric patients

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Introduction: Subvalvular aortic stenosis (SAS) is a rare, but progressive disease. The disease spectrum spans from a minor fibrous ridge on the subvalvular ventricular septum (discrete SAS) to a narrow fibromuscular tunnel-like obstruction of the left ventricular outflow tract (LVOT). Aortic regurgitation is common, due to the turbulent blood flow causing damage, scarring and prolapse of the aortic valve, or alternatively, due to direct extension of the subaortic tissue onto the aortic valve leaflets. Long term outcomes in children concerning late reoperation and valve insufficiency requiring valve repair or replacement remain incompletely defined. Therefore, we reviewed our long-term single-center experience with repair of SAS in pediatric patients. The primary endpoints were mortality and re-operation for recurrence of SAS or aortic valve surgery.

Methods: A chart review of all patients less than 18 years of age at time of surgery who underwent repair for SAS between May 1985 and April 2020 was conducted. During the study period 112 patients underwent 133 SAS repairs. Mortality was cross-checked with the national health insurance data base providing a mortality follow-up until April 2020. Seven patients were transferred from foreign countries and could not be cross-checked in the database. These patients were censored at the last follow-up at the center.

Results: From May 1985 until April 2020, 112 patients (53.6 % male, 17.9 % hypertrophic obstructive cardiomyopathy (HOCM), 22.3 % bicuspid aortic valve) underwent 133 SAS repairs. SAS repair was performed as following: Myectomy: 30

(22.6 %); membrane resection: 50 (37.6 %); membrane resection and myectomy: 42 (31.6 %); modified Konno procedure: 11 (8.3 %). Median age at time of surgery was 6.2 years (IQR 2.3–10.7). Concomitant aortic valve repair was performed in 19 (14.3 %) cases and concomitant aortic valve replacement in 9 (6.8 %) cases. In 9 (6.8 %) cases concomitant right ventricular outflow tract myectomy was necessary. There were 7 early deaths and 3 late deaths. All early deaths occurred in patients with complex congenital heart disease or HOCM. Kaplan-Meier estimated survival was 91.3 % ± 2.8 % at 10 years and 89.2 % ± 3.4 % at 20 and 30 years. Two patients with HOCM underwent cardiac transplantation 0.9 years and 12.4 years after initial SAS repair respectively. Freedom from re-operation for subvalvular aortic stenosis was at 75.8 % ± 4.5 % at 10 years and 66.3 % ± 6.1 % at 20 and 30 years. Freedom from any aortic valve re-operation (repair and replacement) was 87 % ± 3.6 % at 10 years and 81.5 % ± 5.3 % at 20 and 30 years.

Conclusion: Recurrence and re-operation rates remain a concern in pediatric patients with SAS. Close long-term follow up is warranted in these patients, though overall survival is good. The modified Konno procedure is an excellent treatment option in patients with tunnel-like SAS. Re-operation for SAS was associated with younger age at time of surgery (HR 0.9 for each increase in year; $p = 0.021$). SAS re-operation rate plateaus 10 years after surgery.

3-3

First report: Sharp dissection commisurotomy in a beating heart to enhance bioprosthetic unfolding during transcatheter mitral valve replacement

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Introduction: The recent CE mark release of a transapical beating-heart transcatheter mitral valve replacement (TMVR) system incorporating an anchored apical tether expands the therapeutic options for patients with multiple co-morbidities at high surgical risk. While balloon valvuloplasty is often adequate to enable sufficient unfolding of the self-expandable bio-prosthesis, complex patients with severe mitral annular pathology and/or leaflet calcification may benefit from enhanced enlargement techniques as first reported herein.

Methods: A 71-year-old woman presenting with severe symptomatic, post rheumatic mitral stenosis involving a heavily degenerated valve and significant fusion between A1/P1 and A2/P2 (MPG: 11 mm Hg, sPAP: 50 mm Hg, NYHA III; EuroScore II and STS Score of 1.8 % and 3.7 %) along with metastatic uterine cancer was referred to our Heart Team. We determined a rather good long-term prognosis could be achieved with a successful interventional TMVR treatment. Standard transapical access was performed on her beating heart with circulating blood. A positive floating maneuverer confirmed correct positioning of the guide wire in the left atrium. A neuroprotection device was deployed. After balloon-valvuloplasty (26 mm non-compliant balloon) yielded almost no effect, we performed a novel remote commissurotomy using endoscopic scissor mediated sharp dissection. A shortened large bore introducer sheath (length approximately 20 cm) was exchanged at the transapical access site. Under echocardiographic and fluoroscopic guidance, an endoscopic scissor was passed into the left ventricle with careful regard to avoid the subvalvular apparatus. With subsequent alignment and positioning enabling both opened scissor tips to be well visualized by 3D echocardiography, the commissurotomy cut was completed. After switching to the implant introducer sheath, the larger mitral valve opening allowed the bioprosthesis to fully self-expand.

Results: Despite unsatisfactory balloon valvuloplasty, this complex patient recovered well. The implanted tri-leaflet valve showed good valvular function (MPG 4 mm Hg) with only a mild, clinically insignificant, paravalvular leakage. While computed tomography recorded a minor stroke, no residual symptoms were demonstrated at discharge on day 21.

Conclusion: This is the first known report of a beating-heart transapical scissor mediated mitral commissurotomy prior successful TMVR. The new technique proved to be an excellent bail-out strategy after unsuccessful balloon valvuloplasty. Since, thromboembolic risk may be increased due to the added step of cutting stenotic valvular tissue, neuroprotection is strongly recommended during the procedure.

3-4

The role of telocytes and telocyte-derived exosomes in the development of thoracic aortic aneurysm

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Abstract: A hallmark of thoracic aortic aneurysms (TAA) is the degenerative remodeling of aortic wall, which leads to progressive aortic dilatation and resulting in an increased risk for aortic dissection or rupture. Telocytes (TCs), a distinct type of interstitial cells described in many tissues and organs, were recently observed in the aortic wall, and studies showed the potential regulation of smooth muscle cell (SMC) homeostasis by TC-released shed vesicles. The purpose of the present work was to study the functions of TCs in medial degeneration of TAA. During aneurysmal formation an increase of aortic TCs was identified in a murine aneurysm model and in human surgical specimens of TAA-patients, compared to healthy tho-

racic aortic (HTA)- tissue. We found the presence of epithelial progenitor cells (EPCs) in the adventitial layer, which showed increased infiltration in TAA samples. For functional analysis, HTA- and TAA-telocytes were isolated, characterized, and compared by their protein levels, mRNA- and miRNA-expression profiles. We detected TC and TC-released exosomes near SMCs. TAA-TC-exosomes showed a significant increase of the SMC-related dedifferentiation markers KLF-4-, VEGF-A-, and PDGF-A-protein levels, as well as miRNA-expression levels of miR-146a, miR-221 and miR-222. SMCs treated with TAA-TC-exosomes developed a dedifferentiation-phenotype. In conclusion, the study shows for the first time that TCs are involved in development of TAA and could play a crucial role in SMC phenotype switching by release of extracellular vesicles.

3-5

A novel endothelial damage inhibitor reduces oxidative stress and improves cellular integrity in radial artery grafts for coronary artery bypass

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Abstract: The radial artery (RA) is a frequently used conduit in coronary artery bypass grafting (CABG). Endothelial injury incurred during graft harvesting promotes oxidative damage, which leads to graft disease and graft failure. We evaluated the protective effect of DuraGraft®, an endothelial damage inhibitor (EDI), on RA grafts. We further compared the protective effect of the EDI between RA grafts and saphenous vein grafts (SVG). Samples of RA ($n=10$) and SVG ($n=13$) from 23 patients undergoing CABG were flushed and preserved with either EDI or heparinized Ringer's lactate solution (RL). The effect of EDI vs. RL on endothelial damage was evaluated ex vivo and in vitro using histological analysis, immunofluorescence staining, Western blot, and scanning electron microscopy. EDI-treated RA grafts showed a significant reduction of endothelial and sub-endothelial damage. Lower level of reactive oxygen species (ROS) after EDI treatment was correlated with a reduction of hypoxic damage (eNOS and Caveolin-1) and significant increase of oxidation-reduction potential. Additionally, an increased expression of TGFb, PDGFa/b, and HO-1 which are indicative for vascular protective function were observed after EDI exposure. EDI treatment preserves functionality and integrity of endothelial and intimal cells. Therefore, EDI may have the potential to reduce the occurrence of graft disease and failure in RA grafts in patients undergoing CABG.

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3-6

Left ventricular dynamics during experimental hypovolemia and hypervolemia induced by lower body negative pressure

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Objective: Lower body negative pressure (LBNP) has been implemented as a tool to imitate systemic impacts of hypovolemia, orthostatic collapse and G load stress in humans. Blood is shifted from the heart towards the lower parts of the body in a predetermined fashion.

Methods: This study in 8 healthy pigs allows an insight into immediate alterations of volume shift during LBNP via invasive cardiac monitoring up to life threatening degrees of LBNP. Three different sealing positions and three different levels of negative pressures were evaluated. Real time measurements were performed with pressure-volume (PV)-catheters, acquired PV-loops and time intervals were analysed using CircLab® Software

Results: Cardiac output (CO), mean arterial pressure (MAP), central venous pressure (CVP), left ventricular cardiac power output (LV -CPO), stroke work, pressure volume area, maximal LV-pressure (LVPmax), LV-enddiastolic pressure (LV-EDP) and left ventricular-volume (LV-EDV) decreased during application of LBNP (hypovolemia). No significant effects were observed for heart rate (HR), left ventricular end-systolic volume, ejection fraction (EF) or end-systolic elastance. PV loops shifted leftwards and downwards with stimulus intensity. Abrupt release of LBNP (hypervolemia) increased CO, MAP, LV-CPO within three respiratory cycles. LV-EDP, LV-EDV, LV end-systolic volume and LVPmax reached baseline values without volume overload. LV-EF and HR remained stable.

Conclusion: LBNP causes preload-dependent hemodynamic alterations, conditioned by the extent and the level of negative pressure applied. Abrupt release of LBNP immediately restores stable hemodynamics without cardiac impairment due to rapid volume overload.

3-7

Frozen Elephant Trunk technique in acute aortic syndromes – A single center experience

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Objective: To evaluate our experience with the FET method in acute aortic syndromes.

Methods: Between 2003 and 2020, 240 patients underwent emergency surgery for acute aortic syndromes at our department. Patients with standard care were assigned to a nonFET group, patients with FET to a FET group.

Results: GERAADA Score was 19.3 % and 21.2 %. Mean age was 61 ±13.7 and 59 ±12.1yrs. No differences in BMI and preoperative creatinine values were present. Aortic regurgitation grade ≥3 was present in 11.69 % and 12.90 %. Preoperative stroke was present in 3.98 % and 9.09 %. Stanford Type A dissection was present in 97.84 % and 71.43 %. Whereas Stanford Type B and Type nonAnonB dissections lead to surgery in 12.24 % and 16.33 % of FET cases. Overall, 50 patients were treated with the FET method. Concomitant root procedures were performed in 48 % and 36 %; $p = 0.2414$. Lowest body temperature (24.9 vs. 25.9 °C; $p = 0.0067$), SCP time (31 vs. 49min; $p = 0.0000$) and DHCA time (35 vs. 46min; $p = 0.0041$) differed, whereas CPB time (218 vs. 234min; $p = 0.2443$) and x-clamp time (117 vs 124min; $p = 0.5600$) did not. Rate of stroke was 13.64 % and 18.18 %; $p = 0.4747$. No difference in renal failure requiring haemodialysis (18.75 % vs. 13.64 %; $p = 0.5130$) was seen. Operative mortality was 18.42 % and 18.0 %; $p = 0.5586$.

Conclusion: In our series, the FET method did not increase mortality in the treatment of acute aortic syndromes compared to less aggressive arch treatment.

Postersitzung 4 – Herzinsuffizienz 1

4-1

Association between circulating renin concentration and right ventricular function in heart failure with reduced ejection fraction

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Introduction: Heart failure with reduced ejection fraction (HFrEF) is believed to be characterized by an overactivation of the renin-angiotensin-system (RAS). Increased levels of AngII, as the main effector of RAS, were associated with progressing decline in heart function and adverse remodeling. However the regulation and effects of RAS activation in HFrEF remain unclear, as 1. a majority of patients with HFrEF on GDMT do not show an excess in renin levels, 2. renin levels are not related to HF severity reflected by NT-proBNP and NYHA class 3. renin levels do not seem to be able to provide therapeutic useful information on the effectivity of RAS blockade and 4. only excessive renin levels seem to be associated with worse outcome. Renin secretion by the kidneys is mainly regulated by renal perfusion pressure and sodium levels. Especially right ventricular (RV) impairment is associated with poor outcomes in HFrEF. We have hypothesized that impaired RVF leading to backward failure with reduced renal perfusion pressure results in excessive renin secretion and is thereby associated with worse prognosis. The aim of this study was to relate RVF measured by a sophisticated echocardiographic exam with renin levels and to investigate predictors of renin levels.

Methods: Chronic HFrEF patients undergoing routine ambulatory care were consecutively enrolled in a prospective, registry-based, observational study at the heart failure outpatient unit. Medical history, comorbidities, current medication and parameters on clinical status and functional capacity, laboratory parameters, including cardiac specific markers renin, aldosterone and NT-proBNP and echocardiographic examination were documented. Patients with echocardiographic exams at ±30 days to renin values have been included. Echocardiographic exams were reread and various parameters of RVF, i. e. tricuspid annular plane systolic excursion (TAPSE), RV-tissue doppler imaging (TDI), RV-strain, fractional area change (FAC) and RV-end diastolic diameter (RVEDD) were assessed. Relationship of renin with parameters were analyzed as continuous data or as within-population tertile strata. For all tests two-sided P-values lower 0.05 were considered to indicate statistical significance.

Results: A total of 247 patients with chronic HFrEF were enrolled. Detailed baseline characteristics are displayed on Table 1. Median NT-proBNP was 1719 pg/ml (IQR: 585–3690). Median left ventricular ejection fraction was 30 % (IQR: 24–39) and median renin level was 147.7 µIE/m (IQR: 23.8–627.2). Plasma renin concentration was not associated with HF severity reflected by NYHA functional class ($p = 0.165$), and NT-proBNP ($r = 0.042$, $P = 0.513$) (Fig. 1a). Renin levels correlated significantly with systolic blood pressure ($r = -0.475$; $p < 0.001$), serum sodium ($r = -0.210$; $p = 0.001$) and echocardiographic parameters FAC ($r = -0.200$; $p = 0.002$), TAPSE ($r = -0.248$; $p < 0.001$), TDI ($r = -0.217$; $p = 0.004$) RA area ($r = 0.129$; $p = 0.048$) and vena cava

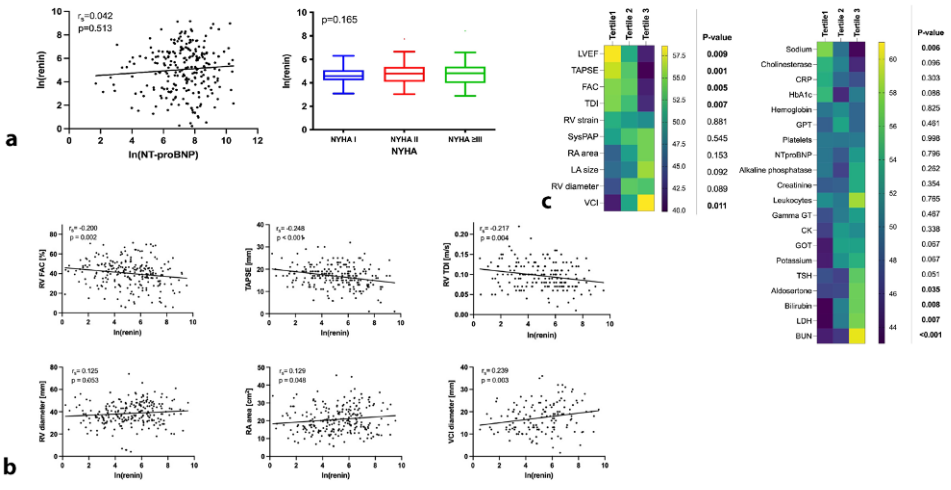

Fig. 1 | 4-1 Association of plasma renin concentration with HFrEF severity (**a**), right ventricular functional (**b, c**) and laboratory parameters (**c**)

	Total study population (n=247)
Age, median years (IQR)	61 (51-73)
Male sex, n (%)	193 (78)
BMI, kg/m² (IQR)	28.3 (24.5 – 31.3)
BPsyst, mmHg (IQR)	122 (110 – 140)
BPdia, mmHg (IQR)	75 (70 – 85)
Heart rate, bpm (IQR)	68 (60 – 76)
Comorbidities	
Diabetes mellitus, n (%)	84 (34)
Arterial hypertension, n (%)	115 (47)
Ischemic etiology of HF, n (%)	130 (53)
Atrial fibrillation, n (%)	54 (22)
NYHA functional class	
NYHA ≤ I, n (%)	47 (19)
NYHA II, n (%)	114 (46)
NYHA III, n (%)	83 (34)
Laboratory parameters	
Hemoglobin, g/dl (IQR)	13.5 (11.7 – 14.4)
Leucocytes, G/l (IQR)	7.30 (6.05 – 8.55)
Sodium, mmol/l (IQR)	139 (137 – 141)
Potassium, mmol/l (IQR)	4.73 (4.32 – 5.15)
Bilirubin, mg/dl (IQR)	0.59 (0.38 – 0.92)
Cholinesterase, kU/l (IQR)	6.66 (5.59 – 8.09)
Gamma-GT, U/l (IQR)	43 (23 – 95)
LDH, U/l (IQR)	191 (161 – 230)
AST, U/l (IQR)	24 (18 – 29)
ALT, U/l (IQR)	23 (16 – 32)
Albumin, g/l (IQR)	43.0 (39.6 – 45.6)
Creatinine, mg/dl (IQR)	1.16 (0.92 – 1.64)
BUN, mg/dl (IQR)	23.5 (17.00 – 33.8)
NT-proBNP, pg/ml (IQR)	1685 (585 – 3690)
Aldosterone, pg/dl (IQR)	104 (55 – 196)
Renin, µIE/m (IQR)	189.9 (37.2 – 685.3)
HbA1c, % (IQR)	5.3 (0.0 -5.9)
Echocardiographic measurements	
Left ventricular ejection fraction, % (IQR)	30 (24 – 39)
Right ventricular end-diastolic diameter, mm (IQR)	38 (32 – 46)
Right ventricular function (visual)	
Mildly reduced, n (%)	59 (24)
Moderately reduced, n (%)	64 (26)
Severely reduced, n (%)	27 (11)
Right atrial area, mm² (IQR)	20 (15 – 26)
TAPSE, mm (IQR)	17 (14 – 20)
Right ventricular TDI (S'), cm/s (IQR)	0.10 (0.07 – 0.12)
Right ventricular FAC, % (IQR)	41.9 (32.2 – 48.4)
RVFW-GLS, % (IQR)	15 (10 – 21)
SysPAP, mmHg (IQR)	46 (36 – 59)
Vena cava inferior diameter, mm (IQR)	18 (13 – 22)
Heart failure medication	
ACEi/ARB/ARNi, n (%)	99/46/81 (40/19/33)
Beta-blockers, n (%)	222 (90)
MRA, n (%)	178 (72)
Ivabradine, n (%)	30 (12)
Loop diuretics, n (%)	102 (41)

Values are median (interquartile range (IQR)) or n (%).

ACE – angiotensin converting enzyme inhibitor; ALT – alanine aminotransferase; ARB – angiotensin receptor blocker; ARNi – angiotensin receptor-neprilysin inhibitor; AST – aspartate aminotransferase; BB – beta blocker; BMI – body mass index; BP – blood pressure; bpm – beats per minute; HFrEF - heart failure with reduced ejection fraction; HR – heart rate; IQR – interquartile range; LVEF – left ventricular ejection fraction; MRA – mineralocorticoid receptor antagonist; NT-proBNP – N-terminal pro B-type natriuretic peptide; NYHA – New York Heart Association,

Fig. 2 | 4-1 Baseline characteristics of total study population ($n=247$)

inferior (VCI) diameter (r = –0.239; $p=0.003$) (Fig. 1b). Color-coded heatmaps (Fig. 1c) show alterations of echocardiographic parameters and laboratory parameters for different renin tertiles, with worsening of the respective parameter by increasing renin levels. When entering variables for clinical, echocardiographic and laboratory cluster into a linear logistic regression model, blood pressure, creatinine, urea with beta-coefficients and GGT as well as RV size and RV function (TDI) with moderate beta-coefficients were found to be the significant predictors for renin levels with an overall R^2 of 0.93.

Conclusion: Circulating renin levels are unrelated to classical indices of HF severity as NT-proBNP and NYHA class. Renin levels increase with worsening of RVF assessed by echocardiography. Morphologic and functional RV parameters were significant predictors of renin levels besides known regulators suggesting that excessive renin levels and worst outcome develop in patients with RVF decline representing end-stage heart failure.

4-2

Funktionelle Effekte von selektiver HDAC-Inhibition auf humanes atriales Myokard

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Einleitung: Herzinsuffizienz (HI) stellt eine große Herausforderung sowohl für Patienten als auch das Gesundheitssystem aufgrund der hohen Prävalenz, Morbidität und Mortalität dar. Obwohl rezente Fortschritte die Mortalität, Leistungsfähigkeit und Lebensqualität bei HI verbesserten, ist die Prognose schlecht. Histon-Deacetylasen (HDAC) sind Enzyme, welche Acetylgruppen von verschiedenen Proteinen entfernen und so sowohl bei der Regulation der Genexpression als auch bei der posttranslationalen Modifikation, eine entscheidende Rolle spielen. HDAC Inhibitoren (HDACi) zeigte antiproliferative und kardioprotektive Effekte in präklinischen Studien. Nichtselektive HDAC-Inhibition kann jedoch zu unerwünschten Nebenwirkungen, wie Myelosuppression und gastrointestinale Symptome verursachen, was die Verwendung in der klinischen Praxis limitiert. Deshalb liegt der Fokus nun auf Isoform-selektive HDAC-Inhibition, welche effektiv und sicherer in der Anwendung sein sollten. Ziele: Das Ziel der Studie ist die Untersuchung funktioneller Effekte Isoform-selektiver HDACi auf humanes atriales Myokard.

Methoden: Atriale humaneTrabekel wurden isoliert, mit 1 Hz elektrisch stimuliert und bis zu einer optimalen Länge (BL) gedehnt. Nach Erreichen des „Steady-States", wurden die Trabekel entweder mit HDAC-A, HDAC-B oder DMSO (crtl) für 2 h inkubiert. HDAC-A und HDAC-B sind kommerziell nicht erhältliche Klasse I HDACi, die jeweils HDAC 1 +2 und HDAC 1 +2 +3 hemmen. Funktionelle Parameter (systolische und diastolische Kraft, Kinetik) wurden kontinuierlich aufgezeichnet und analysiert. Die Experimente wurden unter Verwendung jeweils eines Protokolls in niedriger (HDAC-A 2 µM, $n=7$; HDAC-B 100 nM, $n=13$), und hoher Dosierung (HDAC-A 10 µM, $n=8$; HDAC-B 250 nM, $n=7$), sowie in einer Kontrollgruppe (DMSO 10 µM, $n=8$) durchgeführt.

Resultate: In den hohen Dosierungen zeigten beide HDACi einen signifikanten, akuten Anstieg der entwickelten Kraft (HDAC-A 10 µM: 94,5 %±11,3 %, HDAC-B 250 nM: 100,2 %±7,7 %, ctrl: 70,7 %±5,4 %; $p<0,05$) verglichen zur Kontrollgruppe. Die diastolische Spannung unterschied sich nicht zwischen den Gruppen. Des Weiteren wurden die Kontrak-

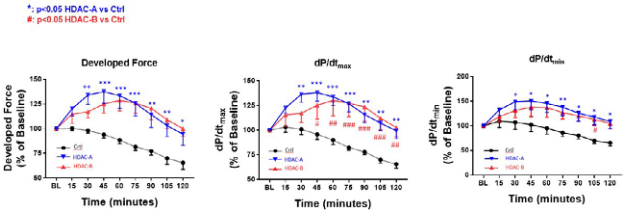

Abb. 1 | 4-2

tions- und Relaxationsgeschwindigkeiten mit HDACi beschleunigt (dP/dtmax und dP/dtmin). HDACi in niedriger Dosierung zeigte ähnliche, aber weniger stark ausgeprägte Effekte.

Schlussfolgerungen: Isoform-selektive HDACi führte zu einem dosis-abhängigen akuten Anstieg der Kontraktilität und beschleunigt Relaxationsparameter im humanen atrialen Myokard. Selektive HDACi, welche direkt die diastolische und systolische Funktion modulieren, könnte eventuell eine vielversprechende therapeutische Option zur Behandlung von Patienten mit Herzinsuffizienz sein.

4-3

Temporal evolution of the key neurohumoral regulator renin in chronic stable HFrEF

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Introduction: Renin is the enzyme catalyzing the rate-limiting step of the Renin-Angiotensin-System (RAS) generating Angiotensin II (AngII). AngII formation is the primary step of a cascade with further generation of angiotensin metabolites as Ang1–7 or AngIII. RAS inhibitors aiming to block the deleterious effects of AngII are the main pillar of current heart failure with reduced ejection fraction (HFrEF). Although used since over 30 years in clinical routine, the effects of RAS-inhibitors on renin status, temporal evolution of renin levels during the course of HFrEF and impact of the dynamic of renin levels on patient outcomes, especially under current guideline directed medical therapy (GDMT), are lacking. The present study aims to evaluate (i) RAS regulation under GDMT, (ii) relationship of renin with HF severity and medication use, (iii) temporal evolution of renin levels and (iv) effect of renin dynamics on outcomes in stable outpatient HFrEF patients.

Methods: Consecutive patients with stable chronic HFrEF and GDMT have been enrolled prospectively from the outpatient unit of heart failure at the Medical University of Vienna between June 2013 and August 2021. Routine laboratory parameters including N-terminal pro B-type natriuretic peptide (NT-proBNP) and active plasma renin concentration (ARC) have been measured by specific immunoassays at routine clinical visits. Renin levels were documented for all patients consecutively at first measurement, i. e. baseline, and at follow-up visits at 12 ± 6 months, 24 ± 6 months, 36 ± 6 months, 48 ± 6 months and 60 ± 6 months, respectively. Baseline renin levels were correlated with NT-proBNP and compared between NYHA class and HF medication use. Comparison was further performed for renin levels between baseline and different follow-up (FUP) timepoints. To assess the effect of changes in renin levels

	Stable HFrEF patients (n=509)
Active renin concentration, uIE/ml (IQR)	126 (27-628)
Basic demographics	
Age, years (IQR)	62 (52-72)
Male gender, n (%)	391 (77%)
BMI, kg/m² (IQR)	27.2 (23.8–31.4)
Systolic blood pressure, mmHg (IQR)	125 (112-140)
Heart rate, bpm (IQR)	71 (61-81)
NYHA class, I / II / III+ (%)	64 (13%) / 248 (50%) / 187 (37%)
Comorbidities	
Ischemic etiology of heart failure, n (%)	277 (54%)
Diabetes mellitus, n (%)	185 (36%)
Arterial Hypertension, n (%)	277 (54%)
History of cancer, n (%)	68 (13%)
Medication and device therapy	
Beta-blocker, n (%)	470 (94%)
ACEi/ARB/ARNi, n (%)	248 (49%) / 109 (21%) / 118 (23%)
MRA, n (%)	378 (76%)
SGLT2-inhibitors, n (%)	31 (19%)
Ivabradin, n (%)	38 (10%)
Loop diuretics, n (%)	241 (49%)
ICD / CRT, n (%)	213 (43%) / 142 (30%)
Laboratory parameters	
NT-proBNP pg/ml (IQR)	1764 (801–3637)
Creatinine, mg/dl (IQR)	1.14 (0.94-1.56)
BUN, mg/dl (IQR)	23 (17-32)
Sodium, mmol/l (IQR)	140 (138-142)
Albumin, g/l (IQR)	43.9 (41.2-46.1)
BChE, kU/l (IQR)	7.08 (5.79–8.31)
AST (GOT), U/l (IQR)	24 (19-30)
ALT (GPT), U/l (IQR)	23 (17-33)
GGT, U/l (IQR)	42 (24-87)
Bilirubin, mg/dl (IQR)	0.57 (0.39-0.80)
Total cholesterol, mg/dl (IQR)	159 (126-191)
Hemoglobin, g/dl (IQR)	13.6 (12.3-14.8)
Thrombocytes, G/l (IQR)	220 (177-263)
Leukocyte count, G/l (IQR)	7.57 (6.35-9.04)
C-reactive protein, mg/dl (IQR)	0.29 (0.13-0.67)

BMI – body mass index; ICD – intracardiac defibrillator; CRT – cardiac resynchronization device. ACEi – angiotensin-converting enzyme inhibitor; ARB – angiotensin receptor blocker; ARNI – angiotensin receptor-neprilysin inhibitor; MRA – mineralocorticoid receptor antagonist; SGLT2 – sodium-glucose co-transporter-2; BChE – Butyrylcholinesterase.

Fig. 2 | 4-3 Baseline characteristics

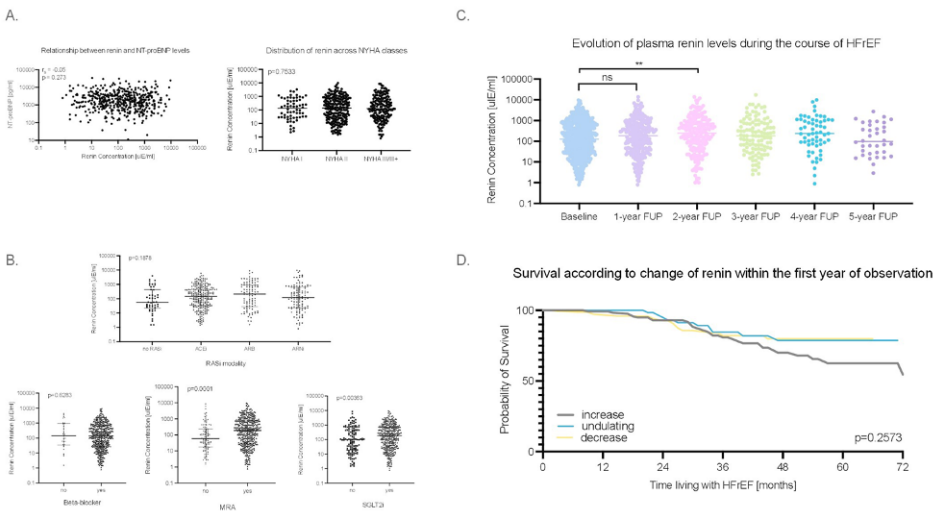

Fig. 1 | 4-3 **a** Association between renin and NT-proBNP and NYHA classes. **b** Renin levels according to neurohumoral therapy. **c** Evolution of plasma renin levels, baseline to 5-year follow-up. **d** Survival according to change of renin within the first year of observation (increase = change >50 %, undulating = change between –50 to 50 %, decrease = change>–50 % from baseline)

patients were categorized into three groups based on the change of renin from baseline within the first year of observation, i. e. decrease = change>–50 %, undulating = change between –50 to 50 %, increase = change >50 % and survival curves were displayed as Kaplan-Meier plots and compared for different ARC categories by the log-rank test.

Results: A total of 491 patients were included in the study. Baseline characteristics are shown in Table 1. Patients were optimally treated with more than 90 % of patients using beta-blockers and RAS-inhibitors, 76 % of patients received MRAs. Median renin was 136.8 µIE/ml [IQR: 27.8–628.3]. Renin levels showed no relationship with HF severity reflected by a lack of correlation with NT-proBNP [rs = –0.05, $p = 0.273$] and comparable levels between NYHA groups [$p = 0.753$] (Fig. 1a). Renin levels were further comparable for different RAS inhibitors and patients with and without beta blockers, however higher in patients with mineralocorticoid receptor antagonists (MRA) [189 µIE/ml vs. 59 µIE/ml, $p = 0.0001$] and SGLT2-inhibitors [280 µIE/ml vs. 100 µIE/ml, $p = 0.0036$] (Fig. 1b). Renin levels at different yearly FUP timepoints are displayed in Fig. 1c. There was no significant difference in renin levels between baseline and 1-year FUP, while renin concentration was increased at 2-year FUP [126 µIE/ml vs. 243 µIE/ml, $p = 0.002$]. Baseline renin levels were not associated with survival [crude HR for an increase of 100 µiE/ml 1.014 (95 %CI: 0.997–1.032), $p = 0.102$]. Within one year following baseline, renin concentration decreased in 77 (27.6 %), was undulating in 74 (26.5 %) and increased in 128 (45.9 %) patients. Kaplan-Meier suggests worse survival for patients with increasing renin concentration though the difference was statistically not significant [$p = 0.2573$] (Fig. 1d).

Conclusion: Renin concentration is increased in patients with MRA and/or SGLT2-inhibitors. Renin tends to increase over time in stable HFrEF. Although RAS is the main target of HFrEF therapy, surprisingly there seems to be no strong association between RAS activation and thereby potential effectivity of achieved RAS-blockade and outcome.

4-4

Impacts on the short-term outcome of patients with Tako-Tsubo syndrome

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Introduction: Tako-Tsubo syndrome (TTS) is a form of acute heart failure which mostly affects postmenopausal women, often following an emotional or physical trigger factor. There are many hypotheses for the development of TTS but the complete pathophysiology still remains unclear. Although most patients recover after a few days, some have to be treated at the intensive care unit (ICU) and may even die from the condition. The aim of this study was to find out if pre-existing diseases, cardiovascular risk factors (CVRF), comorbidities and trigger factors have any impact on the short-term outcome of TTS patients.

Methods: Data of all patients who presented to our centre with TTS from 2009–2017 were gathered retrospectively. Baseline characteristics, including somatic and psychiatric pre-existing diseases, CVRF (smoking, hypertension, diabetes, dyslipidaemia) as well as physical and emotional trigger factors were collected. Somatic diseases were additionally categorised into cardiac, autoimmune, malignant and other diseases. Admission to intensive care unit post TTS and/or in-hospital death were defined as poor short-term outcome. Outcome predictors were analysed with binary logistic regression model using the combined variable admission to an ICU and/or death.

	ICU and/or death		
	All (n=102)	Odds Ratio (CI 95%)	p-value
Trigger factors			
Emotional trigger factor	30 (29,4%)	1,04 (0,34-3,19)	0,95
Physical trigger factor	23 (22,5%)	0,18 (0,06-0,66)	**0,003**
Comorbidities			
Psychiatric diseases	27 (26,5%)	2,39 (0,88- 6,48)	0,09
Somatic diseases (all)	88 (86,0%)	1,77 (0,36-8,55)	0,48
Cardiac diseases	23 (22,3%)	0,99 (0,32-3,05)	0,98
Malignant diseases	16 (15,6%)	1,26 (0,23-6,77)	0,96
Autoimmune diseases	18 (17,5%)	1,46 (0,38-5,59)	0,44
Other diseases	63 (61,2%)	0,70 (0,26-1,91)	0,49
Cardiovascular risk factors			
Hypertension	58 (56,3%)	0,89 (0,34-2,29)	0,80
Diabetes mellitus Type II	18 (17,5%)	0,79 (0,36-1,75)	0,57
Smoking	33 (32,4%)	0,82 (0,22-3,14)	0,78
LDL Cholesterol (mg/dl)	104 ± 36	1,01 (0,99-1,03)	0,35

Fig. 1 | 4-4 Binary logistic regression using the dependent variable ICU and/or death

Results: Out of 102 patients, 84 (82.4 %) were females and 18 (17.6 %) males with a median age of 65 ± 15 years. 30 (29.4 %) patients had an emotional and 23 (22.5 %) a physical trigger factor. 27 (26.5 %) had a psychiatric and 88 (86.3 %) a somatic pre-existing disease. 33 (32.4 %) patients were smokers, 58 (56.3 %) had hypertension and 18 (17.5 %) were diabetics. 19 patients (18.6 %) had to be admitted to ICU and 8 patients (7.8 %) died. No pre-existing diseases, baseline characteristics, CVRF or comorbidities behaved as significant independent predictor for bad outcome in TTS patients. Physical trigger was the only significant predictor for ICU admission and/or death ($p = 0.012$).

Conclusion: Our study showed that patients with physical trigger factors have a higher risk of ICU admission and/or death than patients with emotional or no stressful trigger factors before TTS and should therefore be monitored closely. Interestingly, contrary to many other acute cardiac diseases, pre-existing diseases, cardiovascular risk factors and comorbidities do not seem to have any impact on the short-term outcome of patients with TTS.

4-5

Neutrophile-lymphocyte ratio and outcome in Takotsubo Syndrome

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Introduction: Takotsubo syndrome (TTS) is an important form of acute heart failure with significant risk of acute complications and death. In this analysis we sought to identify predictors for in-hospital clinical outcome in TTS patients by concentrating on routine laboratory parameters at admission.

Methods: In this analysis from the Austrian national TTS registry, univariable and multivariable analyses were performed to identify significant predictors for severe in-hospital complications requiring immediate invasive treatment or leading to irreversible damage, such as cardiogenic shock, intubation, stroke, arrhythmias and death. Furthermore, the influence of identified predictors with long-term survival was evaluated.

Results: A total of 338 patients (median age 72 years, 86.9 % female) from 6 centres were included. Severe in-hospital complications occurred in 14.5 % of patients, including cardiogenic shock (9.8 %), death (3.3 %) and intubation (1.2 %), respectively. Patients with complications during the hospital stay had more prevalent chronic kidney disease (CKD), were less often previous smokers and TTS was less often preceded by an emotional trigger. C-reactive protein and neutrophile lymphocyte ratio (NLR) was higher in patients with complications, and midventricular ballooning and reduced left ventricular ejection fraction (LVEF) were more prevalent. In multivariable analysis, high NLR (OR 1.04 [95 % CI 1.02–1.07], $p=0.009$) and low LVEF (OR 0.92 [0.90–0.95] per %, $p<0.001$) remained significant predictors for severe in-hospital complications (Table 1). Both the highest NLR tercile and the lowest LVEF tercile were associated with significantly reduced 5-year survival (Fig. 1).

Conclusion: Low LVEF and high NLR at admission were independently associated with increased in-hospital complications and reduced long-term survival in TTS patients. NLR is a new easy-to-measure tool to predict worse short and long-term outcome after TTS.

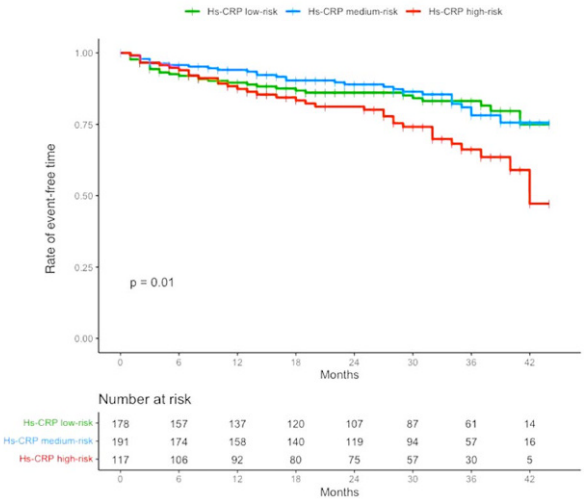

Fig. 1 | 4-5 Cumulative five-year mortality in NLR terciles

Table 1 | 4-5 Univariable and multivariable predictors of in-hospital complications

	univariable analysis		multivariable analysis	
parameter	OR (95 % CI)	*p* value	OR (95 % CI)	*p* value
age (per year)	1.01 (0.98–1.04)	0.437	1.00 (0.97–1.04)	0.822
chronic kidney disease	4.18 (2.03–8.41)	<0.001	1.84 (0.78–4.37)	0.164
previous smoker	0.11 (0.01–0.50)	0.006	0.13 (0.02–1.06)	0.056
emotional trigger	0.28 (0.08–0.72)	0.014	0.55 (0.17–1.76)	0.312
CRP at admission	1.01 (1.00–1.02)	0.002	1.01 (1.00–1.02)	0.076
NLR at admission	1.04 (1.02–1.07)	0.002	1.04 (1.01–1.08)	0.009*
LVEF (per %)	0.92 (0.90–0.95)	<0.001	0.93 (0.90–0.96)	<0.001*
apical ballooning	0.41 (0.20–0.79)	0.010	0.78 (0.08–7.50)	0.826
midventricular ballooning	2.51 (1.31–4.99)	0.006	0.98 (0.10–9.84)	0.986

* $p<0.05$ in multivariable analysis

4-6

A rare genetic cause of left ventricular hypertrophy and heart failure with preserved ejection fraction

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Introduction: The 59 year old women presented to the hypertrophic cardiomyopathy (HCM) clinic with progressive dyspnoea and typical angina. She had New York Heart Association (NYHA) class II to III symptoms. Medical reports indicated a history of arterial hypertension, type 1 diabetes and coronary artery diseases. Otherwise the past medical and surgical history were unremarkable. The physical exam showed signs of heart failure, but was otherwise normal. Transthoracic echocardiography demonstrated (TTE) significant left ventricular (LV) hypertrophy (LVH) with max. Diameter of 20 mm, normal LV ejection fraction (65 %) but elevated LV end-diastolic pressure and reduced global longitudinal strain with apical sparing. Cardiac MRI confirmed the significant LVH and showed late gadolinium enhancement in the subepicardial and mid-myocardial region of the apical and mid-ventricular wall. There was no evidence for a storage diseases. As the blood pressure was well controlled and no other explanations for the patients' symptoms could be found, she was referred to further work-up including coronary angiography and right heart catheterization, without new findings. Overall the patient's condition worsened due to new onset atrial fibrillation and she was thus referred to LV biopsy and genetic testing.

Methods: Case report based on chart review. The patient provided written informed consent.

Results: The LV biopsy showed extensive myocyte hypertrophy with vacuoles and bizarrely increased nuclei. Initial next generation sequencing (NGS) of the typical genes responsible for HCM was unable to identify relevant mutations. As a repeated family history resulted in a clinical suspicion, a whole exom sequencing was added, identifying MT-TL1 associated Mitochondriopathy.

Conclusion: The herein presented case demonstrates the value of multidisciplinary care for HCM patients and underlines the importance of genetic causes beyond commonly included in HCM panel testing.

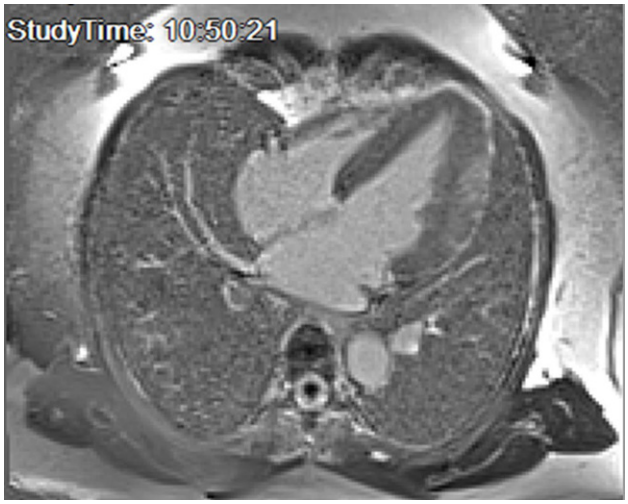

Fig. 1 | 4-6 Cardiac MRI

4-7

Prognostic impact of right atrial function in heart failure with preserved ejection fraction in sinus rhythm vs. atrial fibrillation

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Introduction: We sought to study the prognostic impact of right atrial (RA) size and function in patients with heart failure with preserved ejection fraction (HFpEF) in sinus rhythm (SR) vs. atrial fibrillation (AF).

Methods: Consecutive HFpEF patients were enrolled and indexed RA volumes and emptying fractions (RA-EF) were assessed by cardiac magnetic resonance imaging (CMR). For patients in SR during CMR feature tracking of the RA wall was performed. In addition, all patients underwent right and left heart catheterization, 6 min walk test, and N-terminal prohormone of brain natriuretic peptide evaluation. We prospectively followed patients and used Cox regression models to determine the association of RA size and function with a composite endpoint of heart failure hospitalization and cardiovascular death.

Results: A total of 188 patients (71 % female patients, 70 ± 8 years old) were included of whom 96 (51 %) were in SR. Eighty-five patients reached the combined endpoint during a follow-up of 69 (42–97) months. For patients in SR multivariate cox regression analysis revealed that impaired RA conduit strain rate was significantly associated with worse outcome [hazard ratio 0.990; 95 % confidence interval (0.983–0.998), P = 0.012]. In persistent AF, no RA imaging parameter was related to outcome.

Conclusion: In HFpEF patients in SR, CMR parameters of impaired RA conduit function show the best association with worse cardiovascular outcome. In persistent AF, RA parameters lose their prognostic ability.

Postersitzung 5 – Rhythmologie 1

5-1

Left bundle branch area pacing to optimise cardiac resynchronization therapy (LOT-CRT)– Feasibility, Safety and Efficacy: A single centre experience

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Introduction: Cardiac resynchronization therapy (CRT) is the mainstay in the management of patients with symptomatic chronic heart failure (HF) having left ventricular ejection fraction (LVEF) less than 35 % and a wide QRS complex over 130 ms [1]. However, about one third of the patients are non-responders [2] and do not profit from this therapy. Conduction system pacing, i. e., permanent His bundle pacing (HBP)/left bundle branch area pacing (LBBAP) is proving a promising alternative to biventricular pacing (BiVP) for some HF patients. The combination of physiological pacing with coronary sinus (CS) left ventricular pacing has also been reported as a method to improve responding rate in CRT [3]. The feasibility, safety and efficacy of left bundle branch area pacing (LBBAP) optimized CRT (LOT-CRT) in a single centre is described.

Methods: LBBAP-optimized CRT (LOT-CRT) was performed in nonconsecutive patients with CRT indication over a period of 12 months in a single centre. The addition of the LBBA pacing lead or the CS pacing lead was at the discretion of the implanting physician. The main reason of implanting the additional lead was a suboptimal QRS complex in terms of QRS-width and/or morphology. The baseline and procedural characteristics as well as follow-up data were recorded. All implantations were performed with the use of a three-dimentional electroanatomical system (Ensite NavX).

Results: LOT-CRT was successful in all 7 of the 7 patients attempted (success rate 100 %). The baseline characteristics of the study population were the following: mean age 70.5 ± 9.2 years, male 5/7 (71 %), mean left ventricular ejection fraction 29.1 % ± 11.8 %, left ventricular end-diastolic diameter 60.1 ± 5.7 mm. The QRS morphology was a left bundle branch block in the majority of patients (5/7, 72 %), with one patient having right ventricular pacing (14 %), and another one right bundle branch block (14 %). Three patients had ischemic cardiomyopathy, 2 patients dilated cardiomyopathy, one patient suffered from tachymyopathy due to atrial fibrillation and one from pacing induced cardiomyopathy. The procedure characteristics were the following: mean procedure duration 161.9 ± 42.8 min, mean fluoroscopy time 15.3 ± 9.6 min, mean fluoroscopy dose 1831 ± 1950 µGym^2. No complications occurred in all 7 patients. Mean LBBAP capture threshold at the time of patient discharge was 0.72 ± 0.2 V @ 1.0 ms. LOT-CRT resulted in a significant narrowing of the QRS width from 177 ± 30 ms at baseline to 128 ± 10 ms ($P < 0.05$). At follow-up of 3 months (available in 3 patients), the ejection fraction improved from 24.7–41.7 % in these patients. Pacing parameters remained stable, and an improvement of the New York Heart Association class was achieved in all 3 patients (mean NYHA class 1.6 at follow-up vs 2.6 pre-procedurally).

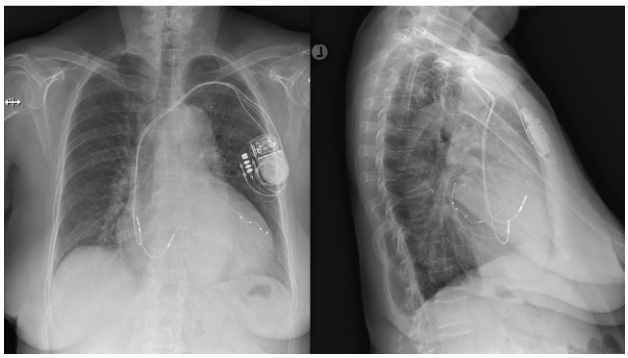

Fig. 1 | 5-1 Chest X-ray of a female patient with a LOT-CRT

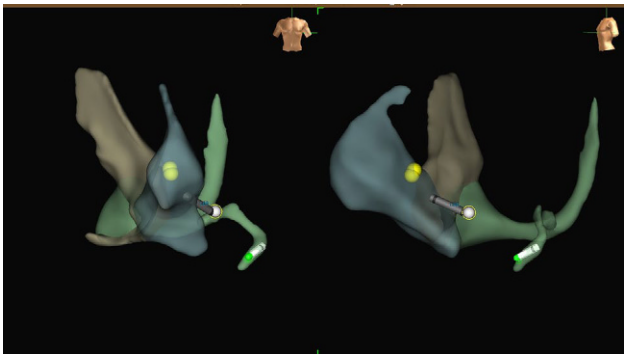

Fig. 2 | 5-1 LOT-CRT implanted with the use of 3D electroanatomical system (Ensite NavX)

Conclusion: Left bundle branch area pacing to optimise cardiac resynchronization therapy (LOT-CRT) is a feasible, safe and effective procedure in providing an optimal electrical resynchronization. It is nevertheless a time-consuming procedure, however radiation can be significantly reduced with the use of a 3D electroanatomical system. It can be an alternative whenever a "simple" conduction pacing or a "simple" biventricular pacing cannot produce the desired electrical resynchronization. However, randomized controlled trials are needed to assess the effectiveness and mostly the efficiency of LOT-CRT in comparison to conventional CRT in heart failure patients.

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5-2

Prevention of early sudden cardiac death after myocardial infarction using the wearable cardioverter defibrillator–Results from a real-life cohort

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Introduction: Patients are at elevated risk of sudden cardiac death (SCD) after acute myocardial infarction (MI). The VEST trial failed to show a significant reduction in arrhythmic mortality in patients prescribed with a wearable converter-defibrillator (WCD), having a lower than expected wearing compliance. We aimed to investigate the incidence of WCD treatments and outcomes of all patients with acute MI and LVEF ≤35 % in a real life and well-compliant cohort in Austria.

Methods: We performed a retrospective analysis of all patients meeting the in- and exclusion criteria of the original VEST trial within the Austrian WCD registry between 2010 and 2021.

Results: 105/896 patients (12 %) with an average age of 64 ± 11 years (12 % female; LVEF 28 ± 6 %) registered in the Austrian WCD registry met the VEST in- and exclusion criteria. 104/105 patients were revascularized and prescribed with a WCD prescription for 69 (1;277) days, the median wearing duration was 23.5 (0;24) hours/day. 4/105 (3.8 %) patients received 9 appropriate WCD shocks, the per patient shock rate was 2 (1;5). No inappropriate shock was delivered. During follow-up, 46/105 patients (44 %) received an ICD after the WCD period, 4/105 (3.8 %) patients died during follow-up. Arrhythmic mortality (1.9 % Austria vs. 1.6 % VEST, p = ns), as well as all-cause mortality (3.8 % vs. 3.1 %, p = ns) in the Austrian cohort were comparable to the VEST cohort.

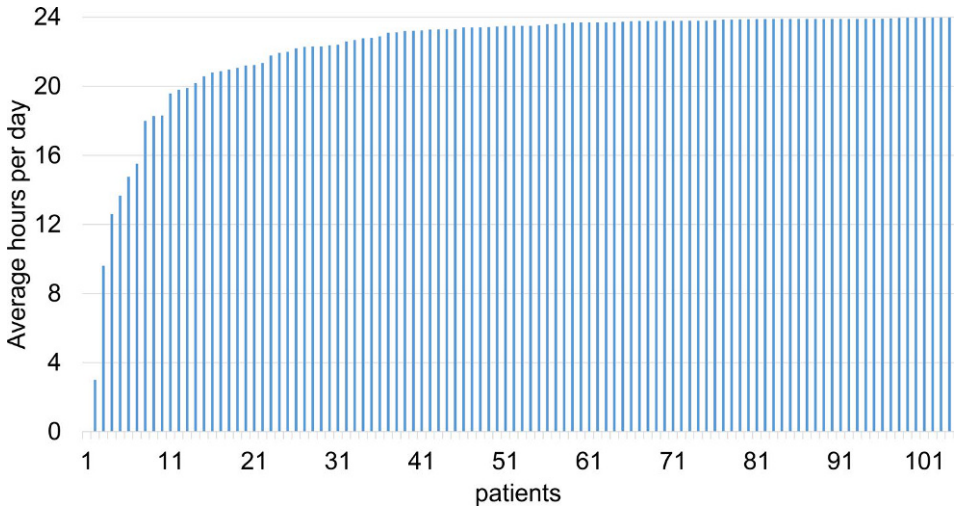

Fig. 1 | 5-2 Average wearing duration/day of Austrian cohort

	Austrian Cohort	VEST trial	P-value
no	105	1524	-
age	64±11 years	61±12.6 years	n.s.
Female	13/105 (12%)	413/1521 (27%)	0.001
LVEF baseline	28±6%	28.2±6.1%	n.s.
Wearing duration	23.5 (0;24) hours/day	18.0 (3.8–22.7) hours/day	
Total mortality	3,8%	3,1%	n.s.
Arrhythmic mortality	1,9%	1,6%	n.s.
Pat with shocks	4/105 (3,8%)	20/1524 (1,3%)	n.s.

Fig. 2 | 5-2 Comparison of Austrian and original VEST device cohort

Conclusion: The WCD is a safe treatment option in a highly selected cohort of patients with a LVEF ≤35 % after acute myocardial infarction. However, despite excellent WCD compliance as opposed to the VEST study, only 3.8 % of patients receive appropriate WCD shocks and the arrhythmic mortality rate was not significantly improved.

5-3

Evaluation der Häufigkeit des erstmaligen Auftretens von Vorhofflimmern nach transfemoralem Aortenklappenersatz mittels kontinuierlichem Device-Monitoring

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Einleitung: Bei älteren Patient:innen mit hochgradiger symptomatischer Aortenklappenstenose ist der transfemorale Aortenklappenersatz (TAVI) eine etablierte Therapieoption. Die Häufigkeit von Vorhofflimmern nimmt ebenso wie die Prävalenz der Aortenklappenstenose mit dem Alter zu und erreicht in der Altersgruppe der über 80-Jährigen eine Prävalenz von knapp 10–15 %. Da Vorhofflimmern mit einem signifikant erhöhten Schlaganfallrisiko und erhöhter Mortalität assoziiert ist, aber bei bis zu einem Drittel aller Patient:innen asymptomatisch verläuft, ist eine rechtzeitige Diagnosestellung von klinischer Relevanz.

Methoden: Im Zuge einer prospektiven klinischen Studie an unserer Abteilung wurde bei Patient:innen, die sich einer TAVI unterzogen mittels kontinuierlichem EKG-Monitoring die Rate an postinterventionellen Arrhythmien in einem Zeitraum von 12 Monaten evaluiert. All jene Patient:innen, bei welchen 48 h nach TAVI keine Indikation zur Schrittmacherimplantation bestand, unterzogen sich einer Loop-Recorder-Implantation. Beide Patientenkollektive wurden nach 1, 3, 6 und 12 Monaten zu einer ambulanten Kontrolle mit 12-Kanal-EKG und Device-Abfrage in unsere Klinik bestellt. Der Fokus der hier beschriebenen Subanalyse lag auf dem erstmaligen Nachweis von Vorhofflimmern.

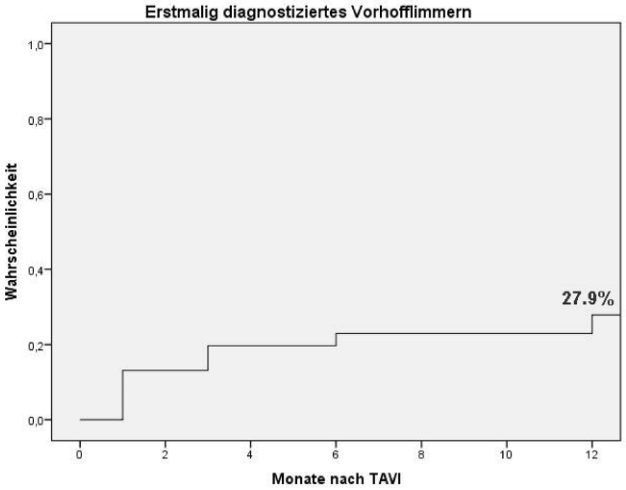

Fig. 1 | 5-3 Kaplan-Meier-Kurve mit Darstellung der Wahrscheinlichkeit des erstmaligen Auftretens von Vorhofflimmern innerhalb von 12 Monaten nach TAVI

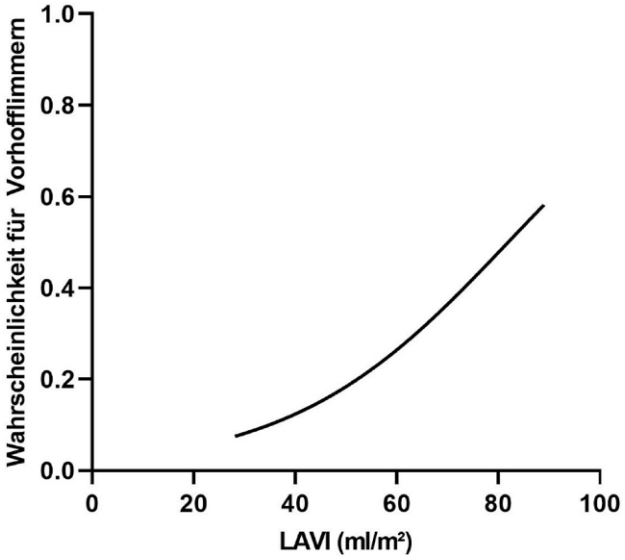

Fig. 2 | 5-3 LAVI als signifikanter Prädiktor für das Auftreten von Vorhofflimmern

Resultate: Insgesamt wurden 61 Patient:Innen (38 Frauen, 23 Männer) mit einem mittleren Alter von 80,4 Jahren in die Subanalyse eingeschlossen (Tab. 1). All jene Patient:innen, bei denen bereits vor der TAVI-Prozedur Vorhofflimmern diagnostiziert worden war, wurden exkludiert. Während 48 Patient:innen (78,7 %) sich einer Loop-Recorder-Implantation unterzogen, bestand bei 13 Patient:innen (21,3 %) nach dem Klappenersatz die Indikation zur Implantation eines Schrittmachersystems. Im Rahmen des einjährigen Follow-Up konnte bei 17 (27,9 %) Patient:innen erstmalig Vorhofflimmern diagnostiziert werden. Dabei lag der Zeitpunkt der Diagnosestellung bei 8 Patient:innen nach 1 Monat, bei 4 weiteren nach 3 Monaten sowie bei 2 nach 6 Monaten und 3 am Ende des Studienzeitraums nach 12 Monaten (Abb. 1). Angesichts des vorliegenden CHADS-VASc-Scores wurde bei sämtlichen Patient:innen eine orale Antikoagulationstherapie etabliert. Der linksatriale Volumenindex (LAVI) als Maß für die Größe des linken Vorhofs, welcher aus den vorliegenden CT-Schnittbildern, die routinemäßig zur Planung der TAVI-Prozedur angefertigt wurden, kalkuliert werden kann, erwies sich als signifikanter Prädiktor ($p = 0{,}048$; OR 1,048 (95 % CI 1,002–1,100), Abb. 2) für das Auftreten von Vorhofflimmern innerhalb von 12 Monaten nach dem Klappenersatz.

Schlussfolgerungen: Innerhalb von 12 Monaten nach TAVI konnte bei 27,9 % aller Patient:innen erstmalig Vorhofflimmern diagnostiziert werden. Der linksatriale Volumenindex (LAVI) erwies sich als signifikanter Prädiktor.

Parameter	
Geschlecht (weiblich; n, %)	38; 62.3
Alter (Jahre; mean ± SD)	80.4 ± 4.3
Body-Mass-Index (kg/m²; mean ± SD)	25.9 ± 4.6
Körperoberfläche (m²; mean ± SD)	1.79 ± 0.19
Euro Score II (median; IQR)	4.1; 3.7
CHADS-VASc (median; IQR)	5; 2
Koronare Herzkrankheit (n, %)	25; 41.0
Zustand nach Schlaganfall (n, %)	2; 3.3
Arterielle Hypertonie (n, %)	42; 68.9
Diabetes mellitus (n, %)	14; 23.0
Chronische Niereninsuffizienz KDIGO III-V (n, %)	7; 11.5
Periphere arterielle Verschlusskrankheit (n, %)	10; 16.4
Chronische Lungenerkrankung (n, %)	7; 11.5
Schrittmacherimplantation (n, %)	13; 21.3
Loop-Recorder-Implantation (n, %)	48; 78.7
PQ vor TAVI (ms; mean ± SD)	178.7 ± 30.5
QRS vor TAVI (ms; mean ± SD)	97.6 ± 21.9
LAVI (ml/m²; mean ± SD)	58.9 ± 13.3

Fig. 3 | 5-3

5-4

Acute and long-term success of left atrial anterior line and mitral isthmus line ablation in patients after mitral valve surgery

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Introduction: Perimitral flutter and atrial fibrillation may occur in patients with prior surgical mitral valve (MV) repair or replacement and can be challenging for percutaneous catheter ablation. This study sought to determine the feasibility, acute success and durability of catheter ablation of atrial fibrillation or atrial tachycardia by way of a mitral isthmus line (MIL) or an anterior line (AL).

Methods: For this retrospective study we analyzed 8594 patients in the period between 2008–2014 who had undergone left atrial CA for atrial fibrillation (AF) or left atrial tachycardia (LAT). A total of 81 patients (49 [60 %] male, mean age 62 ± 11 years) with prior MV replacement ($n = 30$) or repair ($n = 51$) were enrolled; they constituted the "valve group". AF was the presenting arrhythmia in 44 patients (54 %) at the time of the index procedure, which was defined as the first creation of an AL or MIL. In 37 patients (46 %) PMFL was confirmed using entrainment maneuvers and/or a 3D activation mapping. Previous pulmonary vein isolation (PVI) had been performed in 39 patients (48 %). None of the patients had undergone prior creation of a linear lesion. In patients undergoing repeat ablation procedures, durability of the former created lines were checked. If necessary a reablation of the reconnected line was performed or a new line was created. Patients of an control group without prior MV surgery were matched 1:1 with the valve group.

Results: Acute bidirectional block of the MIL was successfully achieved in 24/34 cases and of the AL in 64/72 patients. In the AL control subgroup, acute bidirectional block was achieved in 65/72 patients. Acute blockage in the MIL control subgroup could be achieved in 31/34 patients. Of the 72 valve-group patients who had received an AL, 23 (32 %) underwent a repeat ablation procedure and 8/23 patients (35 %) presented for a third procedure. Of the 72 control-group patients who had initially undergone AL deployment, 31 (43 %) presented for a

	1st Procedure		2nd Procedure		3rd Procedure	
	Lines blocked	Lines not blocked	Lines blocked	Lines not blocked	Lines blocked	Lines not blocked
Valve						
AL	64/72 (89)	8/72 (11)	7/23 (30)	16/23 (70)	2/8 (25)	6/8 (75)
MIL	24/34 (71)	10/34 (29)	2/13 (15)	11/13 (85)	0/4 (0)	4/4 (100)
Control						
AL	65/72 (90)	7/72 (10)	13/31 (42)	18/31 (58)	4/5 (80)	1/5 (20)
MIL	31/34 (91)	3/34 (9)	7/12 (58)	5/12 (42)	2/3 (67)	1/3 (33)

Values are n/N (%)

Abbreviations: AL = anterior line; MIL = mitral isthmus line.

Fig. 1 | 5-4 Long-term outcomes

	Probability of long-term failure	HR [95% CI]	p
AL			
Valve	0.844	1.22 [0.66, 2.26]	0.52
Control	1.030		
MIL			
Valve	2.224	0.27 [0.11, 0.65]	0.004
Control	0.605		
Valve			
AL	0.844	2.64 [1.36, 5.1]	0.004
MIL	2.224		
Control			
AL	1.030	0.59 [0.25, 1.4]	0.22
MIL	0.605		

Abbreviations: AL = anterior line; CI = Confidence Interval; HR = Hazard ratio; MIL = mitral isthmus line.

Fig. 2 | 5-4 Durability

repeat ablation and 5/31 (16 %) underwent a third procedure. Of the 34 valve-group patients who had initially been treated with a MIL, 13 patients (38 %) presented for a repeat ablation and 4 (31 %) of the 13 patients presented for a third procedure. Of the 34 control-group patients with an index MIL, 12 (35 %) presented for a second ablation and 3/12 (25 %) underwent a third procedure (Table 1). The MIL valve subgroup showed the worst results in terms of durability, whereas a similar trend emerged in the control group and the AL valve subgroup (probability of failure in MIL valve subgroup 2.224 vs. MIL control subgroup 0.605 [Hazard Ratio (HR) = 0.27, 95 % confidence interval (CI), 0.11–0.65, $p = 0.004$]; probability of failure in AL valve subgroup 0.844 vs. AL control subgroup 1.03 [HR = 1.22 (95 % CI, 0.66–2.26), $p = 0.523$]). Comparisons of the probabilities of long-term failure of MIL and AL in the two patient groups are given in Table 2.

Conclusion: Percutaneous creation of a MIL and the deployment of an AL is feasible and safe in patients with prior MV replacement or repair. Regarding the acute success rates, the AL shows similar results in both the valve and the control subgroups. In contrast, MIL creation in the valve group was associated with significantly poorer acute success rates than in the control subgroup. The durability of the AL was similar in all patient groups. In contrast, the durability of the MIL was significantly lower in the valve group compared to all other groups. This suggests an aggravating influence of previous MV surgery on the acute success and the durability of the MIL, whereas the creation, acute blockage and durability of the AL appeared to be not affected.

5-5

Feasibility and safety of outpatient catheter ablation with same-day discharge

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Introduction: Percutaneous catheter ablation, especially for atrial fibrillation (AF), is a procedure performed typically in an inpatient setting. The low complication rate and efficacy of catheter ablation in hospitals suggest that it might be feasible to perform it in an outpatient setting and with same-day discharge.

Methods: Consecutive patients with symptomatic cardiac arrhythmias undergoing a percutaneous catheter ablation procedure at the first outpatient clinic in switzerland in a pure outpatient setting were included. Within one year, 122 patients (64 % male; mean age 65 ± 13 years) were enrolled in this study. All patients were admitted to the outpatient clinic in the morning of the ablation procedure, and a transesophageal echo was performed if patients were scheduled for AF ablation. Oral anticoagulation was discontinued on the day of admission. The endpoints of the procedures were–dependent on the type of arrhythmia–termination of the tachycardia, non-inducibility, blockage of the ablation line or isolation of the pulmonary veins. After a 6–8-hour recovery and monitoring period, the patients were discharged on the same day if clinically stable. Oral anticoagulation was restarted 6 h after the procedure. A follow-up visit was scheduled for the next day

Results: A total of 122 ablation procedures were performed, specifically, 2 diagnostic EP studies, and ablation for AF ($n = 69$; 55 [80 %] with cryoballoon), Re-PVI ($n = 16$), SVT ($n = 52$), PVCs ($n = 3$), and VT ($n = 1$). The mean procedure time was 86 ± 52 min. The majority of patients (97/122, 80 %) were anticoagulated with rivaroxaban being the most common NOAC (50 %), followed by edoxaban (24 %), apixaban (23 %), as well as dabigatran and warfarin with 2 % each. Major complications occurred in 7 patients (6 %). In 2 patients (2 %) phrenic nerve palsy was observed. Five patients (4 %) developed pericardial effusion, 3 requiring pericardiocentesis, while one had to undergo surgery due to perforation of the left atrial appendage. Except for the latter patient, all other 121 patients (99 %) were discharged on the same day after a maximum surveillance time of 8 h. No delayed complications have been documented in a 6 month follow up.

Conclusion: Catheter ablation of all arrhythmias on the day of admission is feasible and safe with a low risk of complications. The majority of patients can be discharged on the same day.

5-6

Contrast-induced kidney injury after cryoballoon ablation of atrial fibrillation

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Introduction: Atrial fibrillation (AF) is the most common sustained arrhythmia; catheter ablation provides an efficacious treatment option in symptomatic patients. While radiofrequency-based pulmonary vein isolation (PVI) was considered

	CKD stage 1 (n = 70)	CKD stage 2 (n = 256)	CKD stage 3 (n = 111)	CKD stage 4 (n = 7)
AKI	1 (1.4%)	3 (1.2%)	7 (6.3%)	2 (28.6%)
Creatinine Elevation	21 (30%)	77 (30%)	31 (28%)	2 (29%)
Steady State	49 (70%)	179 (70%)	80 (72%)	5 (71%)
Complications	5 (9%)	31 (13%)	10 (10%)	0 (0%)

Fig. 1 | 5-6 Incidence of acute kidney injury according to CKD stage

the "gold standard" for a long time, the cryoballoon (CB) has emerged as the most common alternative ablation tool. However, this technique is associated with a higher exposure to contrast media, and little is known about postprocedural renal dysfunction and its risk factors. In this study, we assessed the incidence, characteristics, and risk factors of contrast-associated acute kidney injury (AKI) after CB-based catheter ablation in a large patient cohort.

Methods: In this retrospective analysis, patients who underwent cryoablation for symptomatic drug-refractory AF at our clinic between 07/2012 and 11/2019, were included. AKI was defined as a 0.3 mg/dl increase in serum creatinine from baseline within 48 h or an increase in serum creatinine by more than 50 % within 7 days. Chronic kidney disease (CKD) stage was defined via the estimated glomerular filtration rate (eGFR) and the Kidney Disease Improving Global Outcomes (KDIGO) classification. The study population was divided into four subgroups: patients with eGFR >90 mL/min/1.73 m^2 (CKD stage 1), patients with eGFR 60–89.9 mL/min/1.73 m^2 (CKD stage 2), patients with eGFR between 30–59.9 mL/min/1.73 m^2 (CKD stage 3) and eGFR 15–29.9 mL/min/1.73 m^2 (CKD stage 4).

Results: A total of 444 patients (201 female, mean age 66.3 ± 10.6 years, 237 paroxysmal AF) were analyzed. The total volume of contrast medium administered was 125.5 ± 41.5 mL. Serum creatinine level after catheter ablation was measured in all 444 patients. Data at 2–7 days after contrast exposure were available for 331 patients (75 %). An additional 113 patients had their serum creatinine level measured within 10 weeks during the follow-up period. The overall incidence of AKI was 2.9 % (13/444). A comparison of changes in creatinine levels among the CKD groups is shown in Table 1. The prevalence of AKI was greatest at 28.6 % (2/7) in the CKD stage-4 group, followed by CKD stage-3 group with 6.3 % (7/111). The incidence of AKI was lowest at 1.4 % (1/70) and 1.2 % (3/256) in the CKD stage-1 and stage-2 groups, respectively. Despide the initial CDK stage, 30 % of the patients in every group showed an elevation of the serume creatinine during the mean follow up of 10 weeks after the procedure. We only found a weak association between contrast volume and the increase in serum creatinine levels.

Conclusion: Advanced preexisting kidney disease (CKD stage 4) and was identified as an independent predictor of AKI after intravenous contrast exposure. There was no significant dose-ranging relationship between CM volume and contrast nephropathy.

Postersitzung 6 – Basic Science 1

6-1

Insights into the biomechanical implications of endografts (TEVAR) after stress-based testing

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Introduction: Numerous pathologies of the thoracic aorta, such as aneurysms and dissection, can only be treated with thoracic endovascular aortic repair (TEVAR) methods. However, the biomechanical impact on the aorta after TEVAR are largely unknown.

Methods: Human thoracic aortas ($n = 9$) were perfused (6 h) within a mock circulation loop (pre-, post-TEVAR) (E-vita Thoracic, Jotec) under physiological conditions to map compliance mismatch by recording the pressure (paorta) and the proximal convex-concave distensibility in the circumferential direction (dcirc) (Fig. 1a,b). After perfusion and removal of the stents, biaxial tension tests (stress-stretch) were carried out to explore the stiffness profile on stented versus non-stented samples.

Results: Hysteresis loops show a significant reduction in aortic elasticity after TEVAR-explantation, indicating a compliance mismatch and a stiffer behavior of the stented samples compared to the non-stented samples. This strongly suggests an early loss of elastic fibers

Conclusion: The negative influence of the stent-graft on the aortic wall seems to occur in the first few hours after TEVAR. Fig. 1a,b,d show the loss of elasticity between non-stented and stented aortas for all cases. Custom-designed and adaptable stent-grafts may be more beneficial. Conclusion: TEVAR-induced damage to the human thoracic aortas was tested in vitro under physiological conditions. The biomechanical com-

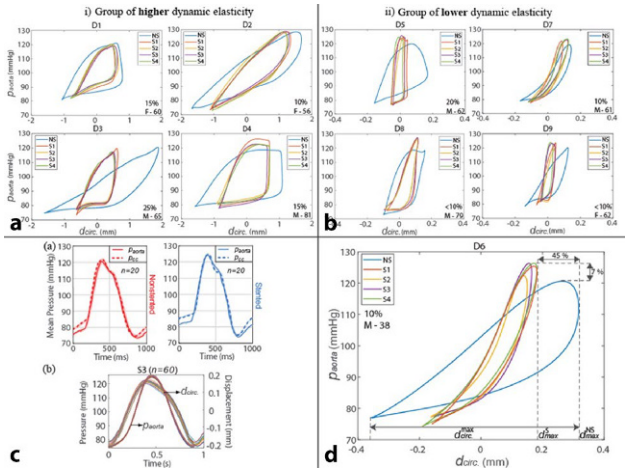

Fig. 1 | 6-1 Hysteresis loops with higher (**a**) and lower elasticity (**b**). The larger loops (*blue*) show aortic compliance in the non-stented period (NS), the other loops show compliance in the stented period (S1–S4). **c**-(a) shows the mean pressure waveforms of the non-stented (*left*) and the stented (*right*) periods ($n=20$). **c**-(b) shows the pressure-displacement measurements of D6 (S3) for 60 cycles

parison of the non-stented and the stented period could provide new insights into the interaction between the stent and the aortic wall.

6-2

Histological fibrosis quantification of pediatric heart samples of operated congenital heart diseases

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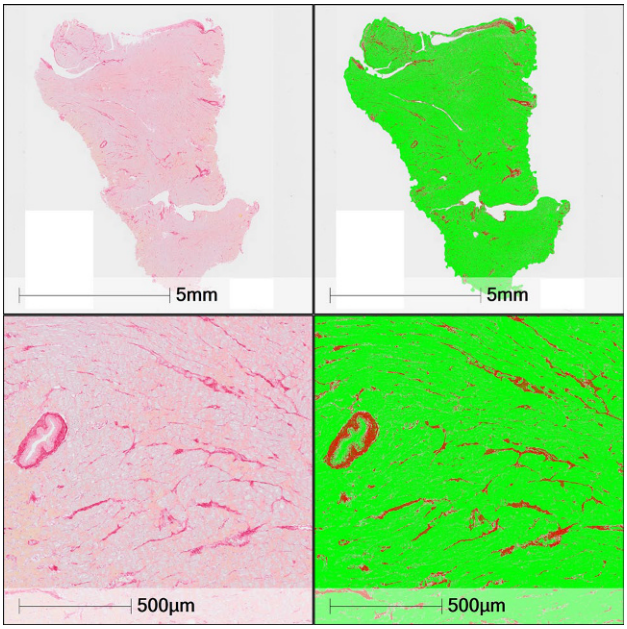

Fig. 1 | 6-2 Fibrosis quantification with HALO

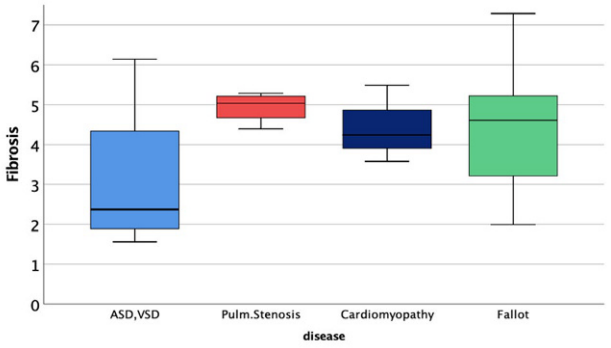

Fig. 2 | 6-2 Mean size of the RV fibrotic area

Introduction: Congenital heart diseases (CHDs) represent the majority of cardiac disorders in pediatric patients. Due to modern cardiovascular medicine and surgery, there is a high chance that most pediatric patients will reach adulthood. Cardiac fibrosis due to congenital anomalies of the heart may lead to left and/or right ventricular remodeling and heart failure or cardiac arrhythmias. Cardiac magnetic resonance imaging is useful to assess myocardial fibrosis in adults, but it has limited accessibility for pediatric patients often instrumented with life-saving MRI-noncompatible equipment. The aim of our study was to quantify myocardial fibrosis of pediatric right ventricular (RV) myocardial samples harvested during cardiac surgery and relate to the type of the CHD.

Methods: RV samples ($n=20$) of pediatric patients were collected during the open-heart surgery (EC: 1565/2019). Clinical data (age, gender, type of CHD) were recorded. The heart samples were fixed in formalin, embedded in paraffin and subsequently Picrosirius Red staining (PSR) was performed. Digitised whole slide images were obtained by using a slide scanner. The quantification of fibrotic tissue in relation to healthy tissue, as well as the measurement of the total section, was performed using the image analysis platform HALO (Indica Labs). For further bioinformatic analyses, the patients were categorized according to their CHD.

Results: The median age of the patients were 4.6 months (1.6–14.8 Interquartile Range, IQR). There were 10 female and 10 male patients. The main diagnose was atrial and/or septal ventricular septal defect ($n=4$), pulmonary stenosis ($n=4$), cardiomyopathy ($n=3$) and Fallot's tetralogy ($n=9$). There was no difference between the male and female patients regarding type of diseases or age at the cardiac surgery. The mean size of the fibrotic area of the RV was 8.5 ± 3.1 % of the entire RV in all patients (Fig. 1). The mean size of the RV fibrotic area was 6.2 ± 4.2 % for ASD/VSD, 9.9 ± 7.8 % for pulmonary stenosis, 8.9 ± 1.9 % for cardiomyopathy and 8.7 ± 3.4 % for Fallot's tetralogy patients, with a trend towards higher fibrosis grade in isolated pulmonary fibrosis (Fig. 2). No correlation was found between age and amount of myocardial fibrosis. No statistical difference

in RV fibrosis area was found between male or female pediatric patients.

Conclusion: This study demonstrates the results of fibrosis quantification of pediatric cardiac tissue samples. Since the clinical outcome of the CHD depends on the structural changes of the diseased left or right ventricles, detecting cardiac fibrosis in the early stage of the disease is of clinical importance. Histological quantification of myocardial fibrosis of cardiac tissue samples ("waste" material of the open heart surgery) is a simple and quick method, which can replace the fibrosis imaging by cardiac magnetic resonance in severely ill pediatric patients.

6-3

Dissecting the progression of cardiac dysfunction in tumor-bearing mice

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Introduction: Cancer patients undergoing heart-related complications result in high incidences of mortality. Nevertheless, it is still not fully understood whether localized tumors affect heart function prior to the onset of cachexia, hence, making the heart more vulnerable for functional abnormalities in later stages of the disease. In addition to analyse heart function, we focus on the expression BCL-2-associated athanogene 3 (BAG3), a co-chaperone protein and Hsp70, which are highly expressed in tumor but decrease in cardiomyocytes (CM) in heart failure (HF).

Methods: Colon-26 adenocarcinoma cells (C26; $n=22$) with/without shIL-6 (C26 shIL-6; $n=22$) were injected subcutaneously into the right flank of 10–11 weeks old BALB/c male mice. Control mice were injected with vehicle (PBS; $n=8$). Cardiac function was assessed by echocardiography and invasive hemodynamic measurements 10 (early) and 20 (late) days after the injection, respectively. In addition, the expression of BAG3 and Hsp70 were determined by Western blot as well as the extend of cardiac fibrosis was determined by Masson-Goldner's trichrome staining.

Results: The tumor size was comparable between the two injected groups. However, only C26 group showed a significant loss of subcutaneous fat and skeletal muscle ($p<0.05$, respectively), suggesting cachexia. Heart weight normalized to tibia length was not changed in the injected groups as compared to controls (day 20). However, left ventricular ejection fraction (LVEF) showed a tendency to decline in the early phase ($p \sim 0.08$) in both injected group and it reached significance at late stage ($p<0.05$). Invasive hemodynamic assessment also confirmed the contractile dysfunction, resulting in a decrease in LV systolic pressure and increase of LV end-diastolic pressure ($p<0.05$, respectively). Importantly, these functional changes in the heart in tumor-bearing mice were associated with a marked reduction in both BAG3 and Hsp70 in the myocardium. Furthermore, there was no sign of cardiac fibrosis in the injected groups.

Conclusion: Our study shows for the first time that tumor rather than cancer cachexia plays a significant maladaptive role in the progression of cardiac dysfunction in a mouse model of C26 injection-induced cachexia. The progression of cardiac contractile dysfunction was associated with a decline in BAG3 and Hsp70 in tumor-bearing mice, suggesting changes of BAG3/Hsp70 signalling may be a critical component as well as target.

6-4

Right ventricular recovery upon myocardial infarction

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Introduction: Despite decades of intensive research, a regenerative therapy of the fibrotic myocardium is not in sight. In contrast to the left ventricle, clinical experience suggests a regenerative capacity of the right ventricle upon myocardial infarction. In this project, we aimed to (a) establish a murine model for right ventricular myocardial infarction and (b) demonstrate a possible regenerative effect of the right ventricle upon infarction.

Methods: Right or left myocardial infarction was induced by ligation of either the right (RCA) or the left anterior descending (LAD) coronary artery in mice. During a 4-week follow up period, right and left ventricular function was evaluated weekly by trans-thoracic-echocardiography. Hearts were harvested after four weeks and histological sections were manufactured. H.E. and Picro-sirius red stainings were performed to assess morphological differences and quantification of scar mass.

Results: Right and left myocardial infarction resulted in a decrease of the right and left ventricular function respectively (TAPSE 1.01–0.68 mm and LV-EF 61.9–34.7 %). In contrast to the left ventricle, right ventricle function recovered almost completely within the 4 week follow up (Delta-TAPSE +35.46 % vs. Delta-LV-EF +5.74 %). Histological sections revealed smaller scar formation within the right ventricle in comparison with the left ventricle (13.18 % vs. 19.77 %). Additionally, increased mass of viable cardiomyocytes within the scar formation was observed in the right ventricle scar (58.35 % vs. 48.82 %).

Conclusion: Our new mouse model for right ventricular myocardial infarction offers the possibility to investigate the regenerative capacity of the right ventricle. In contrast to the left ventricle, a regenerative potential of the right ventricle was observed in initial analyses. Further studies are needed to elucidate (a) differences in the response to ischaemia between the two ventricles and (b) understand the pathomechanisms responsible for the regeneration of the right ventricle.

6-5

Conduction and calcium-handling proteins in left ventricular hypertrophy due to aortic valve stenosis vs. hypertrophic obstructive cardiomyopathy

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Introduction: Marked hypertrophic cardiac alterations often lead to malignant arrhythmias that may result in sudden cardiac death. The acquired disease of aortic valve stenosis (AVS) and the congenital hypertrophic obstructive cardiomyopathy (HOCM) are both pathologies that may be followed by left ventricular hypertrophy (LVH). The concomitant alterations in latter stages promote an arrhythmogenic substrate, which is frequently driven by intracellular calcium overload. Nevertheless, the molecular characterization and the direct comparison of these issues in humans are still rare in the literature.

Methods: To study this issue, we analyzed LV septal specimen of cardiosurgical patients undergoing myectomy and/or aortic valve replacement due to AVS and HOCM by immunofluorescence and western blot. In order to address a proper electrical conduction between the cardiomcoytes, the location of connexin 43 (Cx43) was identified and its expression was quantified by western blot. As calcium plays a fundamental role in the cardiac rhythm, further analyses addressed possible alterations in the expression of cellular, mitochondrial, and sarcoplasmic reticulum calcium handling proteins. Healthy post-mortem septal cardiac specimen served as a control group.

Results: Patient characteristics and echocardiographic parameters are similar in both pathology groups. Cx43 was shown to play a minor role in the present study, not presenting any alteration in its expression or location in LVH. Cardiac calcium ion channel analyses unveiled a significant decrease of RyR2 in both pathologies (AVS: $p=0.0136$, HOCM: $p=0.0041$). On the contrary, the expression pattern of the mitochondrial calcium channel proteins MCU and MICU1 are significantly increased in hypertrophy. Together with the decrease of their regulatory proteins PRMT-1, UCP-2 and UCP-3 in hypertrophy, there is great evidence for an increased activity of the MCU complex in both pathologies.

Conclusion: As expected, there is a higher fibrotic burden in pathologically hypertrophied cardiac tissue. In accordance with previous studies in mouse models, the analyses unveiled that LVH has a marked influence on the mitochondrial calcium handling protein expression of MCU and MICU1, as well as the SR protein RyR2. Consequently, we suggest a possible counterbalance of MCU complex to fulfil the higher energy demand in LVH. Together with the clinical data, we speculate that the hypertrophic alterations in both study cohorts represent an early compensatory stage of disease progression. Further functional analyses of the cellular and mitochondrial calcium handling and their correlation with the arrhythmic burden in both pathologies would give insights into the propensity for calcium mediated arrhythmias. This study was supported by Paracelsus Medical University (R18/02/106-PAA) and by the Austrian Cardiology Society.

6-6

β1-adrenergic receptor signaling during early and late hypertensive cardiac remodeling

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Introduction: Chronic activation of β1-adrenergic receptors (β1ARs) in response to hypertension is consistently linked to maladaptive remodeling in the heart, however, the underlying mechanisms are not well understood. Here, our aim was to determine the subcellular profile and extent of β1AR expression at baseline and upon acute β-adrenergic stimulation in cardiac myocytes during early- and late-stage cardiac remodeling due to systemic hypertension.

Methods: Male Dahl salt-sensitive rats were fed a high-salt diet (HSD; 8 % NaCl) for either five or ten weeks to induce early or late hypertensive cardiac remodeling, respectively. Age-, sex- and weight-matched Dahl salt-sensitive rats on a low-salt diet (LSD; 0.3 % NaCl) served as controls. To test the effect of conventional anti-hypertensive treatment, a subset of HSD-fed animals received daily doses of the angiotensin-converting-enzyme-inhibitor Imidapril (ACE-I; 1 mg/kg/day) starting two weeks after the feeding protocol was switched to HSD. Isolated ventricular myocytes were stimulated either under control conditions or in the presence of β-adrenergic agonist isoprenaline (ISO; 100 nM; 1 h). Confocal imaging of single cardiomyocytes allowed detailed quantification of β1AR in different cellular compartments. Finally, immunoblotting and microarray analyses were applied to quantify β1AR in the left ventricles of the corresponding groups of animals.

Results: In control rats, β1AR was found in a striated pattern throughout the cell typical for T-tubular network and in the perinuclear regions, while its expression significantly dropped upon ISO treatment. During early remodeling, basal β1AR expression was unchanged, but increased on the T-tubules and perinuclear regions upon acute stimulation with ISO. In contrast, late remodeling was marked by reduced β1AR expression at baseline, and significantly blunted increase in response to ISO compared to early time point. Interestingly, daily ACE-I treatment resulted in even more adverse phenotype as compared to untreated HSD-fed rats in early remodeling, but favorable control-like characteristics at late remodeling stage. Immunoblotting and microarrays from left ventricular tissue confirmed the data, where applicable.

Conclusion: Taken together, we showed that early hypertensive remodeling is marked by altered β1AR responsiveness

upon β-adrenergic stimulation, whereas late remodeling also exhibits altered β1AR expression. ACE-I treatment seemed to interfere with early adaptive mechanisms, thereby worsening the phenotype as compared to untreated HSD-fed animals. However, upon prolonged application, it showed a clear protective effect from pathological molecular alterations at late remodeling. Further experiments involving downstream targets of β1AR signaling are required to fully understand the molecular sequence of events leading to early and late alterations in molecular composition of cardiomyocytes in the hypertensive heart.

6-7

Association between insulin and C-reactive protein: Data from athletes and coronary angiography patients

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Introduction: A better understanding of the relationship between insulin and inflammation may have important implications for the treatment and prevention of diabetes. Chronic inflammation and diabetes are associated with coronary artery disease (CAD). That said, many studies found no correlation between insulin and C-reactive protein (CRP), the most common marker of chronic inflammation.

Methods: We hypothesize here that the association of insulin and CRP may differ in different patient groups and may be masked by factors such as age, fitness or diseases, in particular established CAD.

Results: We thus evaluated the correlates of insulin and CRP among healthy professional athletes (Group 1, mean age = 24 years (ranging from 18–35 years), $n = 59$), and patients undergoing coronary angiography (27–88 years, $n = 1574$) who were further divided into younger (<60 years) patients without sign. CAD (Group 2, mean age = 52 years, $n = 258$), younger patients with sign. CAD (Group 3, mean age = 52, $n = 287$) and older (≥60 years) patients without sign. CAD (Group 4, mean age 69, $n = 407$) as well as older patients with sign. CAD (Group 5, mean age 70, $n = 622$). CRP and insulin were positively correlated in athletes (r1 = 0.544, $p < 0.001$) and in patients without sign. CAD (r2 = 0.194, $p = 0.002$ and r4 = 0.134, $p = 0.007$) but not in patients with sign. CAD (r3 = –0.001, $p = 0.981$ and r5 = 0.000, $p = 0.955$). In multivariate models including covariates BMI and age, CRP and insulin remained significantly associated in athletes (T1 = 4.7, $p < 0.001$) and in patients without sign. CAD (T2 + 4 = 3.3, $p = 0.001$), but not in those with sign. CAD (T3 + 5 = 0.0, $p = 0.997$).

Conclusion: We conclude that CRP and insulin correlate in young and fit subjects but also in older subjects who do not have sign. CAD. The presence of CAD may abrogate the association between inflammation and insulin, potentially due to the inflammatory state of atherosclerosis. This appears worthwhile to be considered in future screening and treatment approaches.

6-8

The role of tachycardia and beta-adrenergic stimulation in inducing early cardiac remodelling

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Introduction: Cardiac remodelling encompasses changes at the molecular, cellular and gene expression level following pathologic insult to the heart. Initially, it maintains cardiovascular homeostasis and allows patients to remain asymptomatic, but if untreated, it eventually progresses to symptomatic heart failure. Excessive β-adrenergic stimulation and tachycardia are potent triggers of cardiac remodelling; however, the underlying mechanisms of their cellular effects are not fully understood. Using neonatal rat ventricular cardiac myocytes (NRVCMs), we studied individual and synergistic potency of β-adrenergic stimulation and tachycardia to modulate pathological gene expression profiles, as well as the effectiveness of β-blockers (BB) in preventing these alterations.

Methods: Primary NRVCMs were isolated from 1-day-old neonatal Wistar rats, cultured for 3 days and subsequently stimulated for 3 h at basal (1 Hz) and tachycardia (8 Hz) conditions either in (1) Cell culture medium to determine the sole effect of tachycardia, (2) Cell culture medium supplemented with β-adrenergic agonist isoprenaline (ISO; 10 μM) to investigate the influence of β-adrenergic stimulation and signalling or (3) Cell culture medium supplemented with ISO following 1 h preincubation with propranolol (ISO+BB; 1 μM) to assess the potential of BB in preventing gene reprograming. Screening of relative mRNA levels of hypertrophic marker genes and regulators of ion homeostasis in cardiomyocytes was performed by qPCR and calculated using the 2-ΔΔCt quantification method.

Results: qPCR screening of the known hypertrophic marker genes revealed that tachycardia caused significant transcriptional upregulation of regulator of calcineurin 1 (RCAN1) and interleukin-6 receptor (IL6R). Treatment with ISO additionally upregulated RCAN1, while preincubation with BB resulted in a return towards baseline expression of both genes, completely blocking the effects of tachycardia alone or when combined with ISO stimulation. Interestingly, two potassium channel genes, KCNH2 and KCNJ2, responsible for expression of hERG and Kir2.1 channels, respectively, were unchanged with tachycardia alone but significantly downregulated upon additional stimulation with ISO. Preincubation with BB could–at least partially–reverse the effect.

Conclusion: In conclusion, we could show that apart from the well-documented effect of excessive β-adrenergic stimulation on hypertrophic signalling in cardiomyocytes, it also has a direct, non-tachypacing mediated effect on the expression levels of hERG and Kir2.1 potassium channels, which may be causally involved in inducing early cardiac remodelling. Thus, a previously unidentified benefit of BB therapy may be restoring potassium homeostasis contributing to the prevention of adverse cardiac remodelling and its progression to heart failure.

Postersitzung 7 – Chirurgie 2

7-1

Innate reverse remodeling reveals novel treatment option for heart failure

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Objective: Heart failure represents a severe global socio-economic health burden. Contractile cardiomyocytes are replaced by dysfunctional scar tissue. Subsequent remodeling of the myocardium results in change of ventricular geometry and impairment of cardiac function. Current treatment strategies provide symptomatic relieve and at best stop of disease progression. However, there are no treatment options available to regenerate failing myocardium via reverse remodeling. The right (RV) and the left ventricle (LV) differ markedly in their anatomy, function and capability of reverse remodeling. The RV is able to reverse remodel due to a preserved anti-fibrotic mechanism necessary for physiological postnatal adaptation.

We aimed to

(a) identify the conserved mechanisms of innate reverse remodeling of the RV and thus

(b) reveal therapeutic strategies for reverse remodeling of the LV.

Methods: LV and RV heart failure were induced using absorbable sutures in a murine transaortic constriction (TAC) or pulmonary artery banding (PAB) procedure. Sutures were absorbed after 2 weeks, mimicking afterload relieve. RV and LV function and mass were evaluated weekly via transthoracic echocardiography. Cardiomyocyte size, myocardial thickness and myocardial fibrosis were analyzed in histological sections. 1 week after afterload relieve, RNA sequencing was performed to determine genes involved in the reverse remodeling of the RV. Identified genes were analyzed via siRNA knock down in functional cell culture assays. Overexpression and knockdown of identified genes was performed in vivo. Adaption of the RV postpartum is analyzed via µCT, histological sections and qPCR.

Results: In a murine model of reversible heart failure, we observed reverse remodeling of the RV without fibrosis in contrary to LV. RNA sequencing of the regenerating RV revealed the undescribed gene KIAA0408 as possible underlying cause. In vitro an anti-fibrotic effect of KIAA0408 via the JNK/ELK-1/SRF axis was fund. In contrast to the LV, RV myocardial mass decreased from day 1 to day 3 postpartum upon afterload relief. In line, cardiomyocyte size decreased in the RV on day 3 postpartum. No signs of fibrosis were observed in the same time period. In the adaption of the heart postpartum increased levels of KIAA0408 could be observed in the physiological reverse remodeling RV. Therapeutic application of KIAA0408 reduced fibrosis and heart failure.

Conclusion: Our data suggest a conserved postnatal mechanism behind the regenerative capacity of the RV. We reveal the undescribed gene KIAA 0408 as potential anti-fibrotic agent to treat heart failure.

7-2

Genetic testing in type A aortic dissection in clinical practice

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Objective: The impact of heritable thoracic aortic disease has gained great importance duento the technique of next generations sequencing. Aim of this study is to present data on genetic testing during long-term follow up in patients with type A aortic dissection (AADA).

Methods: 445 patients have undergone surgery for AADA between 2020–2021. During outpatient visits genetic testing was offered to patients with positive family history, phenotypical features or young age (<50) at timepoint of dissection since 2019. Massive parallel sequencing of 14 genes with Nextera Rapid Capture (TruSightTMOne, NextSeq, Illumina) was performed in 35 index patients.

Results: Out of 35 index patients, genetic testing revealed no mutation in 9 patients (25,7 %). Three patients were diagnosed Turner syndrome. Marfan syndrome was confirmed in 9 patients (25,7 %), FBN1 mutation of unknown significance was found in two more patients. Syndromic aortic disease was further detected in three patients (8.6 %)—one patient with Ehlers-Danlos syndrome (COL3A1) and Loeys-Dietz syndrome (SMAD3). Familial aortic disease was present in 9 more patients (25,7 %), with MYH11 mutations being the predominant mutation (20 %). 12 additional affected family members could be identified, 3 underwent prophylactic surgery.

Conclusion: Genetic evaluation and early detection of HTAD helps to prevent fatal aortic events and is important to guide clinical management in a patient-tailored way and to provide stronger recommendations for surgical repair.

7-3

Impact of concomitant replacement of the ascending aorta in patients undergoing aortic valve replacement on operative morbidity and mortality

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Objective: The aim of this study was to evaluate the impact of concomitant ascending aortic replacement on operative morbidity and mortality in patients undergoing aortic valve replacement (AVR).

Methods: We retrospectively analysed our institutional database for all patients undergoing elective isolated AVR and AVR with concomitant replacement of the ascending aorta between January 2009 and May 2020. Patients undergoing surgery for infective endocarditis or requiring hypothermic circulatory arrest were excluded. A 3:1 propensity matching was performed for 688 patients to compare isolated AVR (120 patients) with AVR + ascending aortic replacement (40 patients).

Results: There were significant differences in median cardiopulmonary bypass (CPB) time [92.5 (75–114) vs 118.5 (104–131) min; P < 0.001], median aortic cross-clamp time [65.0 (51.5–78.5) vs 84.5 (77–94) min; P < 0.001] and median intensive care unit stay [1 (1–3) vs 2 (1–6) days; P < 0.01]. There was no significant difference in the use of intraoperative and postoperative blood products, re-exploration for bleeding, postoperative atrial fibrillation, acute renal failure, incidence of stroke, perioperative myocardial infarction and 30-day mortality.

Conclusion: Concomitant replacement of the ascending aorta significantly prolongs CPB and aortic clamp times but does not increase operative morbidity and mortality. Therefore, replacement of a dilated ascending aorta appears to be the most durable and safest treatment option in patients undergoing AVR with an aneurysmatic ascending aorta.

7-4

Mid- term results in congenital aortic disease after valve sparing root replacement

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Objective: Valve-sparing aortic root replacement is recommended for patients with aortic root dilatation and preserved aortic valve cusp morphology. The aim of our study was to analyze the durability of valve sparing root replacement in patients with congenital connective tissue disease.

Methods: Outcomes were evaluated for 101 patients who underwent valve sparing root replacement within the last 10 years at our center. Follow—up was obtained via outpatient clinic and was 95 % complete.

Results: Fifty-three patients (52 %) suffered from underlying connective tissue disease. Mean age at surgery was 44y (36y congenital vs. 50 years non-congenital; $p = 0.01$). Seventy-six percent of patients were male (66 % congenital vs. 88 % non-congenital; $p = <0.01$). Indications for surgery were elective root aneurysm repair + aortic regurgitation (AR) in 74 % vs. type A aortic dissection in 26 %. %. More congenital patients underwent elective surgery (87 % vs. 40 %; $p = <0.01$). Operative times were comparable and in- hospital mortality was 0 %. Congenital patients were more prone to revision due to bleeding ($p = 0.04$) and had a shorter ICU stay (1 vs. 2 days; $p = 0.02$). Postoperative echocardiography showed comparable results between groups (none to mild AR in 91 % of congenital patients and 94 % of none- congenital patients. There was no significant difference in rate of postoperative complications. Death during follow-up occurred in 3 patients (2 congenital vs. 1 none-congenital; $p = 0.62$). Redo- surgery was necessary in 10 patients (5 congenital vs. 5 non-congenital; $p = 0.95$). There was no significant difference in rate of pacemaker- implants, strokes, transitory ischaemic attacks or graft infections. Seventy-four percent of congenital patients and 85 % of non-congenital patients continued to have none to mild AR during follow up.

Conclusion: Valve sparing root replacement is feasible in congenital patients.

7-5

Anatomic repair for congenitally corrected transposition of the great arteries – A single-center experience

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Objective: Anatomic repair for congenitally corrected transposition of the great arteries (ccTGA) seems to improve long-term survival. This single-center retrospective study was conducted to evaluate the outcomes in patients undergoing anatomic repair for ccTGA at our institution.

Methods: Between April 2011 and September 2021 a total of 18 consecutive patients received anatomic repair for ccTGA. Median age at repair was 1.9 (range, 0.4–18.0) years, 33.3 % were female. Double-switch (DS) procedure was performed in 11 (61.1 %) patients, Mustard-Rastelli (MR) repair in 6 (33.3 %) and the hemi-Mustard/bidirectional Glenn with Rastelli operation in 1 (5.6 %) patient. Ten (55.6 %) patients underwent previous pulmonary artery banding (PAB) for retraining of the morphological left ventricle with a median PAB duration of 1.2 (IQR 1.0–2.5) years. Follow-up included survival status and cardiovascular (CV) reinterventions.

Results: Median follow-up time was 2.8 (IQR 0.6–5.5) years. There was only one (of 11; 9.1 %) early death in the DS group and 0 (of 7) in the MR group. No late deaths were documented. The Kaplan-Meier estimate for freedom from any CV reintervention at 2 years was 47 %. Ten (55.6 %) patients had at least one CV reintervention. Three (16.7 %) pacemaker insertions were required perioperatively. In total 5 (27.8 %) stent implan-

tations of the systemic venous pathway were performed which affected only the early period of all procedures.

Conclusion: Anatomic repair for ccTGA constitutes a surgical option with high survival probability. The CV reintervention rate as observed within this report is comparable to previous studies. However, for each repair performed serially, improved operative quality was demonstrated with likely superior patient outcomes regarding the Mustard procedure.

7-6

Retrospective Comparison of the hemodynamic parameters of Carpentier-Edwards PERIMOUNT Magna Ease and Medtronic Avalus Aortic Valve Prosthesis

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Objective: Due to an increased lifespan and improving diagnostic procedures, an increase in the incidence of valvular diseases, with aortic stenosis being the majority, can be observed. Facing the menace of interventional TAVR procedures, cardiac surgeons need to identify the most suitable prosthesis in order to deliver superior results after SAVR. The PERIMOUNT Magna Ease bioprosthesis is the world's most frequently used aortic valve prosthesis. The aim of this work is to re-evaluate the preferred bioprosthesis by comparing it to Medtronic's Avalus valve model.

Methods: We retrospectively compared the data of 80 patients who underwent SAVR with an Avalus or Magna Ease bioprosthesis between 2018 and 2021 at our center. The statistics was conducted in SPSS. The main target of the study was the change in mean and maximum transvalvular pressure gradient across the aortic valve.

Results: We found a significant difference in the change of the pressure gradient. The mean ΔP_{max} in the Avalus group is 58.26 (SD±23.7) mmHg, while it is 42.74 (SD±22.3) mmHg in the Magna Ease group ($p < 0.05$). The mean ΔP_{max} in the Avalus group is 36.73 (SD±16.3) mmHg, while it is 29.54 (SD±15.12) in the Magna Ease group ($p < 0.05$).

Conclusion: Based on the results of the research question, both prostheses represent a good choice for aortic valve replacement, slightly favoring the Avalus bioprosthesis. The results of this study can be a potential basis for decision-making in the selection process of a suitable valve.

7-7

Annulus rupture after TAVI with a balloon expandable valve – always a hopeless complication?

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Objective: Rupture of the aortic annulus during TAVI procedure is a rare but fatal complication, if not emergently operated. Due to age and comorbidities of the TAVI patients, the benefit of emergent "rescue" surgery is unclear.

Methods: Since 2012 we identified 6 (0.8 %) patients out of 720 with an acute annulus rupture during TAVI procedure. Median age was 84.5 years (75–88), median log Euroscore: 18.1 (9.5–32) and median STS score: 4.7 (2.4-6.0). A retrospective analysis for intraoperative course, outcome and 30-day mortality was performed.

Results: In all patients a balloon expendable valve was implanted. One patient got symptomatic five hours after TAVI procedure in the ICU and deceased after prolonged resuscitation. In the other five patients haemodynamic instability was identified in the operation room and emergency surgery was performed immediately. One patient recovered well after pericardial drainage—the TAVI prosthesis might have sealed the rupture. Four patients were treated with conventional aortic valve replacement and repair of the aortic annulus with or without pericardial patch. Two of these patients recovered well. One patient suffered from severe cerebral injury and deceased 3 weeks after. In one patient annulus destruction was too severe and he died in the operating room

Conclusion: Annulus rupture after TAVI is a rare condition. Acute and perioperative mortality is profoundly high but emergency surgery rescued the majority of the patients in our case series. This confirms the additional safety benefit of available cardiac surgery when doing TAVI procedure.

7-8

Challenging transcatheter, transapical mitral valve replacement using the Tendyne© device – A case series of nine complex patients

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Abstract: Transcatheter, transapical mitral valve replacement (TMVR) using the Tendyne© device (Abbott Laboratories, Abbott Park, Illinois, USA) is a novel technique to treat severe mitral regurgitation (MR), offering older patients with multiple comorbidities a lower risk procedure to open heart surgery. Surgical mitral valve repair remains the gold standard

in treating severe MR. However, as the population grows older, new minimally invasive techniques have emerged, specifically designed in treating frail patients without the use of a sternotomy or cardiopulmonary bypass. Yet these procedures are limited to straightforward cases, leaving a small group of patients without an alternative. The Tendyne© device recently emerged as a new alternative. We present a case series of nine challenging cases who were not eligible for any of the standard procedures like annuloplasty or edge-to-edge repair. These include four patients following aortocoronary bypass surgery—one of which has an aortic valve replacement, two patients with not only severe MR but also severe aortic stenosis, one patient following mitral valve annuloplasty, a Jehova's witness and one patient originally not eligible for a Tendyne© procedure due to risk of left ventricular outflow tract obstruction. He then received a modified LAMPOON procedure (Laceration of the Anterior Mitral leaflet to Prevent Outflow ObstructioN) using an apical access in combination with a Tendyne© implantation.

With very promising results, we propose the expansion of the Tendyne© device indication to more challenging cases.

Postersitzung 8 – Interventionelle Kardiologie 1

8-1

Impact of route of access and stenosis subtype on outcome after transcatheter aortic valve implantation

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Introduction: Previous analyses have reported outcomes of LFLG AS patients undergoing TAVR without stratifying by route of access. Differences in mortality between access routes have been established for HG patients and hypothesized to be even more pronounced in LFLG AS patients. This study aimed to compare outcomes of patients with low-flow, low-gradient (LFLG) or high gradient (HG) aortic stenosis (AS) after transfemoral (TF) or transapical (TA) transcatheter aortic valve implantation (TAVI).

Methods: 910 patients, who underwent either TF or TA TAVI with median follow-up of 2.22 (IQR:1.22–4.03) years were included in this multicentre cohort study. 146 patients (16.04 %) suffered from LFLG AS. Patients with HG and LFLG AS were stratified according to route of access and compared statistically.

Results: Operative mortality was significantly increased for patients who underwent TA access (OR: 2.91 [1.54–5.48], $p=0.001$) and patients with LFLG AS (OR: 2.27 [1.13–4.56], $p=0.02$), which could be corroborated in a propensity-score matched subanalysis. The increase in risk of operative mortality was additive (OR for TA LFLG: 5.45 [2.35–12.62], $p<0.001$). LFLG patients who underwent TA access had significantly higher operative mortality rates (17.78 %) than TF LFLG (3.96 %, $p=0.016$) and TA HG patients (6.36 %, $p=0.024$).

Conclusion: Operative mortality of HG and LFLG patients was comparable after TF access. HG patients had two-fold higher operative mortality after TA, compared to TF, access while LFLG patients had five-fold increased operative mortality rates. TA TAVI appears suboptimal for patients with LFLG AS. Alternatives, if TF is not possible, need to be assessed in prospective studies.

8-2

Monitoring of mitral and tricuspid valve interventions with CardioMEMs: insights beyond imaging

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Introduction: Introduction: Mitral and tricuspid regurgitation are common and associated with significant morbidity and mortality. Following recent guideline recommendations, transcatheter interventions are increasingly performed. Hence, pre-interventional risk-benefit assessment and evaluation of interventional success, particularly for tricuspid interventions, remain a major goal. Continuous monitoring of cardiac output (CO) and pulmonary artery pressures (PAP) with CardioMEMs may indicate interventional success beyond imaging and conventional biomarkers. Aim: To assess changes of PAP and CO after transcatheter mitral (TMVR) and tricuspid (TTVR) repair and bicaval valve implantation (bi-CAVI), using an implanted PA sensor, and correlate PAP and CO with imaging data.

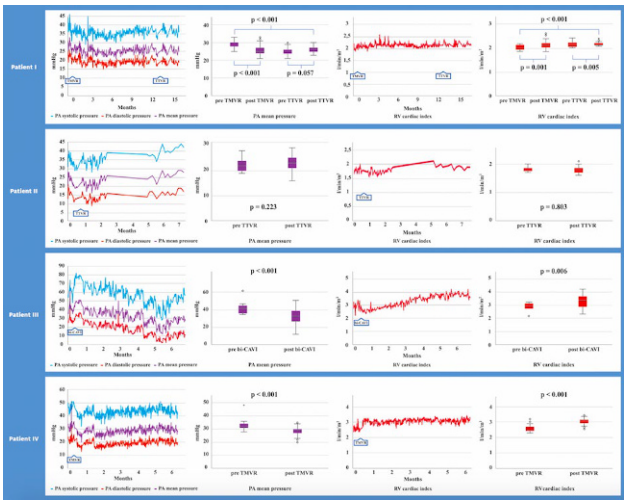

Fig. 1 | 8-2

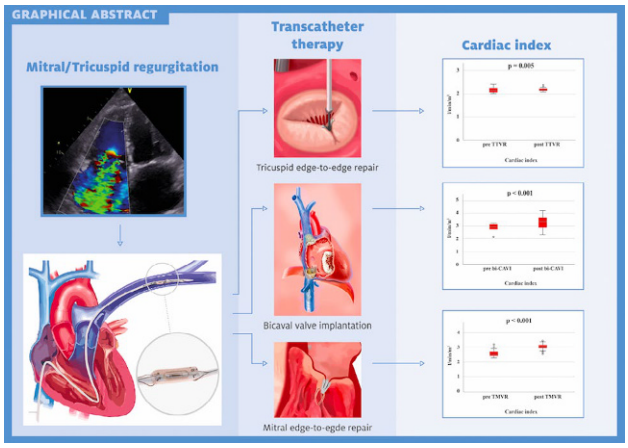

Fig. 2 | 8-2

Methods: Four patients were included and monitored prior to intervention and for 3–12 months thereafter. One patient received isolated TMVR, one bi-CAVI, one both TMVR and TTVR, and one underwent isolated TTVR.

Results: In both patients with TMVR and in the patient with bi-CAVI mean PAP decreased (TMVR 1, 29.5 ± 2.2 to 25.3 ± 2.1 mm Hg; TMVR 2, 32.7 ± 3.4 to 28.6 ± 2.5 mm Hg; bi-CAVI, 42.6 ± 7.4 to 32.9 ± 8.4 mm Hg, all $p < 0.001$) and CO increased significantly (TMVR 1, 3.22 ± 0.16 to 3.40 ± 0.15L/min, $p < 0.001$; TMVR 2, 4.42 ± 0.32 to 5.28 ± 0.24L/min, $p < 0.001$; bi-CAVI 5.83 ± 0.58 to 6.62 ± 0.94L/min, $p = 0.006$) after the procedure while echocardiography and NT-proBNP serum levels were difficult to interpret, unreliable, or both.

Conclusion: Invasive monitoring using CardioMEMs provides important information beyond conventional imaging and changes in biomarker serum levels after mitral and tricuspid valve interventions. Such data pave the way for a deeper understanding of the prerequisites for optimal patient selection for catheter-based interventions, especially on the tricuspid valve.

8-3

Baseline NT-proBNP levels predict 1-year mortality in patients undergoing contemporary percutaneous coronary intervention of chronic total occlusions–a prospective observational study

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Introduction: Percutaneous coronary intervention (PCI) of chronic total occlusions (CTO) is an advanced procedure that provides long term clinical benefits. However, the impact of successful CTO revascularization on survival remains unclear and accurate risk stratification challenging. Therefore, we evaluated the predictive value of N-terminal pro-brain natriuretic peptide (NT-proBNP) on mortality in CTO patients undergoing CTO PCI.

Methods: In this prospective observational study, patients undergoing CTO PCI were consecutively enrolled at a university-affiliated tertiary care center over a three-year period (2018–2020). Technical success was defined as successful restoration of angiographically assessed TIMI-3 flow after PCI attempt. Statistical correlation analyses for baseline parameters and primary endpoint were calculated using cox regression. 1-year follow-up was presented using Kaplan-Maier plot.

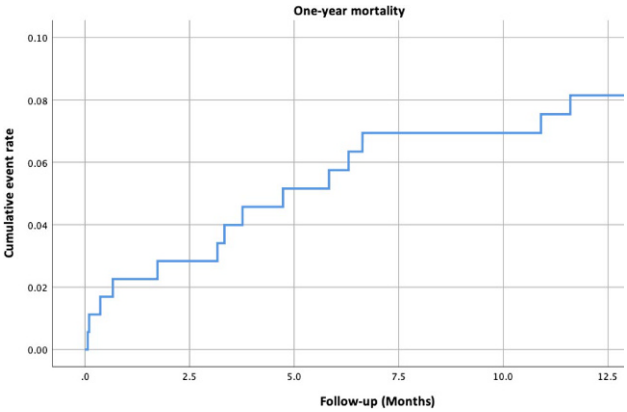

Fig. 1 | 8-3 Kaplan-Meier plot

Variable	Total n=179	Successful PCI n=150 (83.8)	Failed PCI n=29 (16.2)	p-Value
Female	34 (19)	27 (18)	7(24.1)	.440
Age	68.51+11.26	67.99+11.43	71.21+10.12	.160
Diabetes	68 (38)	55 (36.7)	13 (44.8)	.407
COPD	27 (15.1)	18 (12)	9(31)	**.019**
Prior CABG	28 (15.6)	21 (14)	7(24.1)	.169
Heart failure	33 (18.4)	26 (17.3)	7(24.1)	.387
LVEF (%)	48.1±9.82	48.86±9.53	44.19±10.53	**.032**
J-CTO score	1.62±1.21	1.50±1.15	2.24±1.32	**.002**
NT-proBNP (pg/ml)	1417±2382	1321±2156	1967±3423	.241
LDL (mg/dl)	68.6±36.2	69.3±34.2	65.6±45.2	.645

Fig. 2 | 8-3 Baseline patient characteristics

Results: We included 179 patients undergoing CTO PCI. The median follow-up time was 12 months and 15 patients (8.4 %) died. The study cohort is composed of dominantly men (165; 81 %) and has a mean age of 68.5 years. 38 % of patients had diabetes, 15.6 % had prior coronary artery bypass grafting surgery (CABG). 150 patients (83.8 %) underwent successful CTO PCI. The mean J-CTO score was 1.62 ± 1.21: significantly higher in failed CTO PCI (1.50 ± 1.15 vs 2.24 ± 1.32; $p = .002$). Failed CTO PCI patients also had significantly lower left ventricular ejection fraction (LVEF) (48.9 % vs 44.2 %; $p = .032$). Median NT-proBNP was 482.5 pg/ml (IQR 197–1360 pg/ml). Of 15 patients (8.4 %) who died within 1-year after PCI attempt, only one had failed CTO PCI. We found a strong positive correlation between baseline NT-pro-BNP levels and 1-year mortality of patients undergoing CTO PCI ($p = .005$) LVEF, on the other hand, was not correlated to 1-year mortality.

Conclusion: Higher NT-proBNP levels at the baseline associate with higher 1-year mortality in patients undergoing contemporary CTO PCI. Our findings suggest the need for further research on the influence of heart failure in prognosis of patients after CTO PCI.

8-4

Lesion complexity eminently impacts success rates in modern percutaneous coronary intervention of chronic total occlusions–a prospective observational study

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Introduction: Percutaneous coronary intervention (PCI) of chronic total occlusions (CTO) remains a challenging procedure with relatively low success rates. J-CTO and PROGRESS-CTO complexity scores have predicted technical success in several past CTO studies. The aim of this study is to analyse if lesion complexity, revascularization strategy or duration affect technical success of contemporary CTO PCI in an experienced centre.

Methods: Over 3 years (2018–2021), we prospectively enrolled 271 consecutive patients indicated for CTO PCI at our univeristy-affiliated tertiary care centre. All patients underwent antegrade or retrograde revascularization attempts. Complexity of the CTO was evaluated by J-CTO and PROGRESS-CTO score beforehand. Patients were stratified into those undergoing successful CTO PCI, and those with failed intervention attempts. Successful PCI was defined as restoration of TIMI-3 flow after the planned CTO PCI. To compare the outcomes we statistically tested baseline parameters, complexity scores, revascularization strategy and procedure time. Potential correlations were investigated using logistic regression model.

Results: 222 (81.9 %) of the patients were men, 49 (18.1 %) women with an average age of 67.6 ±11.2 years. 219 (80.8 %) patients had a successful CTO PCI, in 52 (19.2 %) patients the attempt was not successful. 60 patients (22.1 %) underwent retrograde CTO PCI attempts. The two groups were comparable in age, sex, hypertension, or dyslipidemia (Table 1). Patients with prior myocardial infarction had a lower CTO PCI success rate (61.5 % vs. 44.3 %; $p=0.025$). Interestingly, final revascularization strategies (antegrade vs. retrograde) did not show significant differences on the success rate of CTO PCI ($p=0.581$). The procedure time was similar in both groups with an average of 189 ±67.5 min in successful PCI and 172 ±61.2 min in failed PCI ($p=0.098$). Higher J-CTO and PROGRESS-CTO complexity scores–and not revascularization strategy or procedure time–were strongly associated with lower success rates, with OR = 0.590; $p<0.001$ and OR = 0.381; $p<0.001$ respectively.

Conclusion: Technical success rate of contemporary CTO PCI in an experienced setting is still significantly influenced by lesion complexity but not prolonged revascularization efforts. Retrograde and antegrade final revascularization strategies deliver comparable success rates in modern CTO PCI, suggesting a plateau of technical advancements.

	Overall (n=271)	Success (n=219)	No success (n=52)	P-Value
Age	67,6 ± 11,2	67,3± 11,1	68,9± 11,6	0,353
Female	49 (18,1%)	37 (16,9%)	12 (23,1%)	0,298
Hypertension	197 (72,7%)	161 (73,5%)	36 (69,2%)	0,533
Dyslipidaemia	200 (73,8%)	162 (74,0%)	38 (73,1%)	0,895
Diabetes	99 (36,5%)	80 (36,5%)	19 (36,5%)	0,999
Prior MI	129 (47,6%)	97 (44,3%)	32 (61,5%)	**0,025**
Retrograde	60 (22,1%)	47 (21,5%)	13 (25,0%)	0,581
J-CTO	1,75 ± 1,25	1,59 ± 1,15	2,40 ± 1,40	**<0,001**
PROGRESS-CTO	0,77 ± 0,80	0,64 ± 0,72	1,59 ± 1,15	**<0,001**
Procedure time	185,75 ± 66,77	189 ± 67,5	172 ± 61,2	0,098

Fig. 1 | 8-4 Baseline patient profile

	OR	CI	P-Value
J-CTO	0,590	0,458-0,761	**<0,001**
PROGRESS-CTO	0,381	0,257-0,564	**<0,001**
Procedure time	1,004	0,999-1,009	0,100
Retrograde	0,820	0,405-1,660	0,581

Fig. 2 | 8-4 Logistic regression analysis

8-5

TEE-guided versus TEE-controlled PFO closure: Single Center Registry

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Introduction: Percutaneous closure of patent foramen ovale (PFO) is conventionally performed under continuous transesophageal echocardiographic (TEE) guidance. Whilst this is considered to increase safety and accuracy, it can also have an impact on procedural consequences, such as longer duration, patient sedation or anesthesia, more personal and patient discomfort. We aimed to evaluate whether a simplified procedural approach, including pure fluoroscopy-guidance and only final TEE control, as well as an aimed 'next-day-discharge' is comparable with the conventional TEE-guided procedure in terms of periprocedural and long-term outcomes.

Methods: All patients who underwent a PFO closure in our department between 2010 and 2021 were retrospectively included. Prior to June 2019 cases were performed with continuous TEE guidance (TEE-guided group). Since June 2019 pure fluoroscopy-guided PFO closures have been performed with TEE insertion and control just prior to device release (TEE-controlled group). In total 265 patients were included in the analysis: 197 in the TEE-guided group and 68 in the TEE-controlled group. We analyzed procedural aspects, as well as long term clinical and echocardiographic outcomes.

Results: Anatomy was similar in both groups regarding channel length (11 ±4 mm vs 10 ±4 mm, respectively; $p=0.65$) and separation (4 ±2 mm vs 4 ±1 mm; $p=0.36$). Cross-over from TEE-control to TEE-guidance group occurred in 9 % due to difficulties with PFO crossing. In 3 cases (4 %) device recapture was needed due to inappropriate position at TEE-control. TEE-controlled procedures took markedly less time (29 ±9 vs 48 ±20 mins, respectively; $p<0.01$; Fig. 1) and performed with smaller devices (left disk diameter 18 ±2 mm vs 26 ±3 mm; $p<0.01$). There was no difference in procedural complications, such as access site bleeding (1.5 % vs 5.6 %, respectively; $p=0.30$) or periprocedural TIA/Stroke (0 % vs 1.5 %, respectively; $p=0.58$). Hospital stay was markedly shorter with the simplified approach (3 ±1 vs 4 ±1 mins, respectively; $p<0.01$) with more same- or next-day discharges (30.3 % vs 9.6 %, respectively; $p<0.01$). At 6 ±3 months echocardiographic follow-up a residual leakage was described in 11 % of the TEE-guided cases and 2 % of the TEE-controlled cases ($p=0.02$). Median follow-up was longer for TEE-guided patients (33 [7;63] vs 6 [0;7] months, respectively; p

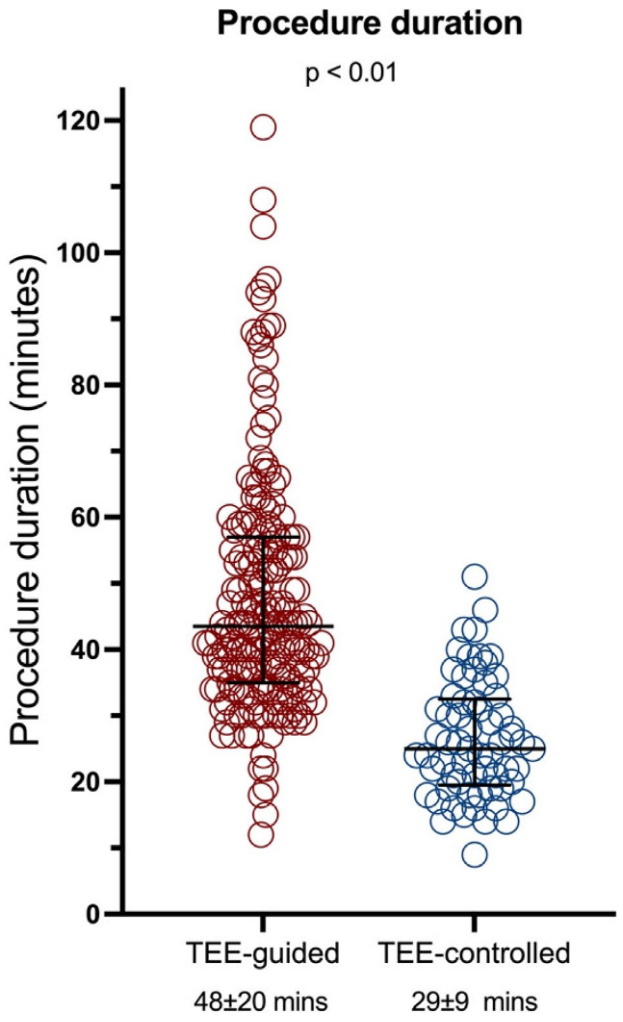

Fig. 1 | 8-5

<0.01). With this respect, there were no differences in thromboembolic events (4.6 % vs 0 %, respectively; $p=0.13$). Atrial fibrillation (7.1 % vs 0 %, respectively; $p=0.02$) and patient-oriented cardiac events (8.6 % vs 0 %, respectively; $p<0.01$) occurred more often in the TEE-guided group, however at later follow-up (22 [9;56] months and 25 [10;60] months, respectively).

Conclusion: While a complete TEE-free PFO closure might have potential procedural risks, a pure TEE-controlled approach seems to be advantageous in terms of procedural aspects with no sign of any acute or long-term hazard.

8-6

Risikofaktoren für linksatriale Thromben/ spontanen Echokontrast in Patienten mit Vorhofflimmern vor transfemoralen Aortenklappenersatz

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Einleitung: Vorhofflimmern ist eine häufige Rhythmusstörung mit hoher Prävalenz bei betagten Patienten. Und viele Patienten, die aufgrund einer Aortenstenose einen transfemoralen Aortenklappenersatz benötigen, leiden an Vorhofflimmern. Ischämische Insulte, basierend auf linksatrialen Thromben oder spontanem Echokontrast, sind eine gefürchtete Folge. Wie bereits bei in diesem Forum von uns gezeigten Untersuchungen (in einem Kollektiv aus Patienten vor einem transfemoralen Aortenklappenersatz) zeigten, ist die Inzidenz solcher Thromben im linken Vorhofsohr bei Patienten mit gleichzeitig bestehender Aortenstenose oder UND Vorhofflimmern besonders hoch. Wir untersuchten mögliche Risikofaktoren, welche Einfluss auf die Entstehung solcher Thromben haben könnte, in unseren Patienten mit Vorhofflimmern ($n=82$).

Methoden: Insgesamt konnten 82 Patienten (38 ♀, mittleres Alter 82 Jahre) für die Analyse herangezogen werden. In 17 Patienten (=20,7 %) konnte mittels transösophagealer Echokardiographie ein linksatrialer Thrombus ($n=5$) beziehungsweise ein linksatrialer Spontankontrast ($n=12$) nachgewiesen werden. Patienten mit Spontankontrast oder solidem Thrombus wurden Patienten ohne Nachweis im weiteren gegenübergestellt. In statistischen Analysen zeigten sich signifikante Unterschiede in serologischen Markern sowie auch klinischer und radiologischer Merkmale (Siehe Tab. 1).

Resultate: Tab. 1

Schlussfolgerungen: Neben Alter, linksventrikulärer Auswurffraktion oder glomerulärer Filtrationsrate konnten auch das Volumen des linken Herzohres als Risikofaktor für die Entstehung von linksatrialen Thromben in unserer Kohorte identifiziert werden. Ein Risikomodell zur Stratifizierung des Risikos betreffend linksatrialer Thromben wäre wünschenswert, um einerseits unnötige transösophageale Echokardiographien oder CT Untersuchungen vor Klappenersatz zu vermeiden, und dennoch das Risiko eines ischämischen Insults basierend auf linksatriale Thromben/Spontankontrast zu vermeiden.

Table 1 | 8-6

Alter (Jahre)	82 (79–85)	81 (78–84)	85 (83–88)	<0.01
Vorhofflimmern - paroxysmal -nonparoxysmal	 26 (31.71 %) 56 (68.29 %)	 24 (36.92 %) 41 (63.08 %)	 2 (11.76 %) 15 (88.24 %)	 0.047
LVEF (%)	60 (55–65)	61 (55 -65)	58 (55 -60)	0.02
Pro-BNP (pg/ml)	2076 (925.5–4298.5)	1705 (773 -3647.5)	4223 (2137.5 -5641)	<0.01
Glomeruläre Filtrationsrate	47.98 (37.08–67.3)	52.52 (37.27–68.46)	40.88 (34.1–46.13)	0.03
Vorhofsohr Volumen (ml/m² KÖF)	147 (112.25–177.75)	140.5 (107.13–170.75)	171.5 (151.75–195)	0.01

8-7

Unveiling cardiac amyloidosis, its characteristics and outcomes among patients with mitral regurgitation undergoing transcatheter edge-to-edge mitral valve repair

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Introduction: Mitral regurgitation (MR) and cardiac amyloidosis (CA) both primarily affect older patients. Data on co-existence and prognostic implications of MR and CA are currently lacking. We aimed to identify prevalence, clinical characteristics and outcomes of MR-CA compared to lone MR.

Methods: Consecutive patients undergoing transcatheter edge-to-edge repair (TEER) for MR at two sites were screened for concomitant CA using a multi-parametric approach including core-lab 99mTc-DPD bone scintigraphy and echocardiography, and immunoglobulin light-chain assessment. Transthyretin-CA (ATTR) was diagnosed by DPD (Perugini Grade-0 negative, 1–3 increasingly positive) and absence of monoclonal protein, and light-chain-(AL)-CA via tissue biopsy. All-cause mortality and hospitalization for heart failure (HHF) served as endpoints.

Results: 120 patients (76.9 ±8.1 years, 55.8 % male) were recruited. DPD was positive in $n=22$ (18.3 %, Grade-1 7.5 %[$n=9$] Grade-2/3 10.8 %[$n=13$]); combined ATTR/AL was diagnosed in one Grade-2, and AL in one Grade-0. Independent predictors of CA were increased posterior wall thickness, and presence of left anterior fascicular block on ECG. Procedural success of TEER (MR reduction ≥1 grade) was similarly good in MR-CA and lone MR (91.3 % vs. 96.9, $p=0.2$). After a median of 1.7 years, 25.8 % had experienced death and/or HHF. MR-CA had worse outcomes compared to lone MR (HR 2.2, 95 % confidence interval [95 % CI] 1.0–4.7, $p=0.039$), driven by a 2.5-fold higher risk for HHF (HR 1.5, 95 % CI 1.1–5.9), but comparable mortality (HR 1.6, 95 % CI 0.4–6.1).

Conclusion: Dual pathology of MR-CA is common in elderly MR patients undergoing TEER, and has worse post-interventional outcomes compared to lone MR.

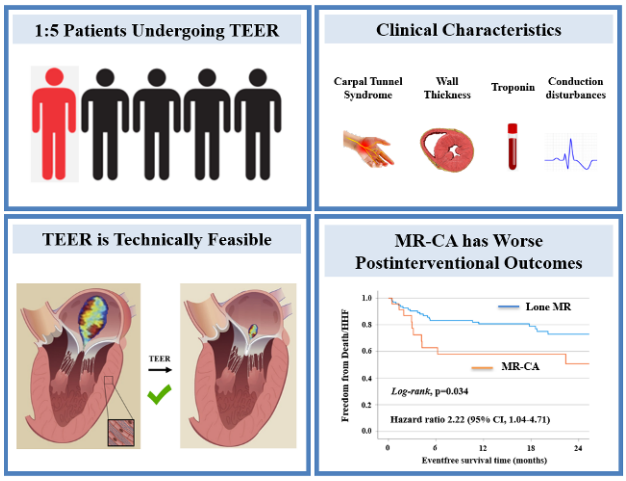

Fig. 1 | 8-7 Dual Pathology Mitral Regurgitation-Cardiac Amyloidosis

8-8

Ten-year trends in unprotected left main percutaneous coronary intervention procedures 2010–2020

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Introduction: Left main coronary artery (LMCA) stenosis is a prime representative of complex and high-risk coronary vessel disease (CVD) that is associated with increased mortality. Due to the development of novel techniques in terms of stent implantation, vascular imaging and hemodynamic support during procedures, indications and possibilities of percutaneous coronary intervention (PCI) have strongly expanded during the last years. Thus, PCI has become increasingly important in the treatment of LMCA disease. However, data on the temporal trends in the treatment of unprotected left main (LM) PCI are lacking.

Methods: In retrospectively screened patient records, a total of 1551 patients who underwent coronary angiography at Vienna General Hospital, Austria, between 2010 and 2020 were found to have a significant LMCA disease, defined as a stenosis of > 50 %. Patients with preexisting coronary artery bypass grafts (CABG) supplying the left coronary artery were excluded from the study. Patient and procedure data was analyzed for linear temporal trends.

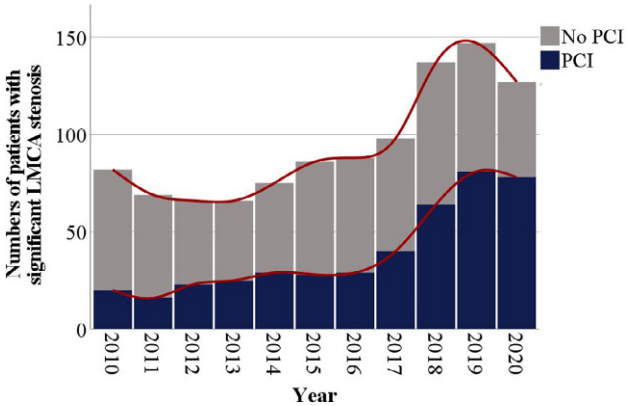

Fig. 1 | 8-8 Increasing number of patients with significant LMCA stenosis between 2010 and 2020

Results: Between 2010 and 2020, a total of 1041 patients with unprotected LMCA disease were recorded, 433 (41.6 %) of which were treated with PCI. Both total patient numbers (2010: $n=82$ vs. 2020: $n=127$) and the relative proportion of LM-PCI (2010: 24.4 % vs. 2020: 61.4 %; $p<0.001$) showed a considerable linear increase over the years (see Fig. 1). We observed increasing patient age (2010: 68.3 ± 12.6 years vs. 2020: 69.4 ± 12.8 years; $p<0.001$) and a higher quantity of individuals presenting with previous PCI (2010: 22.1 % vs. 2020: 49.6 %; $p<0.001$). Most importantly, for the PCI subgroup, 30-day mortality proved to decrease significantly during the studied ten-year period (2010: 40.0 % vs. 2020: 7.7 %; $p<0.001$). For LM-PCI, procedure duration increased (2010: 127 ± 66.3 min vs. 2020: 155.3 ± 64.7 min; $p<0.001$), while contrast agent volume diminished by 20.0 % (2010: 316.3 ± 130.4 mL vs. 2020: 249.8 ± 114.6 mL; $p<0.001$) between 2010 and 2020. Moreover, a trend toward multivessel PCI (in addition to LM PCI) has emerged (2010: 60.0 % vs. 2020: 82.1 %; $p<0.001$).

Conclusion: Data from the years 2010 to 2020 demonstrate the increasing importance of LM-PCI, especially for patients with present indicators of complex and high-risk PCI including higher age and previous PCI. We were able to highlight the increasing expertise of interventional cardiology concerning high-risk PCIs, as illustrated by the impressive decrease in mortality. More attention and research focus should be devoted to this highly vulnerable patient group in the future.

Postersitzung 9 – Herzinsuffizienz 2

9-1

Anemia and iron deficiency in patients with cardiac amyloidosis

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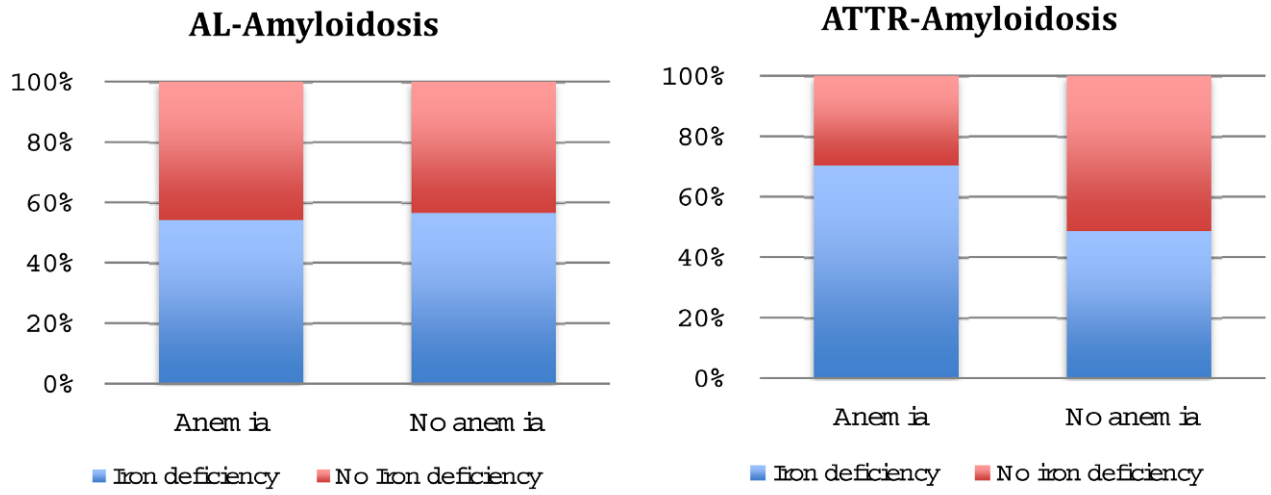
Introduction: Anemia and iron deficiency (ID) are frequent findings in patients with heart failure (HF) and have previously been associated with poor clinical outcomes. Correction of ID has been shown to reduce HF hospitalizations in patients with HF and reduced left ventricular ejection fraction (LVEF). Cardiac amyloidosis (CA) leads to HF regardless of CA subtype (light-chain, AL or transthyretin, ATTR). Data on the prevalence of anemia and ID are scarce in patients with CA and it is not known, whether the presence of these comorbidities have an impact on clinical outcomes.

Methods: Patients with AL and ATTR were prospectively included in a clinical registry. The primary aim of this study was to evaluate the prevalence and the effects of anemia and ID on clinical outcomes in patients with CA and to identify a potential therapeutic target for this patient population.

Results: In total, 289 patients with CA were included in this study (72.7 % with ATTR). Anemia was present in 164 patients and more common in patients with AL. ID was found in 111 patients, the majority of which had ATTR. We found that a low transferrin saturation <20 % is a predictor for adverse clinical outcome in both CA subgroups. When analysing patients according to left ventricular function we found a consistency in these results regardless of LVEF.



* Anemia was present in 56.7% of the entire patient population (69.6% in AL-amyloidosis and 51.9% in ATTR-amyloidosis, p=0.007)
Iron deficiency was defined by a transferrin saturation below 20%.

Fig. 1 | 9-1 Prevalence of anaemia* and iron deficiency in the cardiac amyloidosis patient population, shown for light-chain (AL) and transthyretin (ATTR) amyloidosis. Anemia was present in 56.7 % of the entire patient population (69.6 % in AL-amyloidosis and 51.9 % in ATTR-amyloidosis, $p=0.007$). Iron deficiency was defined by a transferrin saturation below 20 %. * Anemia was present in 56.7 % of the entire patient population (69.6 % in AL-amyloidosis and 51.9 % in ATTR-amyloidosis, $p=0.007$). Iron deficiency was defined by a transferrin saturation below 20 %

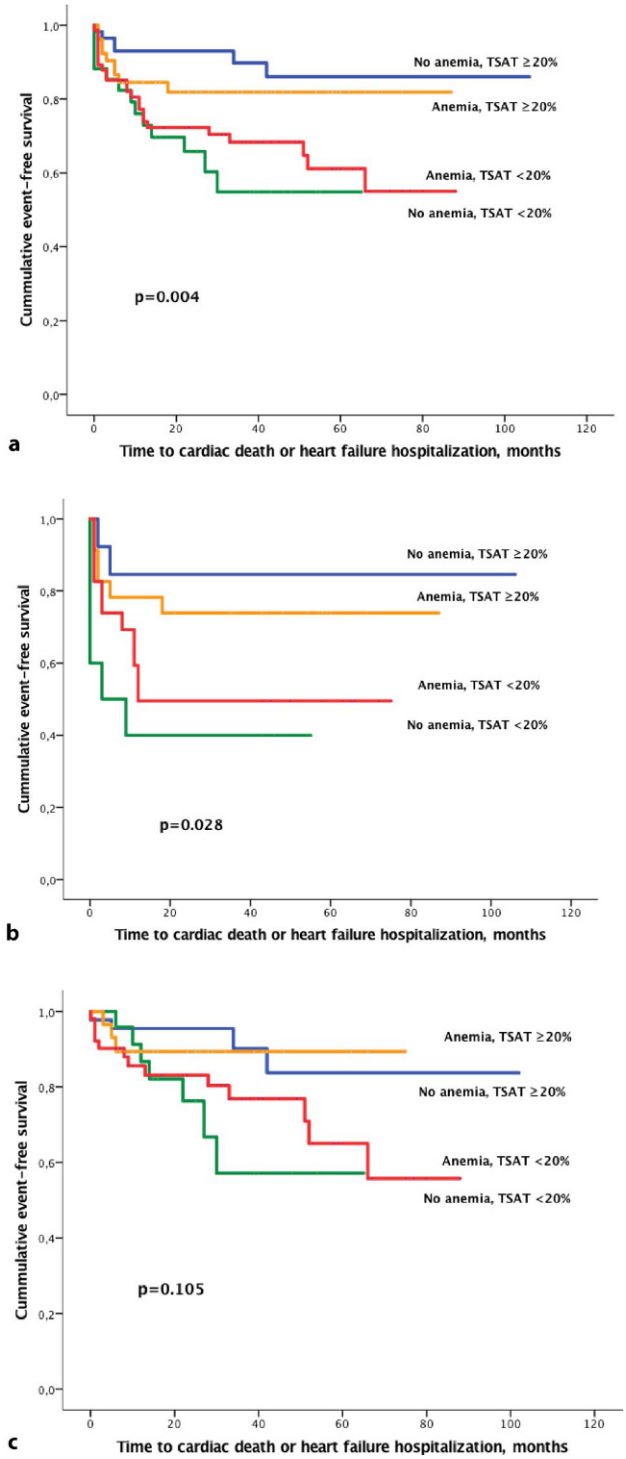


Fig. 2 | 9-1 Kaplan Meier analysis showing outcome of the total cardiac amyloidosis population (panel A), in patients with light-chain amyloidosis (panel B) and in patients with transthyretin amyloidosis (panel C), according to the presence or absence of anaemia and iron deficiency, defined as a transferrin saturation (TSAT) <20 %

Conclusion: Anemia and ID are common and relevant comorbidities in patients with CA. Transferrin saturation is a valuable marker to classify ID, which we identified as an independent predictor for cardiac death and HF hospitalization in patients with CA, regardless of LVEF.

9-2

Predicting HFpEF patient outcome by using the survival tree method

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Introduction: Heart failure with preserved ejection fraction (HFpEF) is a heterogeneous syndrome. Survival tree analyses (STA) are commonly used to investigate event occurrence in complex diseases and depict the algorithmic importance of a group of outcome predictors, which allows a practical approach to decision making in daily routine.

Methods: Consecutive HFpEF patients ($n=427$) were included in this registry. The clinical endpoint was defined as cardiac death or heart failure (HF) hospitalization. STA were used to develop an outcome prediction model. Predictive ability was evaluated by the C-index and the integrated Brier score (IBS).

Results: Out of 76 different variables, a hierarchical combination of three variables was identified by STA: 6-minute walk distance (6-MWD) with a cut-off value of 350 meter (m) was the most important variable regarding clinical endpoint (HR, 18.919; 95 % CI, 2.420–147.887; $p<0.0051$). Patients with 6-MWD <350 m, and Iron <65 µg/dl showed the worst outcome (Fig. 1, node 7).

Conclusion: STA in HFpEF patients identified several easily obtainable clinical parameters as risk factors for a HF-associated event.

9-3

Clinical course of the first 26 patients with cardiac transthyretin-amyloidosis treated with Tafamidis in our center

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Introduction: Cardiac transthyretin (ATTR) amyloidosis, caused by the deposition of transthyretin amyloid fibrils in the myocardium, is a life-threatening disease, characterized by progressive heart failure. The first widely available drug therapy, Tafamidis, which binds to transthyretin, inhibits tetramer dissociation and, thus, amyloidogenesis, was introduced after publication of the ATTR-ACT trial 2018. We report the long-term course of the first set of patients with cardiac transthyretin amyloidosis, treated in our center with Tafamidis.

Methods: All patients were followed regularly at our specialized heart failure outpatient clinic. Tafamidis was provided initially via a compassionate use program from the manufacturer, and via national health insurance afterwards. We performed clinical, functional (6 minute walk test-6 MWT) and biochemical (nt-proBNP) assessment at regular intervals. Tafamidis was withdrawn in case of severe clinical deterioration under treatment. Statistical analysis was performed, using paired t-tests with nt-proBNP values log-transformed for analysis.

Results: We included 27 patients (24 men), mean age at diagnosis was 78 years (SD 6.7, range 59–86). Ejection fraction at baseline was 57 % (range 25–70), median NYHA class was 2 (1–3). Tafamidis was started in all patients, and withdrawn in 4 of them due to progression of heart failure. In the treated cohort, NYHA class was stable (median value 2, range 1–3) throughout follow

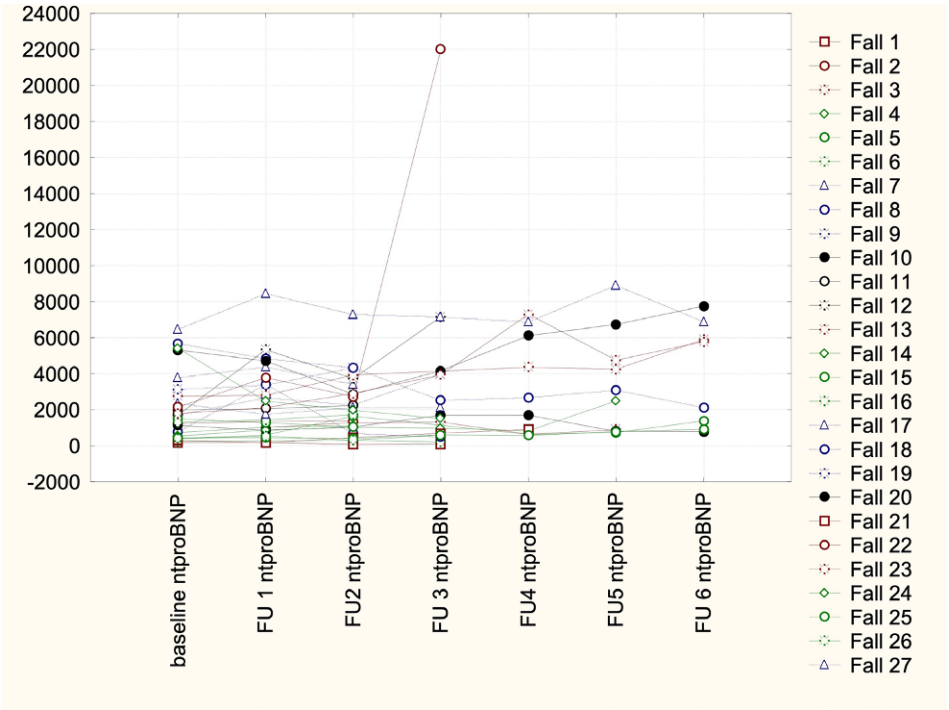

Fig. 1 | 9-3 Time course of nt-proBNP

	n	*Baseline value*	*FU 1 value*	*FU 2 value*	*FU 3 value*	*FU 4 value*	*FU 5 value*	*FU 6 value*	*p-value*
FU days			122	234	349	468	560	627	
Nt-proBNP pg/ml	25	2122	2430						0.13
	26	2049		2124					0.33
	17	2144			3523				0.22
	10	2478				3193			0.06
	9	2721					3614		0.09
	8	2995						3936	0.22
6 MWT m	8	344	371						0.13
	8	387		426					0.11
	4	333			391				0.25

FU ... follow up; 6 MWT ... 6 minutes walk test

Fig. 2 | 9-3 Time course of nt-proBNP and 6 Minute Walk Test

up. There was a numerical increase in nt-proBNP values during follow up, which failed to reach statistical significance (Table 1, Fig. 1). The percentage of patients with decreasing nt-proBNP values during follow up was 28 % (V1), 44 % (V2), 41 % (V3), 10 % (V4), 22 % (V5) and 25 % (V6), respectively. In contrast, 6 minute walk tests (6 MWT) were slightly improved during follow up (Fig. 2). The percentage of patients with improved 6 MWT as compared to baseline was 75 % at visits 1, 2 and 3, respectively. No severe side effects of Tafamidis were observed, and the drug was generally well tolerated.

Conclusion: In our relatively small cohort of patients, the favourable result obtained in the ATTR-ACT trial could be replicated. In particular, the improvement in functional capacity is remarkable.

9-4

Can impaired systolic function be diagnosed by analyses of blood pressure waveforms?

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Introduction: Heart failure with reduced ejection fraction (HFrEF) is a major health problem in Austria. An early diagnosis with widely available and cost-efficient methods remains a desirable goal. In our study we tried to determine whether participants with normal ejection fraction (EF) can be distinguished from patients with reduced EF by using an automated analysis of blood pressure waves.

Methods: The left ventricular ejection fraction of 78 patients was prospectively assessed by echocardiography (apical 4-chamber-view, Simpson method, on a Philips EPIQ-System). The control group was matched in terms of age, gender, height, weight, and brachial blood pressure. We measured pulse waveforms of the radial artery non-invasively using the SphygmoCor-System (AtCor Medical). The measurements were processed with the ARCSolver-algorithm to calculate the parameters S and D from the wave intensity analysis (WIA) and their ratio (SDR). Furthermore we calculated the left ventricular ejection time index (iLVET). Clinical parameters of patients and controls were compared using either t-tests or Mann-Whitney-U-tests, respectively. We also did a Receiver-Operating-Curves (ROC)-analysis in order to determine the capability of discrimination of patients with HFrEF from controls using pulse waveform parameters.

Results: Patients with HFrEF (mean EF = 28 %) and controls (mean EF = 66 %) did not differ significantly concerning age (56 vs. 56 years, $p = 0.597$), gender, height (1.74 vs. 1.73 m, $p = 0.576$), weight (86 vs. 83 kg, $p = 0.590$), prevalence of arterial hypertension (51 vs. 51 %), or systolic (127 vs. 129 mm Hg, $p = 0.359$) or diastolic (79 vs. 79 mm Hg, $p = 0.763$) brachial blood pressure. The mean heart rate of patients and controls was slightly different (65 vs. 61 beats per minute, $p = 0.040$). Diabetes, coronary artery disease and use of ACE-inhibitors/sartans/angiotensin-rezeptor-neprilysin-inhibitors was more frequent among patients than controls. SDR (2.4 vs. 5.7 %, $p < 0.001$) and iLVET (0.39 vs. 0.42, $p < 0.001$) were significantly lower among patients with HFrEF than controls. Furthermore, SDR (AUC = 0.93, 95 % CI = [0.96, 0.88]) and iLVET (AUC = 0.90, 95 % CI = [0.94, 0.83]) could be used to discriminate between patients with HFrEF and controls.

Conclusion: Parameters that have been derived from non-invasive pulse waveform measurements can be used for an early diagnosis of impaired systolic function.

9-5

Malnutrition in patients with chronic heart failure

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Introduction: Malnutrition is highly common in patients with chronic heart failure and often overlooked. It can accelerate disease progression by activating cytokines, causing autonomic dysfunction and cachexia. If malnutrition is detected early, physicians may be able to identify patients who are at high risk for an adverse outcome. Malnutrition in patients can be assessed according to scores, like the prognostic nutritional index [PNI: albumin (g/L) + 5 × total lymphocyte count x 109/L], controlling nutritional status [CONUT: calculated from a sum of scores including albumin, total cholesterol and lymphocyte count] and the geriatric nutritional risk index [GNRI: (1.489*albumin (g/L)) + 41.7* (weight/idealweight)]. Our aim is to assess the prevalence of malnutrition, expressed by PNI, GNRI and CONUT across the spectrum of HF and to further investigate whether these scores are associated with outcome.

Methods: In total, 9733 consecutive patients were included in this study between 2010 and 2020. Patients were classified into one of three heart failure subtypes based on guideline diagnostic criteria: reduced (HFrEF; LVEF <40 %), mildly reduced (HFmrEF; LVEF 40–49 %), or preserved ejection fraction (HFpEF; LVEF ≥50). Malnutrition was assessed based on PNI, GNRI, or CONUT (PNI: Malnutrition <45; absent ≥45/GNRI: <82 severe; 82–91 moderate; 92–98 mild; >98 normal/CONUT: 9–12 severe; 5–8 moderate; 2–4 mild; 0–1 normal). The asso-

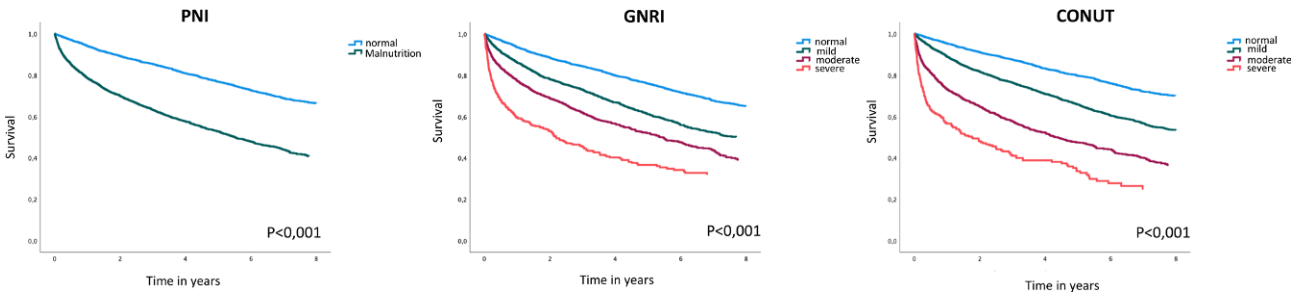

Fig. 1 | 9-5

ciation between the respective nutritional scores and all-cause mortality was assessed.

Results: Of the 9733 patients included, 5680 (58.4 %) were diagnosed with HFpEF, 2214 (22.7 %) with HFmrEF, and 1839 (18.9 %) with HFrEF. Overall, median BMI was 27.5 (IQR 24.5–31.2), 33 % of participants were female, and the median age was 70 years (IQR 61–77). 40 %, 42 %, 63 % of patients were defined as malnourished by PNI, GNRI and CONUT, respectively. During a median follow-up time of 3.71 years (IQR 1.56–6.32) a total of 3159 (32.5 %) deaths were observed. Malnutrition, as indicated by a low PNI or GNRI and a high CONUT score, was associated with worse survival (PNI: HR 2.53 [2.35–2.71], GNRI: HR 1.60 [1.55–1.66], CONUT: HR 1.841 [1.76–1.9], $p < 0.001$ for all). This association remained significant after adjustment for age, sex, kidney status and NT-proBNP (adj. HR, GNRI:1.41 (1.35–1.46), PNI: 1.86 (1.72–2.01), CONUT: 1.52 (1.45–1.60) $p < 0.001$ for all). Interaction analysis confirmed that association with mortality was independent from heart failure type for all scores (p = ns for all). Fig. 1 displays survival curves for nutritional score categories across the spectrum of HF ($p < 0.001$ for all, log-rank test).

Conclusion: Malnutrition as assessed by PNI, GNRI and CONUT is common in patients with heart failure. Malnutrition is associated with higher mortality rates, irrespective of type of heart failure and independent from classical confounder models and even NTproBNP. Based on their additional prognostic value, nutritional scores could be included into routine examination to identify high risk patients.

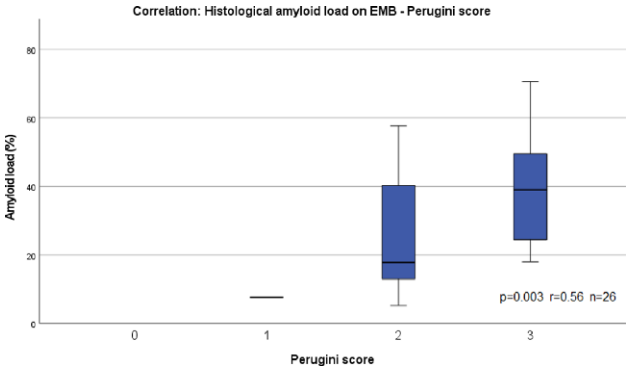

Fig. 1 | 9-6 Correlation between Perugini score and histological amyloid load on EMB

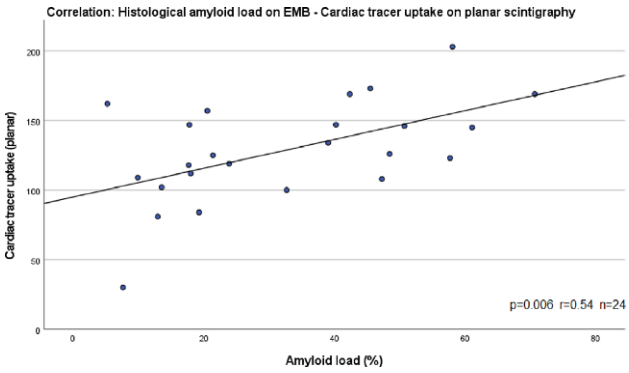

Fig. 2 | 9-6 Correlation between histological amyloid load on EMB and the degree of cardiac tracer uptake on scintigraphic planar scans

9-6

Myocardial amyloid quantification with 99mTc-DPD scintigraphy in cardiac transthyretin amyloidosis

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Introduction: Cardiac transthyretin (ATTR) amyloidosis is a fatal disease caused by the extracellular deposition of misfolded ATTR protein in the myocardium. 99mTc-DPD scintigraphy is a key tool for non-invasive diagnosis of cardiac ATTR amyloidosis. However, its value as a disease monitoring tool has not been systematically assessed. This single-center observational study aimed to compare the extent of histological amyloid infiltration on endomyocardial biopsy (EMB) with the quantification of cardiac 99mTc-DPD uptake (planar, SPECT/CT).

Methods: 26 patients with cardiac ATTR amyloidosis were enrolled. Patients were included in case of (1) EMB-proven ATTR amyloidosis and (2) availability of 99mTc-DPD scintigraphy (reference activity: 550 MBq). Visual interpretation using the Perugini score, quantitative analysis of cardiac 99mTc-DPD uptake by planar scintigraphy and SPECT/CT using regions of interest (ROI) were performed, and heart to whole-body ratio (H/WB) was measured. Histological amyloid load was quantified as percentage of the analysed myocardial tissue using Sulfated Alcyan Blue staining and the Fiji-ImageJ programme. Pearson's and Spearman's correlation were used for correlation analysis and assessment of agreement.

Results: ATTR patients had a median age of 77 [73–79] years and were predominantly male (85 %). An abnormal Perugini score (i. e. 2 or 3) was present in 25 patients (96 %), whereas 1 patient was assigned Perugini score 1 (4 %). Increased cardiac tracer uptake was documented in all patients, both on 99mTc-DPD planar scintigraphy [ROImean 129 ±37] and SPECT/CT [ROImean 369 ±142]. Histologic amyloid burden on EMB was 32 ±19 % on average. It significantly correlated with Perugini score (r = 0.56 p = 0.003), cardiac 99mTc-DPD uptake (planar: r = 0.54 p = 0.006, SPECT/CT: r = 0.48 p = 0.018) and H/WB (r = 0.41 p = 0.046).

Conclusion: We have demonstrated a good correlation between histological amyloid infiltration on EMB and cardiac 99mTc-DPD uptake, illustrating the potential of 99mTc-DPD scintigraphy to yield reliable quantitative information on cardiac amyloid burden.

9-7

Validation of an electrocardiographic algorithm for the detection of cardiac amyloidosis

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Introduction: Despite new therapies, diagnosis of cardiac amyloidosis (CA) is often delayed. We recently developed a simple electrocardiographic (ECG) algorithm to suspect CA without the aid of advanced imaging modalities (Fig. 1).

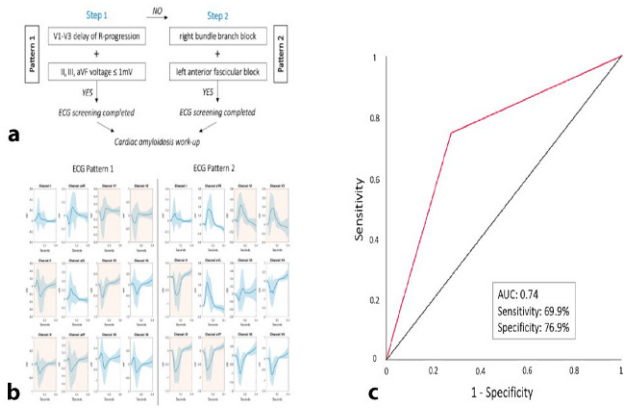

Fig. 1 | 9-7 ECG algorithm for the detection of CA (**a**): In a first step V1 to V3 has to be interpeted. In case of delayed R progression, leads II, III, and aVF should be checked for reduced voltage less than or equal to 1 mV. The presence of both criteria corresponds to pattern 1 and should be followed by guideline-conform diagnostic work-up. In the absence of pattern 1, check for the presence of pattern 2, characterised by a bifascicular block, i. e., RBBB in V1 and V2 and negative concordance in the inferior leads. Mean ECG representations (light blue indicates the standard deviation) of the two ECG patterns (**b**). Receiver operating curve and corresponding area under the curve (AUC) for the diagnostic ECG algorithm for CA (**c**)

Methods: The aim of this study was to validate the algorithms' usefulness in clinical practice. ECG readings from patients with CA, heart failure with preserved ejection fraction (HFpEF), and hypertrophic cardiomyopathy (HCMP) were analyzed in a blinded fashion.

Results: 884 patients were included. Patients with pacemakers were excluded, leaving 827 ECGs (237 CA, 407 HFpEF, 183 HCMP) for final analysis. A characteristic pattern defined by the algorithm was visually perceptible in 165 ECGs (69.6 %) of the amyloidosis patients vs. 114 (28 %) of HFpEF vs. 22 (12.0 %) of HCMP patients ($p < 0.001$). The area under the curve (AUC) for the detection CA was 0.75 with a sensitivity of 69.6 % and a specificity of 76.9 % (Fig. 1). Binary logistic regression analysis revealed that the presence of a distinctive pattern increased the probability of CA with an odds ratio of 7.66 (CI: 5.47–10.72; $p < 0.001$).

Conclusion: This easy-to-use ECG algorithm has proven helpful to suspect CA. Our tool may significantly improve the treatment of heart failure patients by identifying those with amyloidosis-related disease.

Postersitzung 10 – Bildgebung 1

10-1

Prognostic significance of left ventricular functional parameters in relation to infarct location after ST-elevation myocardial infarction

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Introduction: In survivors of ST-elevation myocardial infarction (STEMI), the impact of infarct location on the prognostic significance of left ventricular functional parameters is not well established. The aim of this study was to investigate the prognostic relevance of left ventricular (LV) functional parameters in relation to infarct location in STEMI patients treated with contemporary primary percutaneous coronary intervention (PCI).

Methods: This observational study analyzed 803 patients with STEMI that underwent a cardiac magnetic resonance imaging scan in median 3 (interquartile range [IQR]: 2–5) days after primary PCI. The following LV functional parameters were evaluated: LV ejection fraction, LV global longitudinal strain, fast manual long-axis strain (LAS) and mitral annular plane systolic excursion (MAPSE). Primary endpoint was the occurrence of major adverse cardiac events (MACE) defined as composite of death, re-infarction and congestive heart failure.

Results: Three hundred and sixty nine patients (46 %) had anterior STEMI. These patients had lower LV functional parameters including LV ejection fraction ($p < 0.001$), LV global longitudinal strain ($p < 0.001$), LAS ($p < 0.001$) and MAPSE ($p < 0.014$). MACE was evaluated at a median of 13 (IQR: 12–37) months after STEMI and occurred in 78 patients (10 %). In receiver operating curve analysis, the predictive value of LV ejection fraction, LV global longitudinal strain, LAS and MAPSE was 0.59 ($p = 0.013$), 0.64 ($p < 0.001$), 0.67 ($p < 0.001$) and 0.66 ($p < 0.001$), respectively. When divided according to infarct location, MACE occurred in 47 (13 %) anterior STEMI patients, and in 31 (7 %) non-anterior STEMI patients, respectively. Area under the curve for the prediction of MACE in anterior vs. non-anterior STEMI was 0.59 vs 0.55 for LV ejection fraction, 0.61 vs 0.63 for LV global longitudinal strain, 0.69 vs 0.62 for LAS and both 0.66 for MAPSE. In multivariable analysis, LAS was independently associated with an increased risk of MACE (hazard ratio: 1.20; 95 % confidence interval: 1.10–1.30; $p < 0.001$) in anterior STEMI, whereas in non-anterior STEMI, LV global longitudinal strain was an independent predictor of MACE (hazard ratio: 1.22; 95 % confidence interval: 1.08–1.38; $p = 0.002$).

Conclusion: Fast manual LAS emerged as independent predictor of MACE in anterior STEMI treated with contemporary primary PCI whereas LV global longitudinal strain was independently associated with MACE in non-anterior STEMI.

10-2

Speckle tracking echocardiography in the pre- and postprocedural assessment of His-/left bundle optimised cardiac resynchronisation therapy

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Introduction: There is an emerging role for conduction system pacing to achieve resynchronisation in patients suffering from heart failure with reduced ejection fraction and interventricular dyssynchrony. New data is emerging that resynchronisation may be more complete with pacing at the level of both the specialised conduction system in conjunction with sequential LV pacing in areas of delayed myocardial activation, referred to as His-/left bundle optimised CRT (HOT/LOT-CRT) [1]. This is a clinical case regarding echocardiographic guiding by two dimensional speckle tracking and peak systolic dispersion in particular for conduction system pacing optimised cardiac resynchronisation therapy in a patient with heart failure due to ischemic cardiomyopathy and left bundle branch block.

Methods: A 74 year old male patient with a known history of ischemic cardiomyopathy and broad left bundle branch block (QRS duration 184 ms) was eligible for cardiac resynchronisation therapy (CRT) with a Class I Level A indication according to ESC heart failure guidelines 2021 [2]. The patient had a history of beta-blocker intolerance due to severe bronchial asthma and suffered from chronic arterial hypotension, so that optimal medical therapy was limited. We decided to provide this patient with the most optimal option available and HOT/LOT-CRT implantation was performed. Due to high His-pacing threshold, left bundle branch area (LBBA) pacing was favoured. Pre- and postprocedural transthoracic echocardiography was performed to assess left ventricular volumes, left ventricular ejection fraction (LVEF) and global longitudinal strain (GLS) assessed from 17 strain segments by two-dimensional (2D) speckle tracking analysis. Furthermore, the mechanical dispersion was calculated, defined as the standard deviation of contraction duration of all segments (PSD–peak systolic dispersion, derived from TTP–time to peak systolic strain).

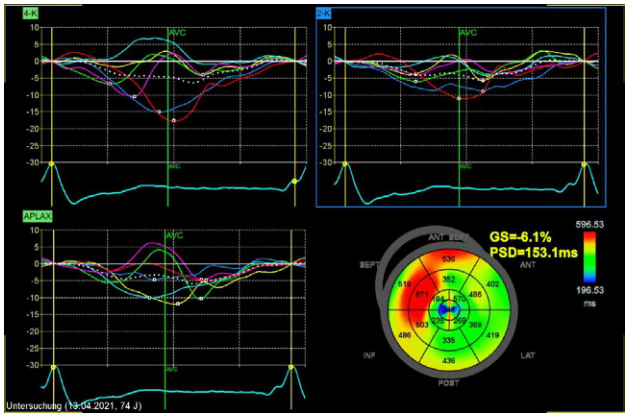

Fig. 1 | 10-2 Initial speckle tracking analysis with GLS and PSD

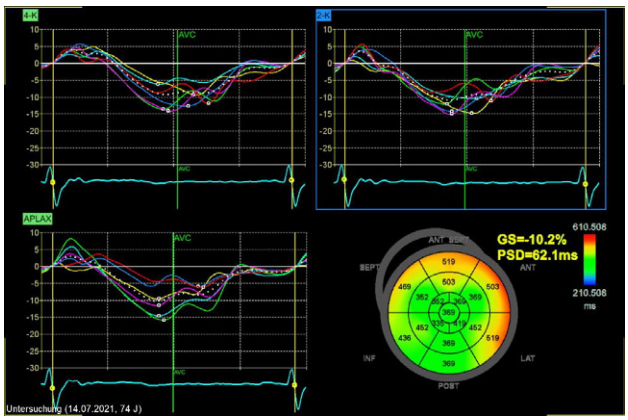

Fig. 2 | 10-2 Follow up speckle tracking analysis with GLS and PSD

Results: During pacemaker implantation the QRS duration reduced from 184 ms at baseline to 150 ms with LBBA-pacing and to 116 ms combined with simultaneous LV-pacing through a lead in a posterolateral branch of the coronary sinus. The patient was discharged two days after implantation in good clinical status. Three months later the patient presented as scheduled to our outpatient clinic to evaluate clinical status and echocardiographic outcome. A significant clinical and echocardiographic improvement was observed. Even though the patient was not on optimal medical therapy he reported less dyspnea on exertion (corresponding to NYHA class I–II), there were no signs of decompensated heart failure, the NT-proBNP level decreased and echocardiographic parameters improved as followed: LVEDV from 224–139 ml, LVESV from 160–79 ml, LVEF from 29–44 %, GLS from –6.1 to –10.2 % and mechanical dispersion by PSD from 153.1–62.1 ms.

Conclusion: Myocardial strain imaging by 2D speckle tracking has a wide spectrum of clinical utility and has proven to be a valuable tool for decision making in different clinical scenarios. To assess the presence of mechanical LV dyssynchrony various echocardiographic techniques have been explored with speckle tracking being one of the most accurate. Time to peak systolic dispersion with PSD in particular is a suitable method to evaluate patients who are eligible for resynchronisation therapy [3]. To our knowledge this is the first case that PSD by speckle tracking echocardiography has been used for pre- and post-HOT/LOT-CRT implantation assessment. With the use of these parameter(s) we could document not only the electrical but also the mechanical resynchronisation, which corresponded to the clinical improvement of the patient. We believe that time to peak systolic dispersion with PSD could prove to be a useful tool in guiding not only classic resynchronisation therapy but especially HOT/LOT-CRT Implantation.

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10-3

Atherosclerosis-Progression in coronary arteries compared to periphery vessels

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Introduction: Cardiovascular diseases, are one of the leading causes of death worldwide. In the last years, the assessment of diagnostic tools for atherosclerosis in cardiac arteries has gained great scientific interest. In recent decades, various non-invasive methods have been developed to accurately assess atherosclerotic burden and thereby evaluate individual patient risk, such as ultrasound and computed tomography. Three-dimensional ultrasound is a promising new approach for the non-invasive quantification of peripheral plaque volume. The aim of this study was to compare the development of atherosclerosis in peripheral, measured by 3D-volumetry, and coronary vessels, examined by computed tomography.

Methods: In this prospective, single-centre study, we included 61 patients with a low to moderate cardiovascular risk (6–20 %) according to the Framingham Risk Score. 25 of these patients were examined only once, while the other 36 were examined a second time after 2–3 years. 3D-sonographic examination was performed to measure peripheral atherosclerotic plaques in carotid and femoral arteries. Furthermore, a computed tomography was established, quantifying the coronary-calcium-score. The plaque volumes were then compared to the CCS-values using IBM SPSS Version 27.0.1. Ultimately, venous blood samples were taken, measuring the values of different cardiac, and systemic markers.

Results: Analyzing the patients' data, a significant correlation ($r = 0.297$; $p = 0.020$) was found between the baseline CCS levels and the combined plaque volume of carotid and femoral arteries. Furthermore, the CCS-score and the total plaque volume correlated significantly within the 3-year follow-up ($r = 0.594$; $p = 0.042$).

Conclusion: These results show a significant correlation between peripheral and coronary plaque volume and even more the progression of atherosclerosis in the peripheral vessels and coronaries after 2–3 years. Furthermore, these findings suggest that total peripheral plaque volume measured by 3D sonography could be used as a diagnostic tool to determine atherosclerosis in the coronary arteries.

10-4

Comparison of hepatic tissue characterization between T1-mapping, non-contrast computed tomography, and liver fibrosis scores

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Introduction: Non-contrast computed tomography (CT) is frequently used to assess liver tissue, especially for non-alcoholic/metabolic fatty liver disease (NAFLD/MAFLD), which is associated with cardiovascular disease and dismal outcomes. Although liver biopsy is considered the gold standard, in clinical routine, standardized scores and non-contrast computed tomography (CT) are used to diagnose NAFLD/MAFLD. On standard cardiac T1-maps on cardiovascular imaging (CMR) exams, used for myocardial tissue characterization, hepatic tissue is also visible. However, hepatic T1-times have never been assessed in a CMR cohort. The aim of the study was to investigate whether hepatic T1-times on CMR are associated with (1) hepatic HU values on non-contrast CT scans and (2) established liver fibrosis scores.

Methods: We retrospectively identified patients undergoing a non-contrast CT including the abdomen, a CMR including T1-mapping, and laboratory assessment within 30 days. Patients with storage disease, including amyloidosis, iron overload and Fabry's disease, were excluded.

Results: We identified 271 patients (62 ± 15 y/o, 49 % female) undergoing non-contrast CT and CMR T1-mapping within 30 days. Mean hepatic HU were 54 ± 11 on CT and native T1-times were 598 ± 102 ms on CMR and there was a weak, but significant, correlation between these parameters ($r = -0.136$, $p = 0.025$). Also there was a weak correlation between the NAFLD score and hepatic HU values ($r = -0.147$, $p = 0.025$) and native liver T1-times ($r = -0.126$, $p = 0.045$). On age and sex adjusted regression analysis, native liver T1-times were associated with a more pronounced cardiac risk profile, including lower LV and RV ejection fractions ($p = 0.045$ for both) and higher NT-proBNP values ($p = 0.004$). Inter-rater variability was excellent for both CT and CMR characterization of hepatic tissue (ICC for HU on CT: 0.87 ICC for native T1-times on CMR: 0.86).

Conclusion: Hepatic T1-times are easy to assess on standard T1-maps on CMR but only weakly correlated with hepatic HU values on CT and clinical NAFLD/MAFLD scores. Liver T1-times, however, are linked to impaired systolic function and higher natriuretic peptide levels. The prognostic value and clinical usefulness of liver T1-times in CMR cohorts warrants further research.

10-5

Changes in cardiac index four months after STEMI–A phase-contrast cardiac magnetic resonance imaging study

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Introduction: A Cardiac Index (CI) of <2.2 l/min/m^2 is one of the strongest predictive parameters after ST-elevation myocardial infarction (STEMI). Phase-Contrast Cardiac Magnetic Resonance Imaging (PC-CMR) allows non-invasive measurement of CI. No data is available about changes in CI after STEMI.

Methods: Comprehensive CMR was performed in 267 stable and contemporarily revascularized patients at baseline and four months after STEMI. Forward stroke volume was assessed at the level of the ascending aorta by PC-CMR and multiplied with heart rate to calculate CI. Left ventricular volumes, ejection fraction (LVEF) and global longitudinal strain (GLS) were determined by cine CMR.

Results: Of 66 patients (24.7 %) with a CI <2.2 l/min/m^2 at baseline 40 patients improved their CI to >2.2 l/min/m^2 after four months. 19.8 % of patients (n = 53) demonstrated a depressed CI of <2.2 l/min/m^2 at follow-up. Significant improvement occurred of EDVI (84 ± 18 vs. 76 ± 19 ml/m^2, p < 0.001), LVEF (49 ± 10 vs. 51 ± 11 %, p < 0.001) and GLS (–12.4 vs. –12.9 %, p = 0.003). Changes in CI demonstrated weak correlation with changes of EDVI, LVEF or GLS (r = –0.2; r = –0.1; r = 0.2). Patients with a CI of <2.2 l/min/m^2 4 months after STEMI were older (62 vs. 55 yrs, p < 0.001) and had higher NT-proBNP values (401 vs. 178, p = 0.004). Functionally they demonstrated larger ventricles (89 vs. 85 ml, p = 0.03), lower LVEF and GLS values (52 vs. 48 %, p = 0.003; –12.1 vs. –13.2, p = 0.008) at baseline.

Conclusion: A depressed CI was still common four months after STEMI despite revascularization. About half of these patients did not demonstrate a depressed CI at baseline.

Postersitzung 11 – Basic Science 2

11-1

Perinuclear mitochondria are functionally affected in heart failure and alter nucleoplasmic Ca2+ signaling

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Introduction: Despite major improvements in available therapeutic options, heart failure (HF) remains one of the leading causes of death worldwide. While commonly used pharmacotherapeutics target systemic changes in the neurohormonal status of HF patients, no intervention that directly improves cardiomyocyte function and viability has been successfully implemented in clinical practice. In cardiomyocytes mitochondrial dysfunction has been identified as a hallmark of heart failure development. And, although initial studies recognized the importance of different mitochondrial subpopulations, there is a striking lack of direct comparison of intrafibrillar (IF) vs. perinuclear (PN) mitochondria during the development and progression of HF. Furthermore, the functional consequences of mitochondrial dysfunction on nuclear signaling, including Ca2+ cycling, are yet to be elucidated.

Methods: Here, we use electron microscopy and live cell confocal imaging to examine the morphology and functional properties of IF vs. PN mitochondria in pressure overload-induced cardiac remodeling and failure in mice. To induce heart failure mice undergo trans-aortic constriction (TAC). As a proof-of-principle for clinical relevance of our findings, we repeated a subset of experiments in non-failing and failing human cardiomyocytes.

Results: We could demonstrate that IF mitochondria in HF are morphologically altered. However, functionally PN mitochondria from failing cardiomyocytes are more susceptible to changes compared to IF mitochondria at baseline and under physiological stress protocol. These measured changes include mitochondrial membrane potential (ΔΨm), ROS generation and impairment in Ca2+ uptake. We also demonstrated, for the first time, that under normal conditions PN mitochondrial Ca2+ uptake shapes nucleoplasmic Ca2+ transients (CaTs) and prevents nucleoplasmic Ca2+ overload.

Conclusion: Loss of PN mitochondria Ca2+ buffering capacity translates into increased nucleoplasmic CaTs and may explain disproportionate rise in nucleoplasmic [Ca2+] in failing cardiomyocytes at increased stimulation frequencies. Therefore, a previously unidentified benefit of restoring the mitochondrial Ca2+ uptake may be normalization of nuclear Ca2+ signaling and alleviation of altered excitation-transcription, which could be an important therapeutic approach to prevent adverse cardiac remodeling.

11-2

Transcriptomic and proteomic profiling of human diabetic heart disease

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Introduction: Studies in animal models demonstrated the capability of type 2 diabetes (T2D) to induce cardiac dysfunction in the absence of vascular disease. However, whether and how T2D also impairs structure and function in human hearts remains poorly understood.

Methods: Here, we performed transcriptional and proteomic profiling of left ventricular samples of 8 subjects with T2D, preserved EF (63.5 %) and no history of ischemic heart disease (= diabetic cardiomyopathy; DbCM), 7 subjects with T2D, reduced EF (26.9 %) and non-significant ischemic heart disease (= diabetic heart failure; DbHF), and 15 non-diabetic individuals with normal EF (64.7 %) serving as controls.

Results: Among 1168 proteins identified by LC-MS/MS, 146 proteins were differentially regulated in DbHF, but only 66 in DbCM. Pathway analysis revealed downregulation of energy metabolic proteins, but upregulation of proteins involved in oxidative stress and inflammatory response. In DbCM, pathways of structural remodeling, cardiomyocyte proliferation, and mechanotransduction were upregulated. Bulk RNA sequencing revealed 1795 differentially regulated genes in DbHF, and 527 in DbCM, with only 128 genes being commonly regulated. DbHF, but not DbCM, could be clearly discriminated from controls by hierarchical clustering. While inflammation/immunity were major regulated pathways in DbHF, extracellular matrix remodeling and cellular growth were the most regulated pathways in DbCM.

Conclusion: Thus, the differential regulation of biological pathways in DbCM versus DbHF suggests the existence of two distinct disease entities rather than DbHF being an advanced disease stage of DbCM.

11-3

Physical exercise promotes DNase activity enhancing the capacity to degrade cell-free DNA

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Introduction: Physical activity is a potent non-pharmaceutical intervention to prevent and reduce chronic conditions. Exercise induces a short-term rise in blood neutrophil counts and plasma cell-free (cf) DNA. Low DNase activity as well as high levels of cfDNA can promote inflammation and are associated with worse outcome in cardiovascular disease. Therefore, we investigated the effect of consequent endurance training on cfDNA levels and the impact on DNase activity as major clearance mechanism.

Methods: In total, 98 subjects were recruited out of the staff of the Austrian Federal Ministry of Defence. Participants were instructed to perform at least 75 min/week of vigorous or 150 min/week of moderate intensity endurance training. Performance at the beginning and the end of the study was assessed by ergometry. Patient characteristics were documented and blood samples were drawn five times in two-month intervals. cfDNA was measured using a fluorescent DNA binding dye and DNase activity was assessed by single radial enzyme diffusion assay.

Results: Subjects showed a significant decrease of cfDNA levels and a concurrent increase of DNase activity comparing baseline to eight-month follow-up. The cohort was then stratified into four groups according to their initial fitness status and the performance gain over the study period. A significant increase of DNase-I activity was exclusively observed in groups having achieved a performance gain, while cfDNA levels declined or remained constant.

Conclusion: The exercise-induced increase of DNase-I activity supports physical activity as therapeutic intervention to lower chronic disease burden. The implicated increased capacity to degrade neutrophil extracellular traps might especially benefit patients with a high-risk cardiovascular profile.

11-4

The role of myeloid-derived suppressor cells in cardiac regeneration after ST-segment elevation myocardial infarction

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Introduction: Cells of the innate and adaptive immune system are important mediators of cardiac regeneration after acute myocardial infarction (AMI). Cardiac regeneration is divided into two distinct phases of immunological intervention: an initial pro-inflammatory phase followed by a reparative anti-inflammatory phase ensuring cellular repair. In particular, regulatory T-cells (Tregs) and myeloid-derived suppressor cells (MDSCs) have been described to positively affect cardiac repair. Both cell types possess immune suppressive properties and elaborate regulative functions throughout the process of anti-inflammatory regeneration. In both cases, various cytokines have been shown to regulate MDSC and Treg expansion and effector functions. Our study aims to identify patient clusters presenting with specific cytokine combinations that determine Treg and MDSC development as well as cardiac regeneration after ST-segment elevation myocardial infarction (STEMI).

Methods: Flow cytometry-based multiplex analysis of 25 cytokines was performed in baseline and 72 h post-event plasma samples of 43 STEMI patients. Linear regression analyses were performed to correlate cytokine levels with left ventricular ejection fraction (EF) and microvascular obstruction (MVO) 6 months after STEMI, as measured by cardiac magnetic resonance (CMR). A bioinformatical pipeline using principal component analysis (PCA) was designed to identify patient-specific cytokine combinations associated with cardiac performance. Confirmatory in vitro experiments to analyse the impact of PCA-identified cytokine patterns on Treg and MDSC frequency/functionality are currently ongoing.

Results: IL-2 plasma concentrations at baseline and 72 h post-STEMI positively correlated with cardiac EF. IL-2, as a Treg stimulatory factor, significantly correlates with cytokines such as IL-1β, IL-4 and IL-6. On the other hand, MRP8/14 being involved in the development of myeloid cells, could be associated with inflammatory markers, for example SAA and MMP-9, promoting the formation of immunosuppressive MDSC. The potential of these cytokine combinations to influence MDSC/Treg functionality are currently assessed using in vitro co-culture assays with primary PBMCs.

Conclusion: The identification of specific cytokine combinations that regulate the development, expansion and effector functions of reparative cell populations such as Tregs and MDSC might represent an interesting approach to increase our insights regarding cardiac repair mechanisms after STEMI.

11-5

Effects of pericyte and smooth muscle specific expression of CXCL12 on cardiac development and repair

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Introduction: Ischemic cardiomyopathy as a result of myocardial infarction represents the most common cause of heart failure. The chemokine CXCL12 and its receptors CXCR4/CXCR7 facilitate myocardial repair after myocardial infarction (MI) and play a fundamental role in cardiovascular development. However, cell- and tissue-specific effects of CXCL12 are poorly understood, limiting the development of targeted therapies. Therefore, we aimed to examine the role of a pericyte and smooth muscle (SM) cell-specific CXCL12 knockout (KO) in cardiac development and after MI.

Methods: We generated a SM 22-alpha-Cre and a pericyte specific NG2-Cre driven mouse model to ablate CXCL12 specifically in smooth muscle cells (SM-CXCL12−/−) and in pericytes (NG2-CXCL12−/−). Genotyping of animals was performed using ear hole biopsy by PCR. Hearts were analyzed morphologically by histology and immunofluorescence. Cardiac function and heart dimensions were determined by echocardiography. In addition, myocardial infarction was induced in the animals and the outcome was further investigated.

Results: Immunofluorescence staining of heart sections revealed high expression of SDF-1 in the smooth muscle layer of arterial blood vessels, whereas very moderately staining was detected in pericytes. Consequently, NG2-SDF-1−/− mice did not show increased mortality, behaved inconspicuously and did not show any obvious developmental defects. Echocardiographic analysis of cardiac function in NG2-SDF-1−/− mice without MI showed no significant difference in left ventricular (LV) performance and no evidence of abnormal geometry of the ventricles compared with controls. The SM-CXCL12−/− mice did show higher embryonic lethality and development abnormalities. They also developed postnatal severe hypertrophy, severe coronary vascular defects and had a reduced heart function. One-way ANOVA analysis showed a significant difference for the SV ($F(2, 22) = [6.125]$, $p = 0.0077$). The mean values of SV between the control group and the SM-CXCL12−/− mice were significantly different ($p = 0.0056$, 95 % C. I. = [3.850, 23.56]) in the following Turkey's multi comparison test, while the difference between control group and NG2-SDF-1−/− mice was not significant. Also, for fraction shortening the one-way ANOVA test showed a significant difference ($F(2, 22) = [7.739]$, $p = 0.04$). But the following Turkey's multi comparison test was not able to find a significant difference between any groups. For other parameters like ejection fraction, LV mass or LV internal dimension could not reach any significant results.

Conclusion: Verification of infarct induction by histology is still required. Perhaps, after exclusion of mice without infarction, we will be able to present even clearer results Ultimately, this work aims to better characterize myocardial repair mechanisms after infarction. This seems to be a crucial step for the implementation of new, targeted therapies for the prevention and treatment of ischemic cardiomyopathies.

11-6

Left ventricular diastolic suction induced by intraventricular negative pressures: an experimental pressure volume study

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Introduction: For decades, lower body negative pressure (LBNP) has been a tool to study compensatory mechanisms during central hypovolemia. So far, underlying hemodynamic mechanisms were assessed non-invasively in most instances.

Methods: The aim of this investigation was to evaluate the impact of graded LBNP at three different levels of seal as well as during beta-adrenergic stimulation by invasive pressure-volume (PV) analysis. Assessment of PV-loops was performed in eight healthy anaesthetized pigs, that were put in a vacuum box to achieve separation from atmospheric pressure. LBNP was applied at three consecutive locations: (i) cranial (10 cm below xiphoid process), (ii) medial (mid-position between cranial and caudal), (iii) caudal (level of iliac spine); Level (iii) was repeated under dobutamine infusion. At each level, baseline measurements were followed by application of incremental LBNP steps of –15, –30 and –45 mm Hg.

Results: According to the Frank-Starling mechanism, graded LBNP progressively reduced left ventricular (LV) stroke volume following a decrease in LV end-diastolic volume. LV capacitance was not affected. Negative intraventricular minimal pressures were observed during dobutamine-infusion as well as at higher levels of LBNP. Of note, incremental LV negative pressures were accompanied by increasing diastolic suction volumes, derived by extrapolating the volume at zero transmural pressure, the so-called equilibrium volume (V0), related to LV SV.

Conclusion: Preload reduction via LBNP shifts the PV loop to smaller volumes and end-systolic volume below V0, which induces negative LV pressures, hence increases LV suction. We conclude that diastolic suction plays a crucial role in counterbalancing central hypovolemia.

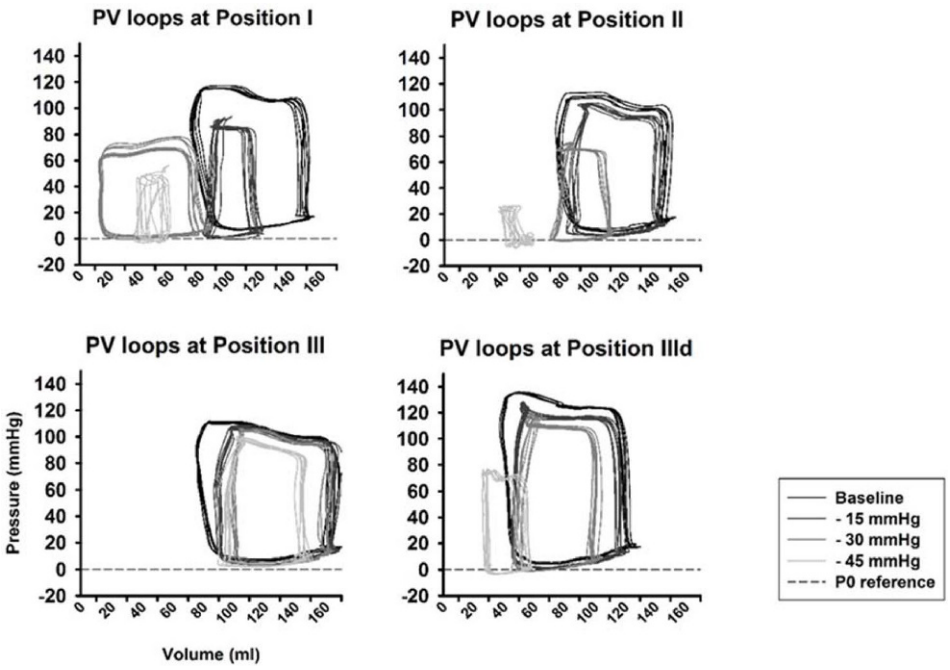

Fig. 1 | 11-6 Direct tracing of pressure-volume loops (PV-loops) (x-axis volume in ml, y-axis pressure in mm Hg) at positions I–III and steps i–iii (amount of negative pressure) show the pressure- and volume lowering effects of LBNP. Compared to position II and III left-shift-effects are more pronounced at sealing position I

11-7

Preventing CD62P-mediated leukocyte infiltration and activation enhances thrombus resolution in mice

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Introduction: Deep vein thrombosis and its complication pulmonary embolism and is a major health problem with an average annual incidence rate of 104–183 per 100,000 person-years. After thrombus formation its resolution is essential to re-establish blood flow. In this study we aim to analyse the effect of CD62P-mediated cell migration and activation on thrombus resolution post thrombus formation.

Methods: Thrombus formation was induced by inferior vena cava ligation and mice were treated after 1 day with a CD62P-blocking antibody or isotype. The thrombus and the surrounding vessel were extracted for immunohistochemistry or flow cytometry. Data were analysed by unpaired Student's t-test or ANOVA.

Results: Localising neutrophils and macrophages in the thrombotic lesion revealed that they enter the thrombus and vessel wall from the caudal site. Neutrophils were predominantly present one day and monocytes/macrophages three days after vessel ligation. As leukocyte extravasation is promoted by endothelial and platelet CD62P, we blocked CD62P at day 1 after thrombus formation. This reduced aggregates between platelets and neutrophils or Ly6Chigh monocytes compared to isotype-treated controls, leading to diminished neutrophils and Ly6Chigh monocytes in the cranial thrombus part. Continuous observation of thrombus volume by ultrasound revealed an accelerated thrombus breakdown after blocking CD62P, confirmed by decreased thrombus weight and length. To identify CD62P-mediated effects on thrombus structure, we applied scanning electron microscopy and observed reduced fibrin density in thrombi of anti-CD62P-antibody-treated mice. Corresponding, we found reduced tissue factor expression associated with macrophages and reduced neutrophil activation after CD62P inhibition.

Conclusion: We propose a CD62P-mediated cross talk of vessel wall, platelets, monocytes and neutrophils resulting in activation of innate immune cells and increased tissue factor expression. This initial activation of immune cells strengthens the thrombus and delays subsequent resolution processes.

11-8

Plasma eicosanoid profiling in the course of proprotein convertase Subtilisin-Kexin type 9 inhibition: Insights from a metabolomic analysis

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Introduction: Treatment with monoclonal antibodies targeting circulating proprotein convertase subtilisin-kexin type 9 (PCSK9) was found to reduce all-cause mortality in addition to cardiovascular events, suggesting pleiotropic effects beyond lipid-lowering. Eicosanoids are bioactive metabolites involved in cardiovascular disease and have not yet been studied in the course of PCSK9 inhibition.

Methods: In this prospective translational single-center study, plasma samples were collected from 64 patients before and after initiation of PCSK9 inhibitor treatment. Metabolomic analyses were performed using liquid chromatography coupled to high-resolution mass spectrometry.

Results: A total of 62 bioactive eicosanoids were detected. Among the metabolites, four were significantly decreased by PCSK9 inhibition after one month and remained stable after 6 months (Fig. 1): arachidonic acid ($p=0.003$), 12,13-DiHOME ($p<0.001$), 9-HpODE_9.91 ($p=0.007$) and HpODE_7.71 ($p=0.011$). Phospholipase A2 levels were reduced by 40 % after 1 month ($p=0.003$) and by additional 50 % after 6 months of treatment ($p=0.015$), but did not correlate with eicosanoids ($p=0.057$). The change in arachidonic acid levels during treatment resulted in a significant increase in the ratio of omega-3 to omega-6 polyunsaturated fatty acids ($p=0.002$).

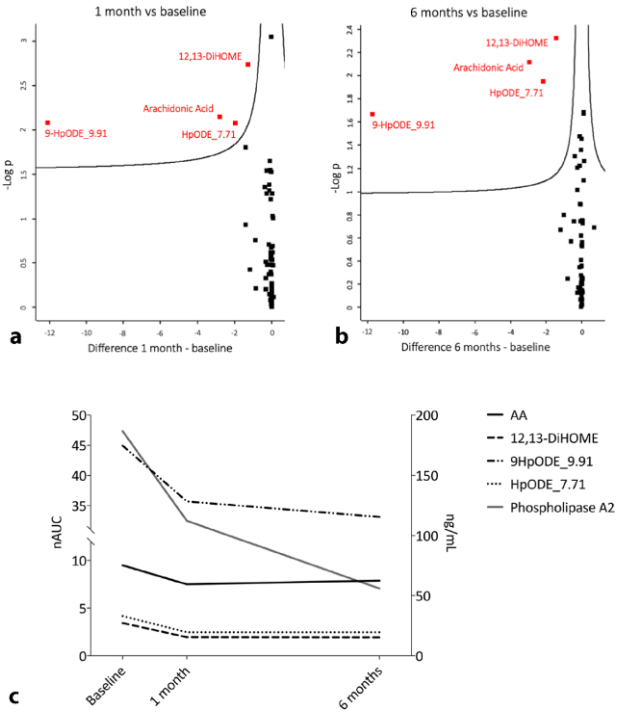

Fig. 1 | 11-8 Changes of eicosanoids and phospholipase A2 following PCSK9 inhibition

Conclusion: PCSK9 inhibition leads to significant changes in the eicosanoid profile already after one month, in particular to a downregulation of arachidonic acid. This discovery complements the presumed pleiotropic effects of PCSK9 inhibition and may provide additional benefit in the treatment of atherosclerotic disease.

Postersitzung 12 – Kardiologisches Assistenz- und Pflegepersonal

12-1

Die Rollen und Aufgaben einer Advanced Practice Nurse in der Versorgung von Menschen mit chronischen Herzerkrankungen – Ein Scoping Review

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Einleitung: Advanced Practice Nurses (APNs) sind hoch spezialisierte Pflegepersonen, die ein großes Fundament an Erfahrungswissen in der klinischen Praxis aufgebaut sowie ein Masterstudium abgeschlossen haben [1]. Sie verfügen über ein breites Feld an Kompetenzen: Zur Darstellung dieser hat sich in der Literatur das Hamric-Modell etabliert [2]. Im deutschsprachigen Raum sind APNs noch nicht verbreitet. Aufgrund der demographischen Entwicklung und der steigenden Anzahl an chronisch herzkranken Personen wird aber auch hierzulande die auf die akute Krankheitsversorgung ausgerichtete Gesundheitsversorgung transformiert und auf die langjährige Versorgung dieser Personengruppe konzentriert. Die Effektivität des APN-Konzeptes in diesem Vorhaben konnte bereits durch vorangegangene Forschungsarbeiten festgestellt werden [3]. Diese Arbeit soll die möglichen Rollen und Aufgaben der APN in der Versorgung von chronisch herzkranken Patient*innen aufzeigen und dadurch weitere Ansätze für die Umsetzung des APN-Konzeptes liefern.

Methoden: Als Methode wurde das Scoping Review gewählt, da durch diese Art von Review ein breiter Überblick über den Stand der Forschung gegeben werden kann. Die Literaturrecherche wurde in PubMed, Cumulative Index to Nursing and Allied Health Literature (CINAHL) und der Cochrane Library durchgeführt. Zur Datensammlung wurden anhand der Forschungsfrage die zentralen Schlüsselwörter definiert und Einschlusskriterien bestimmt. Nach einem Titel- und Abstract- sowie Volltextscreening wurden alle Artikel einer Qualitätsbewertung durch die Bewertungsbögen des Joanna Briggs Institute oder dem AGREE II Instrument unterzogen.

Resultate: In 23 Studien konnten Rollen und Aufgaben von APNs identifiziert werden. Es handelte sich dabei um sechs Berichte, vier RCTs, drei Querschnittsstudien, drei quasi-experimentelle Studien, einen Essay, eine Sekundäranalyse einer ethnographischen Studie, eine prospektive Kohortenstudie, eine qualitative phänomenologische Studie, einen Fallbericht, eine systematische Übersichtsarbeit und eine Guideline. 21 Artikel beschäftigten sich mit den Aufgaben in der klinischen Praxis der APNs, 16 Artikel beschrieben die Tätigkeiten der APNs in der multidisziplinären Zusammenarbeit und 14 in der beratenden Rolle für Patient*innen. Zehn Artikel stellten die Praxis der APNs als Evidenz-basiert dar und erläuterten deren Aufgabenbereich in der Beratung von Mitarbeiter*innen. In vier Artikeln wurden die APNs in einer Führungsrolle wahrgenommen.

Schlussfolgerungen: Durch dieses Scoping Review konnte festgestellt werden, dass die Aufgaben und Rollen von APNs in der Betreuung chronisch herzkranker Menschen unterschiedlich und vielfältig gestaltet werden können. Für APNs ist es daher wichtig, Definitionen und Abgrenzungen ihres Tätigkeitsbereiches festzulegen, um diesen an ihre eigenen Kompetenzen, sowie die regionalen gesetzlichen Regelungen anzupassen.

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12-2

Comparison of dose values of radiofrequency and cryoablation for pulmonary vein isolation in atrial fibrillation

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Introduction: Atrial fibrillation (AF) ablation is an effective therapy, especially when the arrythmia cannot be controlled with medical treatment. The standard ablation technique is called pulmonary vein isolation (PVI) and is performed using

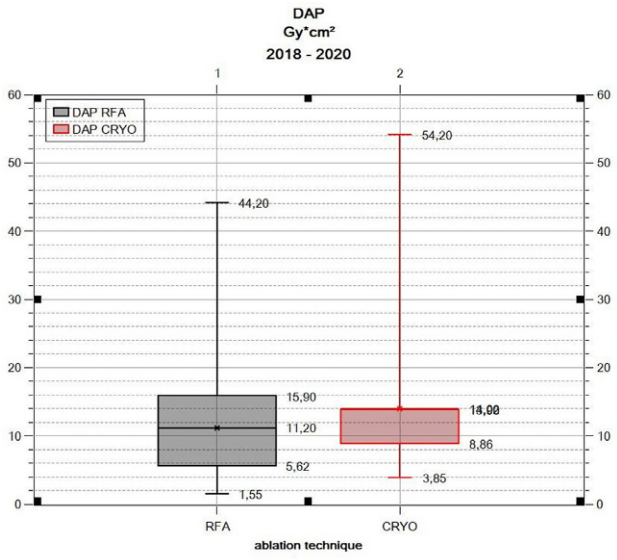

Fig. 1 | 12-2 Comparison of dose area product of RFA vs. Cryo

either radiofrequency (RFA) or cryoablation (Cryo). These two methods differ in terms of their energy source, ablation technique (point-by-point vs. single shot), and their workflow (use of 3D-mapping in RF patients). As a result, there is a difference in the examination duration, fluoroscopy time, and radiation procedure

Methods: Patients undergoing AF ablation between 2018 and 2020 were included in the study. For the retrospective data collection, anonymized patient data were analysed using descriptive statistics and a t-test. The primary endpoints were fluoroscopy time and dose area product.

Results: A total of 100 patients underwent a PVI, 71 patients underwent RFA and 29 patients Cryo. The mean procedure duration for RFA is 164.01 ± 60.09 min with a range of [62.53; 370.50] and for Cryo 123.36 ± 41.25 min [53.58; 230.43]. Fluoroscopy times were shorter using RFA (18.76 ± 7.82 min [7.45; 42.45] vs. 28.45 ± 10.21 min [11.26; 50.57]), while the difference between the dose area product was not significant (RFA: 11.23 [1.55;44.2]; Cryo: 14.02 [3.85; 54.2] Gy*cm^2; $p = 0.08$).

Conclusion: Radiation exposure in this retrospective analysis did not differ between RFA and Cryo ablation. According to the published studies, Cryo ablation has a shorter procedure duration than RFA, but a longer fluoroscopy time and a higher radiation exposure. This highlights the importance of radiation protection measures (low frame rate (f/s), collimation of the image field, monoplane imaging instead of biplane imaging, not using the scatter grid during exposure, etc.), including forward-looking approaches such as US-targeted catheter ablation or High power short duration ablation (HPSD).

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12-3

Interdisziplinäre Zusammenarbeit trotz non-Compliance: Das Ergebnis von außergewöhnlicher interdisziplinärer Teamarbeit über mehrere Fachgebiete der Kardiologie, obwohl der Patient selbst nicht zum Therapieerfolg beiträgt und daher nicht für eine HTX gelistet werden kann

Yamuti S

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Einführung und Patientenvorstellung: Der junge Patient (48 a) wird seit 2012 vom Team der Kardiologie der Klinik Floridsdorf (ehemals Klinik Hietzing) betreut. Es handelt sich um einen adipösen männlichen Patienten, der an einer Herzinsuffizienz leidet. Über die Jahre verschlechterte sich sein Krankheitsbild zunehmend. Er leidet an einer hochgradigen Trikuspidal Klappeninsuffizienz, Mitralinsuffizienz, Adipositas I, Diabetes mellitus II, NYHA Klasse III, LVEF <15 %, rezidivierende Apexthromben, Dyspnoe, chronischem Nierenversagen, ischämischer Hepatitis, VT's und an Hypokaliämie. Der Patient ist nicht HTX-gelistet, da er lange Zeit geraucht hat und abnehmen sollte. Zudem ist er unzuverlässig – er erscheint unregelmäßig zu seinen Terminen.

Therapien: Der Patient erhielt im Laufe der Jahre verschiedenste Therapien: CRT-D, Mitraclips, Barostim (Neurostimulationstherapie für HI und Hypertoniker), mehrmalige VT Ablation und viele stationäre Aufenthalte. Da der Patient CRT Non-Responder ist und seine NYHA Klasse sich auf III verschlechterte, kam der Entschluss dem Patienten einen Barostim im AKH Wien zu implantieren. Ein Barostim ist eine Neurostimulationstherapie. Die Sonde wird an der Bifurkation der Karotis angenäht. Hier befinden sich Barorezeptoren, die durch den IPG und die Sonde stimuliert werden und dadurch ein Gleichgewicht zwischen Sympathikus und Parasympathikus erzeugen. Dies ist der Grund, weswegen die Therapie für Herzinsuffizienz als auch für Patienten, die an Hypertonie leiden wirksam ist. Die Indikation bei Herzinsuffizienz ist NYHA Klasse III und eine LVEF ≤35 % trotz optimaler medikamentöser Therapie. Bei der Hypertonie lautet die Indikation: Drei antihypertensive Medikamente inkl. eines Diuretikums und trotzdem ein systolischer Druck ≥140 mm Hg.

Mitwirkende Abteilungen: Viele Disziplinen und verschiedene Fachgebiete der Kardiologie mussten zusammenarbeiten.

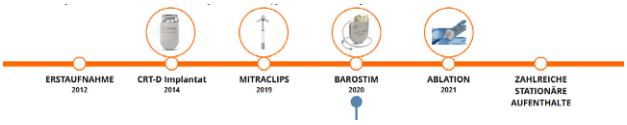

Abb. 1 | 12-3

Abb. 2 | 12-3

Ohne die hervorragende Zusammenarbeit aller mitwirkenden Abteilungen, wäre dieser Therapieerfolg nicht umsetzbar gewesen.

Ausblick: Weitere denkbare Schritte wären ein LVAD-System oder eine bariatrische OP.

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Postersitzung 13 – Rhythmologie 2

13-1

Growth differentiation factor 15 as marker for chronic right ventricular pacing

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Introduction: Growth differentiation factor 15 (GDF 15) is not expressed in the normal adult heart but is up-regulated in cardiomyocytes via multiple stress pathways, and has been associated with mortality in patients with heart failure. While right ventricular (RV) pacing is an important and effective treatment in patients with atrioventricular block it has been shown to promote left ventricular systolic dysfunction. This study aimed to investigate the role of GDF 15 as marker for chronic RV pacing.

Methods: In this single-center prospective cohort study data from 267 consecutive patients (61.8 % male) with single or dual chamber pacemaker and no preexisting heart failure who presented in the outpatient department for routine follow-up was analyzed. Chronic RV pacing was defined as greater than 40 %, as described previously. Serum blood samples were drawn and GDF 15 determined using a commercially available immunoassay (R&D Systems Inc., Minneapolis). Student's t-test was performed to test for group differences and receiver operating characteristics (ROC) to illustrate the diagnostic ability.

Results: Chronic RV pacing was found in 66.7 % of patients. Baseline patients' characteristics are shown in Fig. 3. When separated by stimulation threshold GDF 15 was significantly elevated among patients with >40 % (789 ± 293 pg/ml versus 1186 ± 592 pg/ml; $p < 0.001$), see Fig. 1. ROC revealed GDF 15 as a marker for chronic RV pacing with an area under the curve of 0.713 (95 % confidence interval 0.650–0.776; $p < 0.001$), see Fig. 2.

Conclusion: In this pilot-study GDF 15 was identified as potential marker for chronic RV pacing.

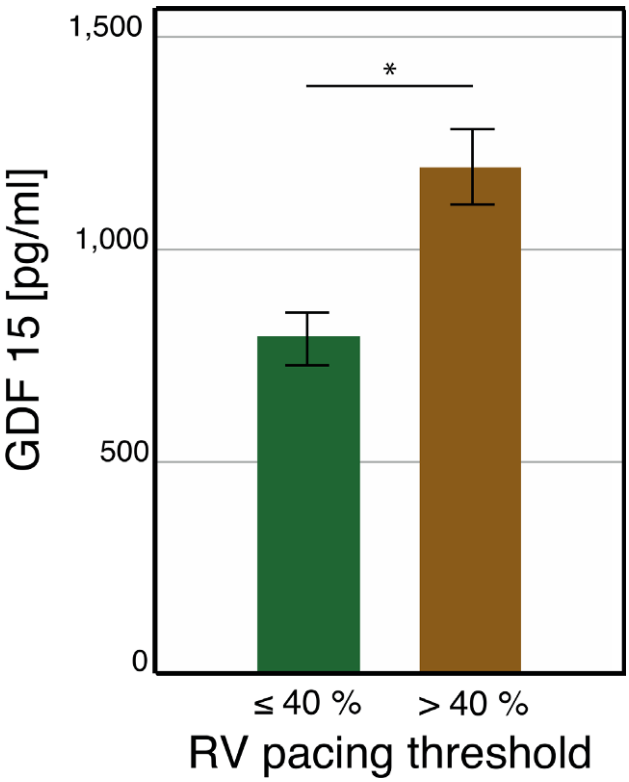

Fig. 1 | 13-1 GDF 15 levels separated by pacing threshold of 40 %

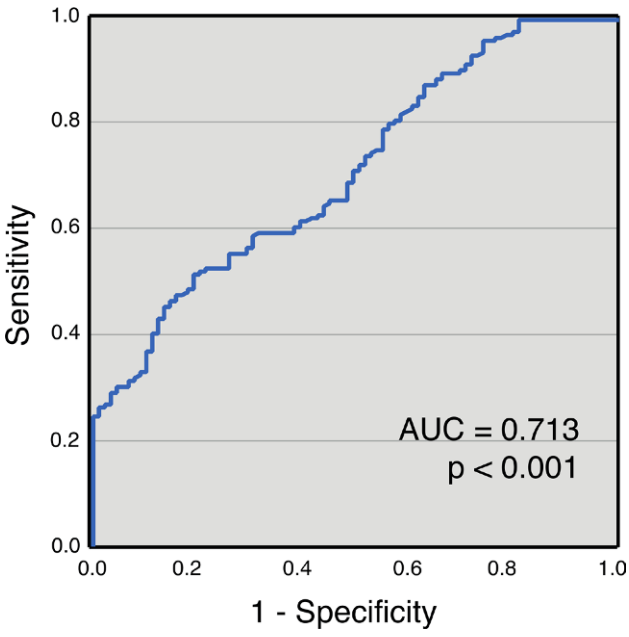

Fig. 2 | 13-2 Receiver operating characteristics for GDF 15 levels and chronic RV pacing

	All
Sex male [%]	61.8
Age [years]	72.1 ± 11.4
Body mass index [kg/m^2]	27.3 ± 4.8
Single chamber pacemaker [%]	19.1
Dual chamber pacemaker [%]	80.9
Pacing threshold [%]	62.5 ± 41.4
Left ventricular ejection fraction [%]	54.3 ± 5.8
Hypertension [%]	79.4
Type II diabetes [%]	20.2
Coronary artery disease [%]	27.0
Chronic kidney disease [%]	22.5
Atrial fibrillation [%]	63.3

Fig. 3 | 13-1 Baseline patients'characteristics

13-2

Optionen zur Behandlung maligner Rhythmusstörungen im Setting der subakuten Koronarischämie – was tun, wenn sonst nichts mehr hilft

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Einleitung: Rückverlegung eines 66-jährigen, intubiert-beatmeten Patienten im elektrischen Sturm wenige Tage nach Drug-eluting Stent (Biotronik Orsiro 3,5/15 mm) Versorgung des proximalen Ramus interventricularis anterior (RIVA) im Setting eines kollateralisierten subakuten Vorderwandinfarkts. Vorangängig extern erfolgte Einleitung einer antiarrhythmischen Therapie mittels Amiodarone und Betablocker sowie Substitution von Kalium und Magnesium in den hochnormalen Bereich, zudem Überstimulation des bradykarden Sinusrhythmus über einen passageren, externen Schrittmacher. Am Übernahmetag erfolgte bei anhaltender elektrischer Instabilität mit Kammerflimmer-bedingter konsekutiver Notwendigkeit der rezidivierenden externen Defibrillation die Entscheidung zur Akut-VT-Ablation.

Methoden: Diesbezüglich wurde unter medikamentöser Kreislaufunterstützung am intubiert-beatmenten Patienten via transseptalem Zugang ein hoch-auflösender Mapping-Katheter (Biosense PentaRay) via steuerbarer Schleuse (SJM Agilis) in den linken Ventrikel vorgebracht. Nunmehr wurde unter Verwendung des Biosense Carto 3-Systems das Purkinje-System abgebildet, wobei wiederholt rasche Purkinje-bedingte polymorphe ventrikuläre Kammertachykardien mit schneller Degeneration in Kammerflimmern auftraten, welche mehrfach durch externe Defibrillation terminiert werden konnten. Therapeutisch erfolgte die Abgabe von Radiofrequenzenergie (Ablationskatheter Biosense QDOT, maximale Energie 50 W) im Bereich des links-posterioren Faszikels proximal midseptal bis distal midseptal sowie im Bereich des mittleren bis distalen links-anterioren Faszikels. Basal, in unmittelbarer Nähe zu zuvor annotierten HIS-Signalen wurde bewusst keine Energie abgegeben. Dies führte zu einem Sistieren der kurz gekoppelten ventrikulären Extrasystolen, sodass in Zusammenschau mit der Abwesenheit von Purkinje-typischen Signalen im Ablationsgebiet entschieden wurde, den Eingriff zu beenden. Postinterventionell traten bereits nach wenigen Stunden unter aufrechter antiarrhythmischer Therapie mit Amiodarone und Ajmalin erneut rasche Kammertachykardien mit wiederholter Notwendigkeit der externen Defibrillation auf, sodass am Folgetag zur Möglichkeit der raschen atrialen Stimulation ein Zweikammer-ICD submuskulär implantiert wurde.

Resultate: Im weiteren Verlauf konnten unter unveränderter antiarrhythmischer Therapie sowie unter Sedations-Fortführung rezidivierend monomorphe nicht-anhaltende Kammertachykardien mit anterolateralem Exit dokumentiert werden. Dies stellte die Indikation zur VT-Re-Ablation. Analog zum Ersteingriff wurde ein hochauflösender Mappingkatheter (Biosense PentRay) in den linken Ventrikel vorgebracht. Im Substratmapping zeigte sich ein ausgedehntes low-voltage Areal (v. a. antero-)septal von basal nach midventrikulär rei-

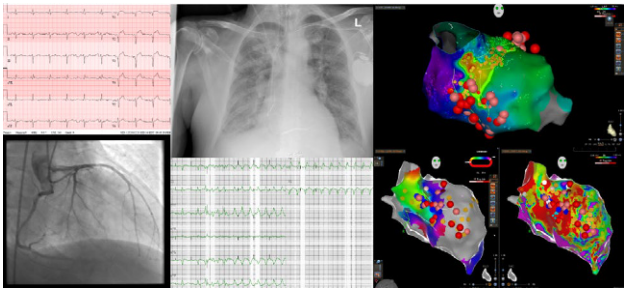

Abb. 1 | 13-2

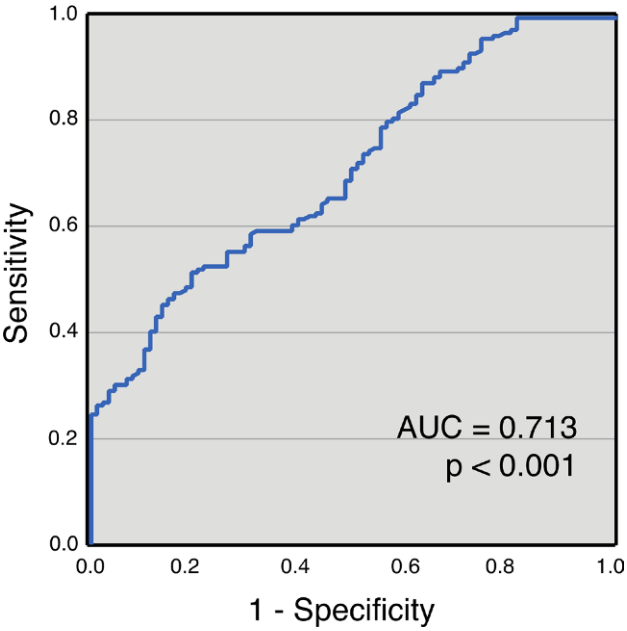

Fig. 2 | 13-2 Receiver operating characteristics for GDF 15 levels and chronic RV pacing

chend. Trotz sensibler Kathetermanipulation konnten hier wiederholt ventrikuläre Runs sowie eine anhaltende Kammertachykardie mit 2 unterschiedlichen Morphologien/Exits, einer davon jener der Monitoraufzeichnungen entsprechend ausgelöst werden, welche im Verlauf spontan terminierte. In besagtem Areal konnte unter laufender Tachykardie der komplette diastolische Pfad abgebildet werden, sodass im Wissen um die örtliche Nähe der zuvor markierten HIS-Region bei darüberhinaus bestehend nahezu identem Pacematch extensiv Radiofrequenzenergie (Biosense QDOT, 50 W) abgegeben wurde. Nach erfolgter Substratablation konnte weder durch Stimulationsmanöver mit unterschiedlichen Zykluslängen und jeweils bis zu 3 kurz-angekoppelten Extraschlägen, noch durch Kathetermanipulation eine Kammertachykardie ausgelöst werden. Der, wenngleich bisher kurze, weitere Verlauf ist verheißungsvoll, sodass bei Ausbleiben ventrikulärer Rhythmusstörungen die Antiarrhythmika sistiert wurden.

Schlussfolgerungen: Wider der lange Zeit bestehenden gängigen Lehrmeinung stellt die Herzkatheterablation auch im Setting der akuten/subakuten Ischämie-bedingten malignen Rhythmusstörungen ein relevantes therapeutisches Mittel dar. Die Zielregion der Herzkatheterablation befindet sich im Falle von Kammerflimmern im Purkinje-Areal. Darüberhinaus konnte gezeigt werden, dass es, wenngleich lediglich für eine geringe Anzahl von Patienten zutreffend, bereits in den ersten Wochen nach einem Infarktgeschehen zur Ausbildung monomorpher Kammertachykardien kommen kann. Unter Rücksichtnahme dieser Erkenntnis stellt sich die Frage, in wie weit die aktuell gültigen Indikationen zur Implantation eines ICD-Devices, insbesondere jene in der Primärprävention nach Myokardinfarkt reevaluiert werden müssen.

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13-3

Impact of socio-economic aspects on cardiac implantable electronic device therapy and application of the EHRA guidelines–A European comparison

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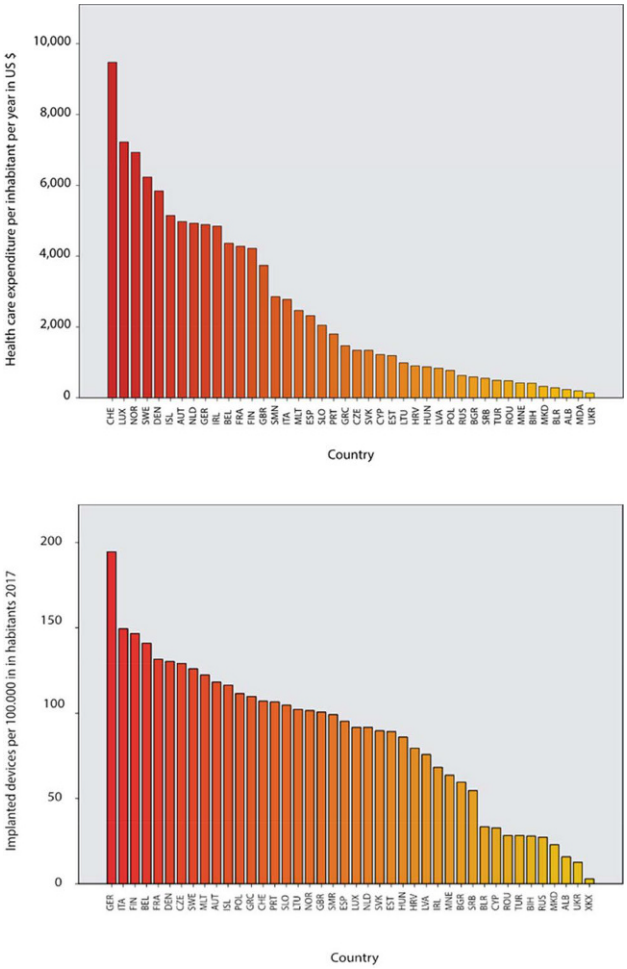

Fig. 1 | 13-3 Health care expenditure vs. CIED implantation rates within the EHRA countries

Introduction: Cardiac implantable electronic devices (CIED) have become an indispensable part in everyday clinical practice in cardiology. The indications of CIED implantations are strongly based on the guidelines of the European Heart Rhythm Association (EHRA). Nevertheless, the numbers of CIED implantations in Europe are subject to considerable differences.We hypothesised that reimbursements linked to the respective health systems may influence implantation behaviour.

Methods: Based on the EHRA White Book 2017, CIED implantation data as well as socio-economic key figures were collected, in particular gross domestic product (GDP) and share of gross domestic product spent on healthcare. Implantation numbers for pacemakers, implantable cardioverter defibrillators and cardiac resynchronization therapy as well as all in total

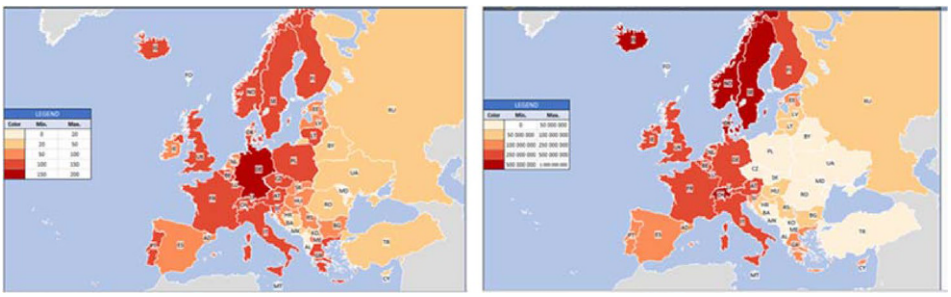

Fig. 2 | 13-3 Heat maps: CIED implantations v.s financial resources per 100,000 inhabitants (in Million Dollars)

were assessed, compared with the health care expenditures and visualized with the heat maps.

Results: Total implantation numbers per 100,000 inhabitants varied immensely from 194.16 (Germany) to 2.81 (Kosovo). Higher implantation numbers correlated moderately with a higher GDP ($r = 0.453$, $p < 0.001$) and higher health expenditures ($r = 0.587$, $p < 0.001$). The annual financial resources per inhabitant were also subject to immense fluctuations ranging from 9476 $ (Switzerland) to 140 $ (Ukraine). However, there were countries with high financial means, such as Switzerland or the Scandinavian countries, which showed significantly lower implantation rates.

Conclusion: The considerable differences seem to be explained on the one hand by the socio-economic disparities within Europe. Nevertheless, there are regions where a potential influence by the respective remuneration system is likely.

13-4

Pulsed field ablation (PFA) for pulmonary vein isolation in patients with atrial fibrillation: A single-center experience

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Introduction: Catheter ablation is a well-established therapy for patients with symptomatic paroxysmal (PAF) and persistent (persAF) atrial fibrillation, but ablation procedures remain time-consuming and have limited success rates, mostly because of non-durable ablation lesions. Pulsed field ablation (PFA) is a promising new ablation technology that has shown to be safe and effective with regards to lesion durability and success rates, and to limit procedure time. Here, we want to share our first experience using PFA for pulmonary vein isolation (PVI) in patients with atrial fibrillation.

Methods: We report our single-center experience with pulsed field ablation therapy for AF by using the Farapulse® system (Boston Scientific), having started to use of this technique in June 2021. In short, a basket catheter is used to apply high energy pulses around the pulmonary vein ostia in a flower and basket configuration (Fig. 1). All patients (44), who were treated with PFA until March 2022 were analysed.

Results: Mean age was 61 ± 11 years, 61 % were male, 39 % were female. 70 % suffered from PAF, 25 % from persAF and 5 % had long standing persistent AF at the time of ablation. Additional cavotricuspid isthmus ablation using conventional linear radiofrequency catheters was performed in 5 % of patients. 91 % had normal LVEF, in 49 % the left atrium was enlarged. Median CHADS-VASc score was 2. First pass isolation of PVs was achieved in 84 %, primary success rate to achieve PV isolation was achieved in all patients. In one person a periprocedural complication (haematoma at the punction site) was described. Furthermore 2 patients with persAF underwent posterior wall isolation in addition to PVI. Median procedure duration was 71 (39–149) minutes, as the procedure was fluoroscopy guided median radiation dose was 13 Gycm2. After 111 ±88 days arrhythmia-free survival was 86 %. In those patients with an AF recurrence, mean time until recurrence was 56 ± 28 days after the procedure.

Conclusion: PFA is a promising, safe and effective catheter ablation technique for AF. Success rates are comparable to standard RF and single shot techniques, but mean procedure duration is short, even when procedural experience is still low.

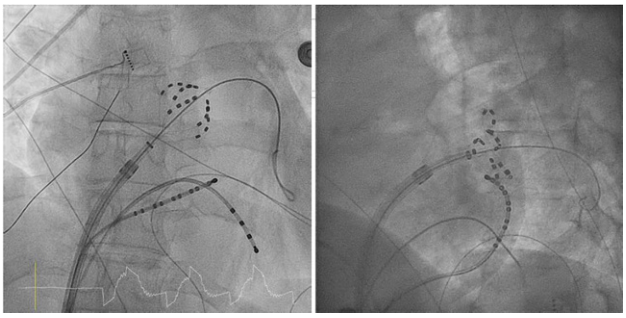

Fig. 1 | 13-4 Basket and flower configuration of Farapulse® ablation catheter

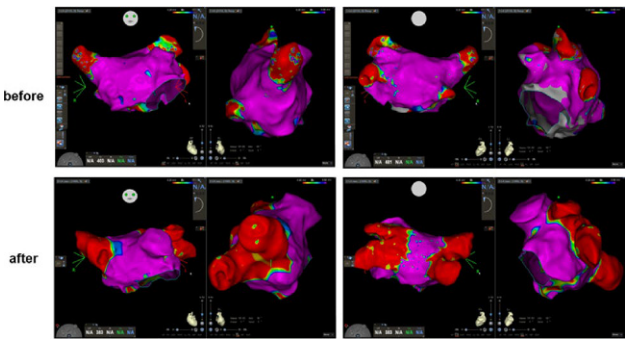

Fig. 2 | 13-4 CARTO® voltage map before and after PFA

13-5

Nahezu fluoroskopiefreie Implantation einer Linksschenkel-Sonde (Left Bundle Branch Pacing)

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Einleitung: Seit wenigen Jahren steht in der kardialen Devicetherapie die Option zur Stimulation des spezifischen Reizleitungssystems zur Verfügung (Conduction System Pacing (CSP): HIS Bündel Stimulation (HBP), Linksschenkelstimulation (LBBP)). Insbesondere bei aufwändigeren Prozeduren können – ähnlich einer CRT-Implantation – lange Fluoroskopiezeiten und eine erhebliche Strahlenbelastung für den Patienten und das implantierende Team entstehen. Wir berichten über die nahezu fluoroskopiefreie Implantation eines Systems zur Linksschenkelstimulation (Left Bundle Branch Pacing) unter Verwendung eines elektroanatomischen 3D-Mapping-Systems (EAMS). Bei der Patientin bestand aufgrund hochsymptomatischen, persistierenden Vorhofflimmerns mit Zn nach Mitralklappenrepair, hochgradig dilatiertem linkem Vorhof und mehrfacher VHF-Rezidive trotz wirksamer Serumspiegel von Amiodaron eine Indikation zur Therapie nach dem pace&ablate Konzept. Guidelines schlagen hierfür die Implantation einer RV-Sonde (Indikation IIa), alternativ entweder ein CRT-System oder HBP (beide IIb) vor [1]. Im aktuellen Fall wurde die Entscheidung zur Linksschenkelstimulation getroffen, da hiermit eine weitgehend physiologische Form der Kammererregung möglich ist, üblicherweise exzellente Reizschwellen vorliegen und die Sonde – im Gegensatz zu HBP – fernab des Koch'schen Dreiecks liegt, sodass im Anschluss eine gefahrlose AV-Knoten-

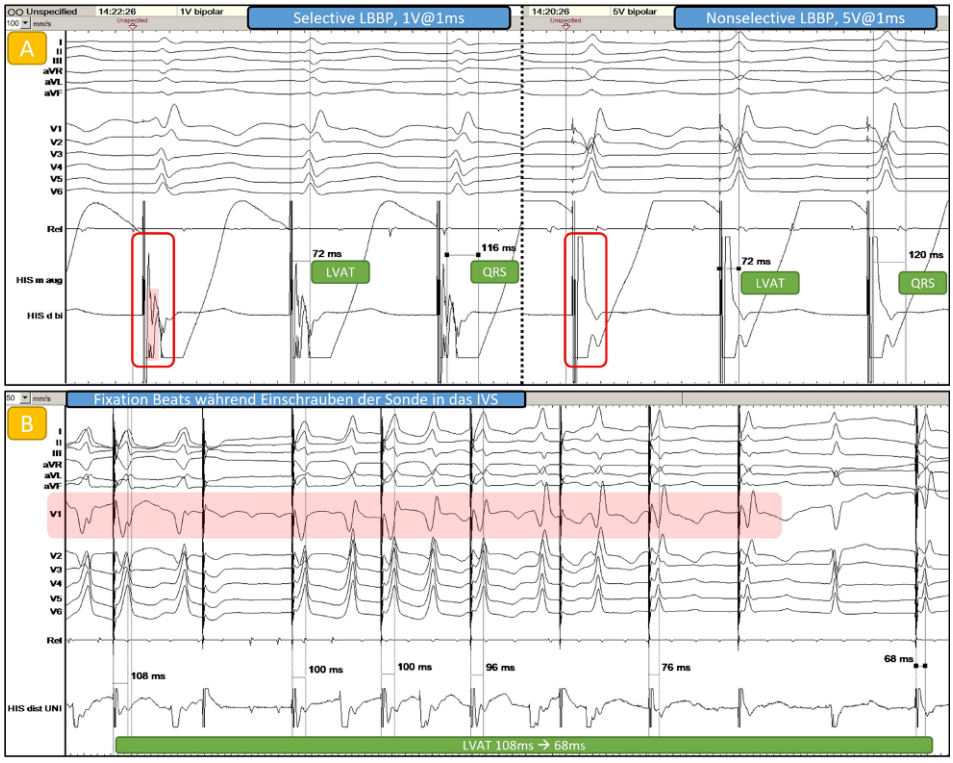

Abb. 1 | 13-5 a Selektives (separates Elektrogramm nach Stimulusartefakt) und nichtselektives LBBP. b Fixation Beats beim Einschrauben der Sonde in das IVS mit progredienter Entwicklung der gewünschten QRS-Morphologie

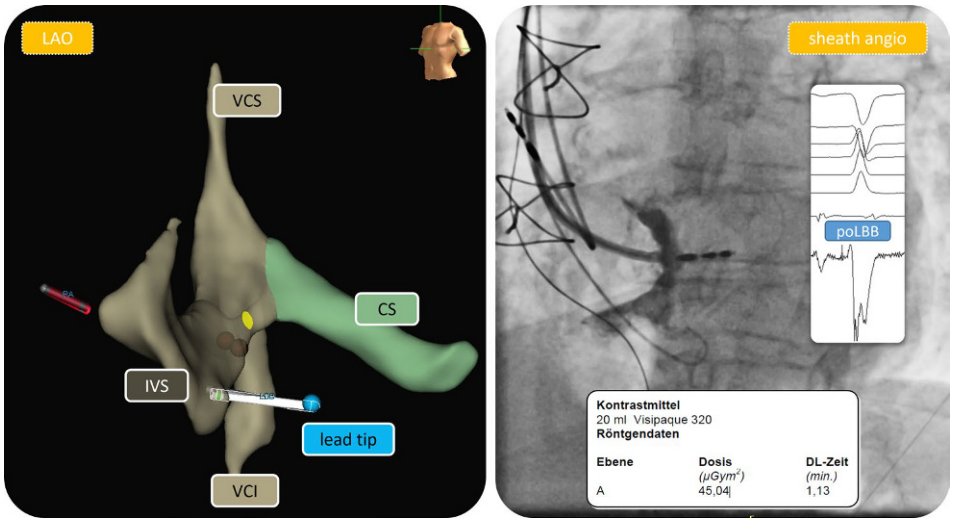

Abb. 2 | 13-5 Elektroanatomisches 3D-Map (links) und Schleusen-Angiographie (rechts), jeweils in LAD-Projektion mit Darstellung der tief im IVS verschraubten LBBP-Sonde; Linksschenkelpotential (poLBB) an der geschraubten Sonde, Fluoroskopie Daten (IVS interventrikuläres Septum, VCS Vena cava sup, VCI Vena cava inf, CS Coronarsinus)

(AVN) Totalablation erfolgen kann und in der Regel keine zusätzliche RV-Backupsonde erforderlich ist.

Methoden: Der operative Eingriff erfolgte standardmäßig in tiefer Sedierung und Lokalanästhesie. Nach Einlegen von Schleusen in die V. cephalica sinistra wurde mittels Standard-EP-Katheter fluoroskopiefrei ein rudimentäres 3D-Map (EnSite NavX, Abbott) der zur Implantation relevanten Strukturen angefertigt (Venae cavae, rechtes Atrium, Klappenebene, Coronarsinus, interventrikuläres Septum (IVS)). Anschließend wurden hierin zur besseren Orientierung Positionen mit typischen HIS-Signalen markiert, dann eine vorgeformte CSP-Schleuse (Selectra 3D, Biotronik) in den RV eingebracht und eine Standard-Schrittmachersonde (Solia S60, Biotronik) nachgeführt. Die elektrisch leitfähige Sonden-Spitze wurde hierbei im EAMS visualisiert und ohne benötigte Röntgenstrahlung manövriert. Nach Heranführen des Equipments an das rechtsseitige, proximale IVS konnte durch unipolare Stimulation eine geeignete Implantationsstelle bestätigt und die Sonde unter Impedanz-Monitoring tief im IVS verankert werden. Die Lage im Septum wurde mittels Schleusen-Angiographie (Kontrastmittelbolus) bestätigt.

Resultate: Während des Einschraubens konnten LBBP-typische EKG-Veränderungen (Entwicklung einer schlanken RSB-Morphologie, Fixation Beats) beobachtet und danach ein typisches Linksschenkel-Potenzial an der Sonde dokumentiert werden. Die linksventrikuläre Aktivierungszeit (LVAT) als Marker einer rapiden LV-Erregung über das Reizleitungssystem wurde von initial 108 ms auf letztlich 68 ms verkürzt (Abb. 1b). Vom maximalen Output weg lag nichtselektives LBBP bis 3 V@1 ms vor, darunter bis zur Reizschwelle von 0,7V@0,4 ms selektive

Linksschenkelstimulation vor (Abb. 1a). Mittels Extraschlag-Stimulation (S1–S2) konnte zusätzlich Capture des spezifischen Reizleitungssystems bestätigt werden. Das Ventrikel-Sensing betrug 8,7 mV, Impedanz 643 Ω; die stimulierte QRS-Breite lag bei 112 ms. Es folgte die Implantation einer Vorhofsonde bei anamnestisch intermittierendem Sinusrhythmus; als Aggregat wurde ein MRT-geeigneter Zweikammerschrittmacher (Enitra 8 DR-T, Biotronik) gewählt. Fluoroskopie wurde zum Einschrauben der Sonde in das IVS bzw der Vorhofsonde in das rechte Herzohr sowie zur Schleusenangiographie und Optimierung der Sondenlängen vor Fixierung der Sleeves benötigt. Die gesamte Fluoroskopiezeit betrug 1,13 min mit einer Strahlendosis von 45,04 µGym^2, die OP-Dauer (Schnitt/Naht) 64 min. Tags darauf wurde komplikationslos die AVN-Totalablation durchgeführt. Abb. 2 veranschaulicht die beschriebene Prozedur.

Schlussfolgerungen: Die Stimulation des Reizleitungssystems (CSP) ist eine moderne und aufstrebende Methode zur physiologischen Herzschrittmachertherapie. Insbesondere LBBP kann aufgrund exzellenter und stabiler Reizschwellen sowie satter Sonden-Verankerung im IVS die Implantation zusätzlicher (Backup-)Sonde vermeiden helfen und bietet bei AVN-Totalablation zusätzliche Sicherheit durch großen Abstand zu kritischen Strukturen. Die Implantation unter Verwendung eines 3D-Mapping-Systems ist einfach, dient in großem Ausmaß dem anatomischen Verständnis der wichtigen Strukturen und vermag die benötigte Strahlendosis auf ein erforderliches Minimum zu reduzieren. In Einzelfällen gelingt hierbei eine nahezu fluoroskopiefreie Prozedur.

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13-6

Durchführbarkeit, Sicherheit und Effektivität durchleuchtungsfreier elektrophysiologischer Untersuchungen – eine Single Center Fall-Kontroll-Pilotstudie

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Einleitung: Durch kontinuierliche Verbesserungen in den letzten Jahren erlaubt die gute örtliche und zeitliche Auflösung der 3D Navigationssysteme die Durchführung strahlungsfreier elektrophysiologischer Untersuchungen („zero fluoro"). Durch den Strahlungsverzicht können stochastische Strahlenschäden bei Patientinnen und Patienten sowie bei dem Personal des EP-Labors verhindert werden. Darüber hinaus bringt die exakte Darstellung der Ablationskatheter in Echtzeit während der Ablation eine erhöhte Sicherheit mit sich. Auch chronische orthopädische Wirbelsäulenprobleme des Personals durch das häufige und lange Tragen von schwerer persönlicher Schutzausrüstung (Bleischürze, Bleihaube, Schilddrüsenschutz, Bleiglasbrille) können auf diese Weise verhindert werden. Ziel dieser Studie war es, die prozeduralen Erfolgs- und Komplikationsraten sowie die Prozedurdauer von sogenannten „zero fluoro" Eingriffen mit denen konventioneller Untersuchungen mit fluoroskopischer Katheternavigation zu vergleichen.

Methoden: Elektrophysiologische Untersuchungen ohne den Einsatz von Strahlung („zero fluoro" Indexprozeduren), die an unserer Abteilung zwischen August 2018 und Februar 2021 durchgeführt wurden, wurden mit historischen Kontrolluntersuchungen mit fluoroskopischer Katheternavigation (Kontrollgruppe) vor dem August 2018 hinsichtlich der prozeduralen Erfolgs- und Komplikationsraten sowie der Prozedurdauer verglichen. Dabei wurden die historischen Kontrolluntersuchungen hinsichtlich der zu behandelnden Rhythmusstörung 1:1 gematcht. Diskrete Variablen wurden als Anzahl mit Prozentsatz angegeben und mittels Chi-Quadrat-Test verglichen, kontinuierliche Variablen wurden als Median mit Interquartilsbreite angegeben und mittels Mann-Whitney-U-Test verglichen. Bei mehr als 40 Indexeingriffen wurde für die jeweilige Prozedur ein lineares Regressionsmodell zur Veranschaulichung einer möglichen Lernkurve für die Verkürzung der Prozedurdauer berechnet. Alle Berechnungen wurden mit der Software Intercooled STATA, Version 14.1, vorgenommen.

Resultate: Im genannten Zeitraum konnten von 146 geplanten „zero fluoro" Indexprozeduren alle bis auf 3 (2,1 %, kurze Durchleuchtung bei Gefäßkinking) ohne Einsatz von Durchleuchtung durchgeführt werden. Die analysierten 143 Untersuchungen setzten sich wie folgt zusammen: 13 (9,1 %) rein diagnostische Untersuchungen, 49 (34,3 %) Isthmus-Ablationen (Abl.), 43 (30,1 %) Slow Pathway Abl., 12 (8,4 %) Abl. rechtsseitiger akzessorischer Leitungsbahnen, 6 (4,2 %) Abl. rechtsatrialer Tachykardien sowie jeweils 10 (7,0 %) Abl. fokaler RVOT und LVOT Extrasystolen. Die Indexpatienten (Indexgruppe IG) unterschieden sich hinsichtlich Alter, Geschlecht und BMI nicht signifikant von der historischen Kontrollgruppe (KG). Die Durchleuchtungszeit in der KG betrug 6,5 [3,8–12,9] min. Das Ablationsziel wurde bei 3 Patienten der Index- und 8 Patienten der KG (jeweils 130 Ablationen) nicht erreicht (2,3 % vs. 6,2 %, $p=0.216$). Bei einem Patienten der IG (Perikarditis) sowie 3 Patienten der KG (Perikarditis, 2 × kompletter AV-Block) kam es zu einer Komplikation (0,7 % vs. 2,1 %, $p=0.477$). Die Prozedurdauer war in der IG um median 18,5 min länger als in der KG (I: 100 [80–120], K: 81,5 [60–115] Minuten, $p<0.001$). Dabei war in der IG im Vergleich der späteren zu den ersten 10 Slow Pathway Ablationen bzw. Isthmusablationen eine Abnahme der Prozedurdauer um im Mittel 7,5 bzw. 8,8 min. ersichtlich. Diese „Lernkurve" war jedoch in einer linearen Regressionsanalyse nicht signifikant ($p=0.522$ bzw. $p=0.242$).

Schlussfolgerungen: In unserer Kohorte an Patienten mit „zero fluoro" EP Prozeduren, die keiner transseptalen Punktion oder röntgenologischen Visualisierung von intrakardialen Devices/Sonden bedurften, konnten wir im Vergleich mit einer historischen Kontrollgruppe keine signifikanten Unterschiede in der Effektivität und Sicherheit der Prozeduren feststellen. Durch die ständige Visualisierung des Ablationskatheters in der Indexgruppe traten aber im Vergleich zur Kontrollgruppe keine kompletten AV-Blockierungen auf. Die Prozedurdauer der „zero fluoro" Untersuchungen war durch die Erstellung der 3D Anatomie im Median um 18,5 min länger. Ein nicht signifikanter Trend eines Lerneffekt konnte nach den ersten 10 Slow Pathway bzw. Isthmus-Ablationen mit einer Verkürzung der Prozedurdauer um ca. 7–9 min festgestellt werden.

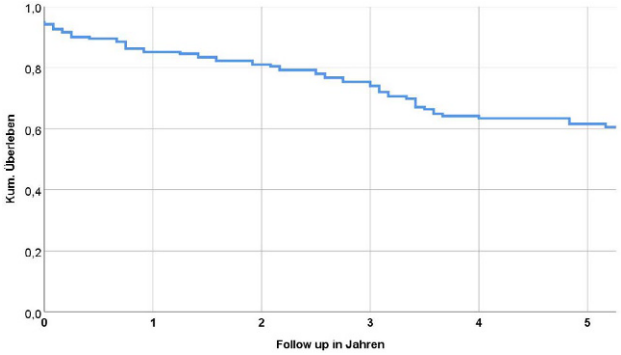

Abb. 1 | 13-7 Überlebensfunktion Gesamtkollektiv ICMP

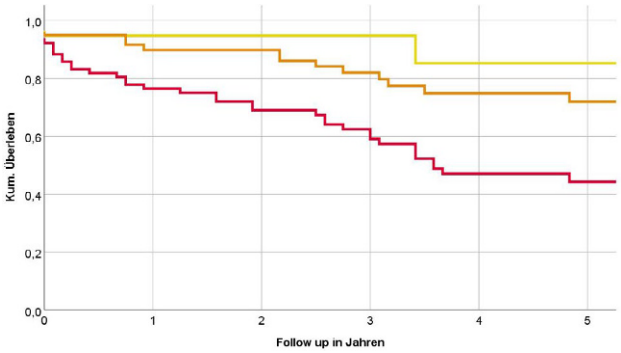

Abb. 2 | 13-7 Überlebensfunktion nach EF

13-7

5-Jahres-Überlebensrate nach VT-Ablation bei Ischämischer Kardiomyopathie

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Einleitung: Die ischämische Kardiomyopathie (ICMP) ist mit dem Auftreten von potenziell lebensbedrohlichen ventrikulären Tachykardien (VT) assoziiert. Zur Behandlung des Rhythmusstörungen spielt neben der Therapie der zugrunde liegenden Erkrankung und der Devicetherapie die Katheterablation eine zunehmende Rolle. Diese steht als komplexe elektrophysiologische Prozedur meist nur in einem spezialisierten Zentrum zur Verfügung. In der Literatur konnte bereits gezeigt werden, dass mit Hilfe einer Katheterablation eine signifikante Reduktion der VT-Episoden erreicht werden konnte. Zusätzlich gibt es Hinweise für eine Mortalitätsreduktion. Welche Parameter in der Nachsorge für die weitere Prognose der Patienten eine Rolle spielen, ist dennoch nicht völlig geklärt.

Methoden: Von Patienten ($n=564$) die zwischen 01/2007 und 03/2021 einer VT-Ablation unterzogen wurden, wurden demographische sowie medizinische Daten mit Hilfe eines Registers erfasst und zusätzlich mit den aktuellen Sterbedaten abgeglichen. Von diesem Register wurden alle Patienten mit ICMP ($n=192$) für diese Analyse herangezogen. Mittels Log Rank-Test und Kaplan-Meier-Kurve erfolgte die statistische Auswertung und Grafikerststellung.

Resultate: Demographisch besteht das Kollektiv der Patienten mit ICMP und VT-Ablation aus vorwiegend männlichen Patienten (88 %). Das Durchschnittsalter beträgt 63,7 (± 11,7) Jahre. 48 % davon hatten eine hochgradig reduzierte, 37 % eine mittelgradig und 12 % eine leichtgradig reduzierte LVEF. Vom Gesamtkollektiv der Patienten mit ICMP sind nach 5 Jahren 38 % verstorben. Wird dieses Kollektiv nach Einschränkung der Linksventrikelfunktion unterteilt, dann versterben in der Gruppe mit schwer eingeschränkter LVEF 56 %, mit mittelgradig reduzierter LVEF 28 % und in der Gruppe mit leicht reduzierter LVEF 15 % nach 5 Jahren. Es zeigte sich, dass die Prognose im Follow-up signifikant ($p=0{,}002$) von der Linksventrikelfunktion beeinflusst wird.

Schlussfolgerungen: Die reduzierte Linksventrikelfunktion ist der Hauptfaktor für eine reduzierte Überlebenswahrscheinlichkeit nach einer VT-Ablation. Eine reduzierte LVEF steigert die Mortalität nicht nur akut, sondern auch im weiteren Verlauf. Dies ist zum einen möglicherweise auf die erhöhte Mortalität hinsichtlich der Herzinsuffizienz oder aber auch auf das gehäufte Auftreten von Rhythmusstörungen zurückzuführen.

Postersitzung 14 – Risikofaktoren/ Stoffwechsel/Lipide 1

14-1

Elevated high-sensitivity C-reactive protein and the risk for cardiovascular events in chronic cardiac disease

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Introduction: High sensitivity C-reactive protein (hs-CRP) is a biomarker used for risk prediction for cardiovascular disease by assessing low concentration of inflammation. Studies have shown that patients with elevated hs-CRP have a higher risk for major adverse cardiovascular events (MACE) [1–3]. The purpose of this study was to assess the event-free time for the composite outcome (acute myocardial infarction, stroke or transient ischemic attack, coronary intervention (including percutaneous coronary intervention and coronary artery bypass graft and death)) between patients of different hs-CRP risk groups and the possible predictive value of hs-CRP for event occurrence in patients with chronic cardiac disease.

Methods: Data from 607 consecutive patients referred for cardiovascular risk assessment with hs-CRP from November 2017 to October 2018 were reviewed retrospectively. Routine peripheral venous blood samples were taken on the day of study inclusion and sent to the local laboratory, where laboratory parameters were analyzed and processed in accordance with local laboratory standards. 570 patients who had hs-CRP measurement by immunoturbidimetric assay were included in the analysis and classified into three (low-, medium- and high-risk) groups (hs-CRP cut-off: <1, 1–3, >3 mg/L). Association between hs-CRP and occurrence of the composite outcome (acute myocardial infarction, stroke, coronary intervention (percutaneous coronary intervention or bypass surgery) or death) was determined with Cox regression analysis and visualized with Kaplan Meier curves. All statistical analyses were performed using R,

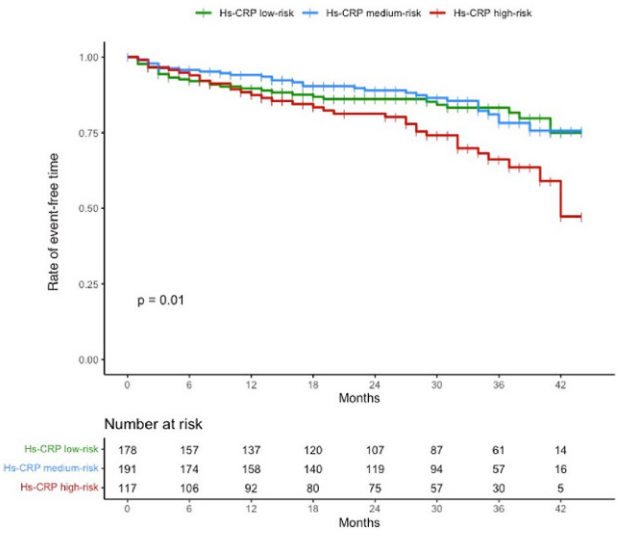

Fig. 1 | 14-1 Kaplan-Meier plot for the composite outcome stratified by hs-CRP risk groups. Log-rank test showed significant difference between high-risk hs-CRP group and low-risk hs-CRP group, as well as between high-risk hs-CRP group and medium-risk hs-CRP group. Number of events in hs-CRP risk groups: low-risk: 29/178 (16.3 %); medium-risk: 30/191 (15.7 %); high-risk: 34/117 (29.1 %). Composite outcome defined as acute myocardial infarction, stroke or transient ischemic attack, coronary intervention (including percutaneous coronary intervention and coronary artery bypass graft) and death

	Univariate		Multivariable	
Variable	**HR, 95% CI**	**P value**	**HR, 95% CI**	**P value**
Age	1.04, 1.02 – 1.06	**<0.001**	1.03, 1.01-1.05	**<0.001**
Sex	1.52, 0.99 – 2.33	0.052	-	
Hs-CRP group (high-risk group as reference)				
Low-risk	0.54, 0.33 – 0.89	**0.015**	0.57, 0.34-0.94	**0.027**
Medium-risk	0.51, 0.31 – 0.84	**0.008**	0.44, 0.27-0.72	**0.001**
BMI	1.01, 0.97 – 1.05	0.656	-	
Hypertension	4.52, 1.66 – 12.32	**0.003**	-	
Diabetes	1.89, 1.21 – 2.94	**0.005**	-	
Hyperlipidemia	2.41, 1.36 – 4.25	**0.002**	-	
Smoking	1.17, 0.72 – 1.89	0.523	-	
Positive family history	1.32, 0.87 – 1.97	0.192	-	
Coronary artery disease	4.80, 2.97 – 7.75	**<0.001**	4.35, 2.69-7.06	**<0.001**
Peripheral artery disease	2.47, 1.35 – 4.54	**0.003**	-	
Cerebrovascular disease	2.42, 1.41 – 4.16	**0.001**	-	
Atrial fibrillation	1.30, 0.85 – 1.97	0.228	-	
History of cancer	0.88, 0.48 – 1.62	0.692	-	
log(NT-proBNP)	1.30, 1.14 – 1.49	**<0.001**	-	
Creatinine	1.41, 1.07 – 1.85	**0.014**	-	

Composite outcome: acute myocardial infarction, stroke or transient ischemic attack, coronary intervention (including percutaneous coronary intervention and coronary artery bypass graft), death.
CI – confidence interval; CRP – C-reactive protein; HR – hazard ratio.

Fig. 2 | 14-1 Univariate and multivariable cox regression analysis

version 4.0.5 (R Foundation for Statistical Computing, Vienna, Austria). A two tailed P-value <0.05 was considered significant.

Results: In total, 570 patients from our cardiology outpatient clinic were included in this study. Follow-up was available for 486 (85.3 %) patients, median follow-up duration was 28 months (maximum 44 months). Cohorts were formed according to hs-CRP risk groups, 209 (36.7 %), 226 (39.6 %) and 135 (23.7 %) patients were classified as low-, medium- and high-risk, respectively. The composite endpoint occurred in 93 (19.1 %) of the 486 patients with available follow-up. Events occurred in 29 (16.3 %), 30 (15.7 %), 34 (29.1 %) patients of the low-, medium- and high-risk group, respectively ($p=0.016$). There was a significant difference in the event-free survival time patients of the low- and medium-risk groups compared with patients in the high-risk group ($p=0.015$). The difference between groups becomes evident after 24 months of follow-up as shown by the Kaplan-Meier curve in Fig. 1. Univariate Cox proportional-hazard analysis identified age, hs-CRP risk group, hypertension, diabetes, hyperlipidemia, coronary artery disease, peripheral artery disease, cerebrovascular disease log(NT-proBNP) and creatinine as significant predictors for the primary study outcome. In multivariable analysis coronary artery disease and age were found to be highly significant predictors for the occurrence of an composite event during follow-up, while patients categorized in the low- and medium-risk groups appeared to predict a lower likelihood for events (Fig. 2).

Conclusion: Cardiovascular events were more likely to occur in patients who were older, with hs-CRP >3 mg/L and a history of coronary artery disease. However, assessment of inflammation markers alone may play a secondary role compared to other established cardiovascular risk factors, elevated CRP appears helpful to detect higher risk and in prediction of further cardiovascular events and mortality.

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14-2

Type 2 diabetes, chronic kidney disease and major cardiovascular events In patients with established cardiovascular disease

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Introduction: Type 2 diabetes (T2 DM) and chronic kidney disease (CKD) both confer a high risk of cardiovascular disease

(CVD), and these conditions frequently coincide. The aim of this study was to investigate the single and joint effects of T2 DM and CKD on major cardiovascular events (MACE) in patients with established cardiovascular disease.

Methods: We prospectively investigated 1738 patients with established cardiovascular disease–angiographically proven coronary artery disease (CAD) or sonographically proven peripheral artery disease (PAD)–over 10.0 ± 4.7 years.

Results: MACE occurred more frequently in T2 DM patients (n = 575) than in non-diabetic subjects (42.5 % vs 29.8 %, p < 0.001) and in patients with CKD (eGFR <60 ml/min/1.73 m2; n = 302) than in those who did not have CKD (52.2 % vs 30.1 %, p < 0.001). When both, T2 DM and CKD were considered, 996 subjects had neither T2 DM nor CKD, 440 had T2 DM but not CKD, 172 did not have diabetes but had CKD, and 130 had both T2 DM and CKD. Compared to the incidence of MACE among patients with neither T2 DM nor CKD (26.5 %), MACE occurred more frequently in patients with T2 DM who did not have CKD (38.2 %; p < 0.001) as well as in non-diabetic patients with CKD (48.0 %; p < 0.001); the incidence of MACE was highest in patients with both, T2 DM and CKD (57.8 %; p < 0.001), in whom it was higher than in those with T2 DM but not CKD (p < 0.001) or those without T2 DM but with CKD (p = 0.007); the incidence of MACE was higher in non-diabetic CKD patients than in T2 DM patients who did not have CKD (p = 0.040). In Cox regression analysis, T2 DM (HR = 1.53 [1.29–1.83]; p < 0.001) and CKD (HR = 1.85 [1.51–2.26]; p < 0.001) proved to be mutually independent predictors of MACE after adjustment for age, sex, BMI, hypertension, history of smoking, LDL-C, HDL-C and HbA1c.

Conclusion: We conclude that T2 DM and CKD in patients with established cardiovascular disease are mutually independent predictors of MACE. Cardiovascular disease patients with both CKD and T2 DM are at an extremely high risk for MACE.

14-3

The A body shape index and Type 2 diabetes are mutually independent predictors of major cardiovascular events In patients with established cardiovascular disease

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Introduction: The A Body Shape index (ABSI) is a validated measure of visceral adiposity that is calculated based on waist circumference, height and BMI. Its power to predict major cardiovascular events in patients with established cardiovascular disease (CVD) is unclear and is addressed in the present study.

Methods: We prospectively recorded cardiovascular events in a large cohort of 1544 patients with established CVD (1297 patients with angiographically proven stable coronary artery disease and 247 patients with sonographically verified PAD) over a mean follow-up time of 10.0 ± 4.6 years.

Results: At baseline, the ABSI was higher in patients with type 2 diabetes (T2 DM; n = 502) than in those who did not have diabetes (8.4 ± 0.6 vs. 8.3 ± 0.6; p < 0.001). Prospectively, the ABSI significantly predicted the incidence of MACE (n = 507) after adjustment for age, gender, smoking, hypertension, LDL cholesterol, HDL cholesterol, and T2 DM (standardized adjusted HRs 1.14 [1.04–1.24]; p = 0.004, respectively). T2 DM in turn in this model also significantly predicted MACE with a HR of 1.61 [1.33–1.94]; p < 0.001 after adjustment for ABSI.

Conclusion: We conclude that ABSI and T2 DM are mutually independent risk factors for MACE in patients with established cardiovascular disease.

14-4

Chronic kidney disease, Type 2 diabetes and the risk of major cardiovascular events in coronary artery disease versus peripheral artery disease patients

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Introduction: Chronic kidney disease (CKD) is a paramount indicator of cardiovascular risk and is highly prevalent in patients with established cardiovascular disease, especially among those with type 2 diabetes (T2 DM). Peripheral artery disease (PAD) confers an even higher risk than coronary artery disease (CAD). How cardiovascular risk compares between PAD and CAD patients when analyses are stratified by the presence of CKD is unclear and is addressed in the present study.

Methods: We prospectively recorded major cardiovascular events (MACE) over 10.0 ± 4.7 years in 1356 patients who had stable CAD, of whom 18.4 % had CKD, and in 382 patients with PAD, of whom 20.9 % had CKD. Four groups were analyzed: CAD patients without CKD (CAD/CKD–; n = 1106), CAD patients with CKD (CAD/CKD+; n = 250), PAD patients without CKD (PAD/CKD–; n = 316) and PAD patients with CKD (PAD/CKD+; n = 66).

Results: The incidence of MACE was lowest in CAD/CKD- patients (27.2 %) and significantly higher in CAD/CKD+ patients (49.6 %; p < 0.001), in PAD/CKD- patients (40.9 %; p < 0.001), and in PAD/CKD+ patients (56.9 %; p < 0.001), who in turn were at a higher risk than CAD/CKD+ or PAD/CKD- patients (p = 0.015 and p < 0.001, respectively). The risk of MACE did not differ significantly between CAD/CKD+ and PAD/CKD- patients (p = 0.063). In Cox regression analysis after multivariate adjustment including gender, age, BMI, hypertension, history of smoking, LDL-C, and HDL-C the presence of PAD versus CAD

(HR = 1.51 [1.25–1.84]; $p < 0.001$), CKD (HR = 1.85 [1.51–2.26]; $p < 0.001$) and T2 DM (HR = 1.53 [1.29–1.83]; $p < 0.001$) were mutually independent predictors of MACE.

Conclusion: We conclude that CKD, T2 DM and the presence of PAD versus CAD are mutually independent predictors of MACE.

14-5

Non-alcoholic fatty liver disease and Type 2 diabetes are mutually independent predictors of major cardiovascular events In patients with established cardiovascular disease

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Introduction: Non-Alcoholic Fatty Liver Disease (NAFLD) is associated with insulin resistance, type 2 diabetes (T2 DM) and cardiovascular disease. However, data on NAFLD in patients with established cardiovascular disease (CVD) are scarce.

Methods: Here, we therefore aimed at investigating the association of NAFLD with T2 DM as well as its impact on the incidence of major cardiovascular events (MACE) in a large series of 1517 patients with established CVD (1199 patients with angiographically proven coronary artery disease and 318 patients with sonographically proven peripheral artery disease), using the validated fatty liver index for the diagnosis of NAFLD.

Results: At baseline, the prevalence of NAFLD was significantly higher in patients with T2 DM than in non-diabetic subjects (61.3 % vs. 39.8 %; $p < 0.001$) respectively. Prospectively, we recorded 498 MACE over a mean follow-up period of 10.0 ± 4.5 years. The risk of MACE was higher in NAFLD patients than in those who did not have NAFLD (49.5 vs. 43.5 %; $p = 0.020$) and in patients with T2 DM than in non-diabetic subjects (41.4 vs. 28.1 %; $p < 0.001$). Cox regression models adjusting for conventional cardiovascular risk factors proved NAFLD and T2 DM to be mutually independent predictors of MACE, with adjusted hazard ratios of 1.34 [1.06–1.69] $p = 0.013$ and 1.59 [1.32–1.92]; $p < 0.001$, respectively.

Conclusion: We conclude that NAFLD and T2 DM are mutually independent predictors of MACE in patients with established CVD.

14-6

Prävalenz und Determinanten von gesundem und vorzeitigem Gefäßalter in Österreich

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Einleitung: Biologisches und chronologisches Alter kann unterschiedlich sein. Von prognostischer Bedeutung ist das Gefäßalter, das eine individuelle Risikoeinschätzung erlaubt.

Methoden: In der österreichischen LEAD (Lung, Heart, Social, Body) Studie, einer longitudinalen populations-basierten Observationsstudie, die stratifiziert nach Alter, Geschlecht und Wohnort eine Stichprobe aus der Bevölkerung von Wien und 6 niederösterreichischen Gemeinden umfasst, wurde im Rahmen der ersten Untersuchungsphase 2011–2016 bei mehr als 10.000 Proband/Innen die Carotis-femoralis Pulswellengeschwindigkeit (cfPWV) mittels Applanations-Tonometrie (SphygmoCor Gerät, AtCor medical) gemessen. In einer definierten Normalpopulation (Nichtraucher, keine kardiovaskuläre Erkrankung, keine Medikamente wegen Hypertonie oder Hyperlipidämie, keine Hyperlipidämie, kein Diabetes, Blutdruck <130/85 mm Hg) wurden altersspezifische Z-Scores der cfPWV errechnet (Abbildung). Healthy Vascular Aging („HVA“) war als <10. Perzentile der cfPWV definiert, Early Vascular Aging („EVA“) als >90. Perzentile, und Normal Vascular Aging („NVA“) als der Bereich dazwischen.

Resultate: Nach Ausschluss invalider Messungen sowie aller Teilnehmer/Innen <18 Jahren wurden 7934 Teilnehmer/innen eingeschlossen. Die cfPWV zeigte einen Anstieg mit zunehmendem Alter. Die Prävalenz von HVA und EVA lag insgesamt bei 7,8 % (HVA) und 24,1 % (EVA). Mit zunehmendem Alter nahm die Prävalenz von HVA ab, und von EVA zu (Tab. 1). Wesentliche Determinanten von EVA sind männliches Geschlecht (OR 0,695 für Frauen), Adipositas (OR 1,335), metabolisches Syndrom (OR 2,357), Hypertonie (OR 2,473), aktives Rauchen (OR 1,937 für 30+ pack years versus alle anderen Kategorien), passives Rauchen (OR 1,293), ungünstigere sozioökonomische Verhältnisse (OR 0,781 für SES Score 3 versus 1, 2), regelmässiger Alkoholkonsum (OR 2,088), Prädiabetes (OR 1,73), Diabetes (OR 2,799), Osteopenie oder Osteoporose (OR

Tab. 1 | 14-6 Prävalenz von healthy (HVA), normal (NVA) und early (EVA) vascular aging in der LEAD Population

	HVA	NVA	EVA
Gesamtpopulation	0.078	0.681	0.241
18–30 Jahre	0.117	0.748	0.135
30–40 Jahre	0.104	0.760	0.136
40–50 Jahre	0.074	0.746	0.180
50–60 jahre	0.068	0.673	0.259
60–70 Jahre	0.051	0.579	0.370
über 70 Jahre	0.034	0.482	0.484

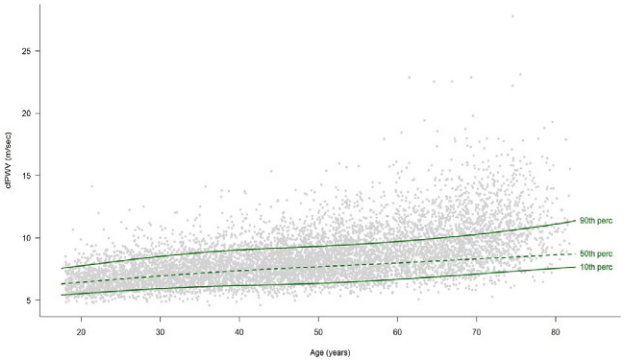

Abb. 1 | 14-6 CfPWV-Perzentilen der definierten Normalpopulation innerhalb der Gesamtpopulation

1,461), schlechtere Nierenfunktion (OR 0,88 pro Anstieg der GFR um 10 ml/min), höheres Gesamt-Cholesterin (OR 1,047 pro Anstieg um 10 mg/dl), erhöhte Entzündungs-Parameter (OR 1,032 pro Anstieg des hsCRP um 1 mg/dl), sowie manifeste Herz-Kreislauferkrankungen (OR 2,208) und Krebserkrankungen (OR 1,435). Alle ORs sind statistisch signifikant mit $p < 0{,}001$.

Schlussfolgerungen: In einer großen österreichischen Populationsstudie findet sich ein hoher Anteil an „early vascular aging". Ungünstiger Lebensstil dürfte einen großen Anteil daran haben.

14-7

Unsaturated ceramides as independent predictor for cardiovascular mortality in diabetic and non-diabetic subjects with coronary artery disease

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Introduction: Recent studies have shown that levels of plasma ceramides are closely related to cardiovascular (CV) mortality. Moreover, there is evidence that ceramides are involved in the pathogenesis of diabetes. Here, we evaluated the predictive value of plasma ceramides for CV mortality in subjects with type 2 diabetes and coronary artery disease (CAD).

Methods: Ceramide levels were measured using ESI-MS/MS in a total of 2583 subjects with proven CAD (924 subjects with Type 2 diabetes, 1659 non-diabetic subjects) previously included in the Ludwigshafen Risk and Cardiovascular Health (LURIC) study. Univariate and multivariate Cox proportional hazard models were used to assess the association with CV mortality.

Results: Using Cox proportional hazard models, CV mortality was markedly higher in diabetic individuals (32 % versus 16 %, $p < 0.05$) compared to non-diabetic subjects after a median follow up of 7.7 years. Adjustment for traditional CV risk factors revealed that plasma levels of unsaturated but not saturated ceramides were independently associated with increased CV mortality in non-diabetic subjects (HR 1.48; 95 %CI 1.12–1.95; $p = 0.006$). In diabetic individuals, this association was even stronger (HR 1.61; 95 %CI 1.26–1.95; p-value <0.001), with the strongest association in diabetic subjects with ceramides in the highest quartile.

Conclusion: Thus, plasma levels of unsaturated ceramides were independently associated with CV mortality in subjects with CAD, and this association was even stronger in diabetic individuals.

Postersitzung 15 – Bildgebung 2

15-1

Bioimpedance spectroscopy reveals important association of fluid status and T1-mapping by cardiovascular magnetic resonance

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Introduction: Extracellular matrix expansion is a key pathophysiologic feature in heart failure and can be quantified non-invasively by cardiac magnetic resonance T1-mapping. Free water within the interstitial space of the myocardium, however, may also alter T1-mapping results. The study aim was to investigate the association between systemic fluid status and T1-mapping by cardiac magnetic resonance.

Methods: 285 consecutive patients (44.4 % female, 70.0 ± 14.9 years old) underwent cardiac MRI due to various cardiac diseases. MR parameters including native myocardial T1-times using MOLLI and extracellular volume (MR-ECV) were assessed, additionally, we performed bioimpedance analysis (BIA). Furthermore, demographic data and comorbidities were assessed. Wilcoxon's rank-sum test, Chi-Square tests, for correlation analysis, Pearson's correlation coefficients were used. Regression analyses were performed to investigate the association between patients' fluid status and T1-mapping. A p-value <0.05 was considered statistically significant.

Results: The mixed cohort presented with a mean overhydration (OH) of +0.2 ± 2.4 liters, as determined by BIS. By MR, native T1-times were 1038 ± 51 ms and MR-ECV was 31 ± 9 %. In the multivariable regression analysis, only OH was significantly associated with MR-ECV (adj.beta 0.711; 95 % CI 0.28–1.14) along with male sex (adj.beta 2.529; 95 % CI 0.51–4.55). In linear as well as multivariable analysis, only OH was significantly associated with native T1 times (adj.beta 3.750; 95 % CI 1.27–6.23).

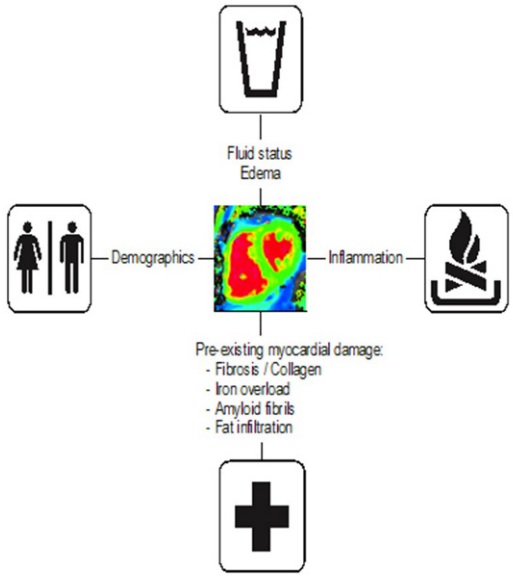

Fig. 1 | 15-1

Conclusion: T1-times and MR-ECV were significantly associated with the degree of overhydration on BIS measurement. These effects were independent from age, sex, body mass index, and hematocrit. Patients' volume status may thus be an important factor when T1-time and MR-ECV values are interpreted.

15-2

Positioning of the image plane in phase contrast CMR impacts aortic stenosis assessment

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Introduction: To determine the phase-contrast cardiovascular magnetic resonance imaging (PC-CMR) level above aortic leaflet attachment-plane (LAP) that generates the most valid measures of flow-velocity and -volume compared to cardiac catheterization in aortic stenosis (AS).

Methods: Fifty-five patients with moderate to severe AS underwent cardiac catheterization, transthoracic echocardiography (TTE) and CMR including cine-imaging and PC-CMR. A total of 171 image-planes parallel to LAP were measured via PC-CMR, at 22 mm below to 24 mm above LAP at end-diastole. Aortic valve area (AVA) via PC-CMR was calculated as flow-volume divided by peak velocity during systole. Stroke volume (SV) and AVA were compared to volumetric SV and invasive AVA via the Gorlin-formula, respectively.

Results: Above LAP, SV by PC-CMR showed no significant differences depending on image-plane position and correlated strongly with volumetry (rho: 0.633, $p < 0.001$, marginal mean difference (MMD): 1 ml, 95 % confidence interval (CI): -4 to 6). AVA assessment in layers from 0–10 mm above LAP differed significantly from invasive measurement (MMD: -0.14 cm², 95 %CI: 0.08–0.21). In contrast, AVA-values by PC-CMR measured 10–20 mm above LAP showed good agreement with invasive determination without significant MMD (0.003 cm², 95 %CI: -0.09 to 0.09). Within these measurements, 15 mm above LAP displayed the lowest bias (MMD: 0.02 cm², 95 %CI: -0.29 to 0.33). SV and AVA via TTE correlated moderately with volumetry (rho: 0.461, $p < 0.001$; bias: 15 ml, $p < 0.001$) and with cardiac catheterization (rho: 0.486, $p < 0.001$, bias: -0.13 cm², $p < 0.001$), respectively.

Conclusion: PC-CMR measurements at 0–10 mm above LAP should be avoided due to significant AVA-overestimation compared to invasive determination. AVA-assessment by PC-CMR between 10–20 mm above LAP did not differ from invasive measurements, with the lowest intermethodical bias measured 15 mm above LAP.

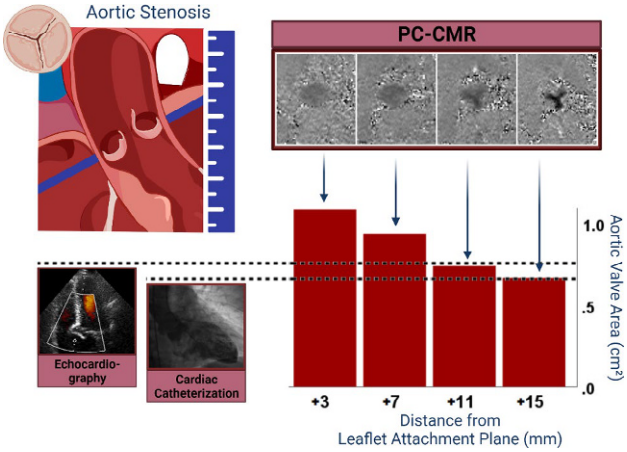

Fig. 1 | 15-2

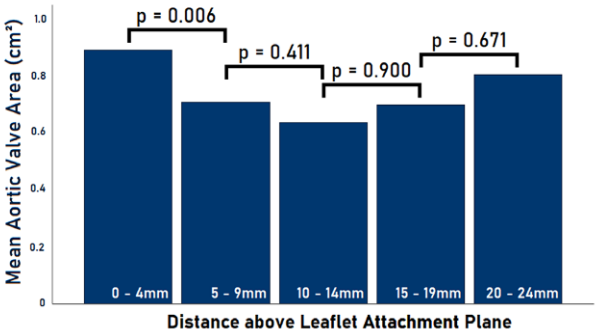

Fig. 2 | 15-2 Bar chart displaying the changes of AVA values via PC-CMR depending on the position of measuring layer

15-3

Right ventricular function and outcome in patients undergoing transcatheter mitral valve repair

Koschutnik M[1], Donà C[1], Nitsche C[1], Dannenberg V[1], Koschatko S[1], Mascherbauer K[1], Beitzke D[2], Loewe C[2], Hülsmann M[1], Schneider M[1], Bartko P[1], Goliasch G[1], Hengstenberg C[1], Kammerlander A[1], Mascherbauer J[1]

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Introduction: The prognostic value of left and right ventricular global longitudinal strain (LV- and RV-GLS) on cardiovascular magnetic resonance feature tracking (CMR-FT) in patients undergoing transcatheter mitral valve repair (TMVR) is unknown.

Methods: Consecutive TMVR patients underwent pre-procedural and follow-up CMR-FT analysis. Kaplan-Meier estimates and multivariable Cox-regression analyses were performed, using a composite endpoint of heart failure hospitalization (HFH) and death.

Results: 62 patients (78.3 ± 7.0y/o, 45 % female, EuroSCORE-II: 9.6 ± 7.1 %) underwent CMR-FT prior to TMVR, 24 % had concomitant tricuspid edge-to-edge repair (TTVR). On presentation, 23 (37 %) patients suffered RV dysfunction (RVD), defined as RV-GLS >–20 % on CMR-FT. RVD was associated with reduced LV and RV ejection fraction (LVEF: 39.2 vs. 48.7 %, $p = 0.008$, RVEF: 35.1 vs. 46.7 %, $p < 0.001$), as well as impaired LV-GLS (–14.0 vs. –19.5 %, $p = 0.012$). Eighteen events (12 deaths, 6 HFH) occurred during follow-up (11.4 ± 9.1 months). On multivariable Cox-regression adjusted for baseline, procedural, imaging, and biomarker data, RV but not LV-GLS was significantly associated with outcome (adj.HR 2.50, 95 % CI: 1.29–4.86, $p = 0.007$ and 1.46, 95 % CI: 0.50–4.28, $p = 0.491$, respectively). Among various definitions of RVD on echocardiography and CMR, only RV-GLS on CMR-FT was significantly associated with outcome (RV-GLS >–20 %: adj.HR 7.53, 95 % CI: 2.07–27.42, $p = 0.002$), but not RVEF on CMR or echo-indices of RV function (Central Illustration). Follow-up CMR-FT was performed in 21 (34 %) patients and RV-GLS significantly improved after TMVR (–20.6 to –25.2 %, $p = 0.016$), irrespective of additional TTVR.

Conclusion: RV-GLS, as determined on CMR-FT, rather than LV-GLS or RVEF, is an independent predictor of outcome in patients undergoing TMVR.

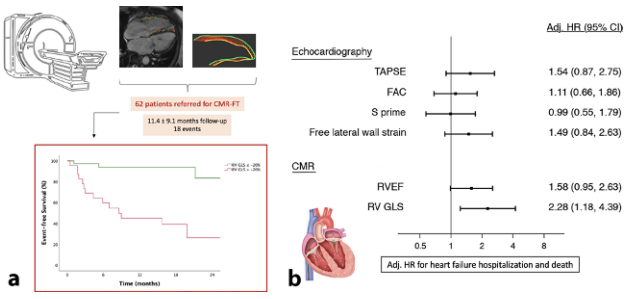

Fig. 1 | 15-3 A Kaplan-Meier estimators demonstrating differences in time to the composite endpoint (heart failure hospitalization and death) stratified for right ventricular global longitudinal strain (RV-GLS) on cardiovascular magnetic resonance (CMR) imaging. B Forest plot demonstrating the association of RV function parameters with the composite endpoint. Hazard ratios (HR) are presented per 1-SD increase for strain measurements and per 1-SD decrease for all other parameters. After adjustment for the EuroSCORE-II and NT-proBNP levels, only RV-GLS on CMR feature tracking (CMR-FT) emerged as strong and independent predictor of outcome

15-4

ST-Strecken Senkungen während einer Fahrrad-Ergometrie durch ein apikales ventrikuläres Divertikel

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Einleitung: ST-Strecken Senkungen während einer Fahrrad Ergometrie sind meistens ein Hinweis für belastungsinduzierte Ischämien. Jedoch können auch ventrikuläre Divertikel in der Ergometrie zu ST-Strecken Senkungen führen wie diese Fallvorstellung veranschaulichen soll.

Methoden: In der kardiologischen Ambulanz wurde ein 49 Jahre alter männlicher Patient mit einem pathologischen Ergometrie-Befund ohne bekannte kardiovaskuläre Erkrankungen vorstellig. Neben einem Nikotinkonsum war eine positive Familienanamnese bezüglich einer koronaren Herzerkrankung dokumentierbar. In der nach dem WHO-Protokoll durchgeführten Ergometrie zeigte sich ab einer Belastung von 120 W ST-Streckensenkungen in den Brustwandableitungen V4–6. Die Ergometrie konnte bis 200 W asymptomatisch fortgesetzt werden. In der Echokardiographie zeigte sich eine normale linksventrikuläre Funktion ohne regionale Wandbewegungsstörungen. Zur Abklärung der belastungsabhängigen ST-Streckensekungen wurde eine Coronarangiographie vereinbart.

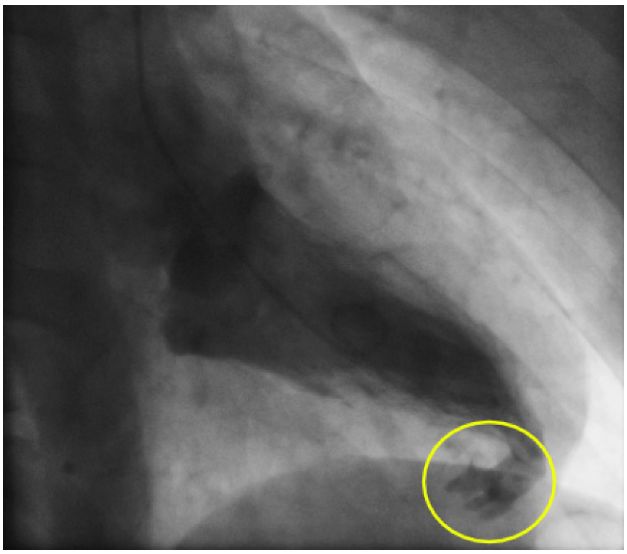

Abb. 1 | 15-4 Levocardiographie, apikoseptales Aneurysma

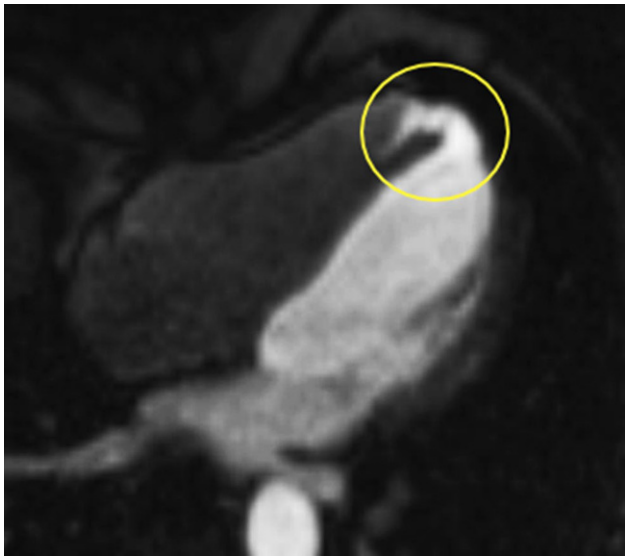

Abb. 2 | 15-4 Cardiales MRT, inferoseptales Divertikel

Resultate: In der Koronarangiographie zeigte sich ein glattwandiger Hauptstamm, der sich in einen unauffälligen Ramus Circumflexus und eine unauffällige LAD aufteilte. Die rechte Kranzarterie wies keine Auffälligkeiten auf. In der Lävocardiographie zeigte sich eine gute Linksventrikelfunktion ohne Wandbewegungsstörungen jedoch ein Hinweis auf ein gestieltes trabikuliertes apikoseptales Aneurysma von ca 20 × 15 mm. Zur weiteren Abklärung wurde ein kardiales MRT durchgeführt. Dabei zeigte sich apikal inferoseptal ein Divertikel des linken Venrikels mit einem Diameter von 12 × 17 mm. Die Divertikelwand imponierte deutlich verdünnt. Der normalgroße rechte Ventrikel hatte durch ein ausgeprägtes Pectus excavatum eine Einschnürung, wodurch die rechtsventrikuläre Ejektionsfraktion reduziert war und eine Dyskinesie bestand. Ein Thrombus oder ein Links-Rechtsshunt konnten nicht nachgewiesen werden. Da der Patient beschwerdefrei war, wurde eine jährliche echocardiographische Kontrolle sowie die Durchführung eines Holters alle 2–3 Jahre sowie sofort bei Paplpitationen vereinbart. Damit sollen ggf. entstehende Thromben frühzeitig erkannt werden und Rhythmusstörungen aus diesem Areal diagnostiziert werden und gegebenenfalls mittels Katheterablation therapiert werden können.

Schlussfolgerungen: Durch die immer höhere Qualität und breitere Verfügbarkeit von kardialen MRTs können kongenitale ventrikuläre Aneurysmen und Divertikeln häufiger diagnostiziert werden. Eine einheitliche Nomenklatur liegt nicht vor und hängt stark von dem befundenden Zentrum ab. Desweiteren gibt es wegen der geringen Fallzahl keine evidenzbasierte Therapievorschläge. Daher empfehlen wir die Einführung einer einheitlichen Nomenklatur sowie die Etablierung eines europaweiten Registers.

15-5

Infarct severity and outcomes in STEMI patients without standard modifiable risk factors: A multicenter cardiac magnetic resonance imaging study

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Introduction: Standard modifiable cardiovascular risk factors (SMuRFs) are well-established players in the pathogenesis of acute ST-elevation myocardial infarction (STEMI). However, in a significant proportion of patients with STEMI no SMuRFs can be identified and the outcomes of this subgroup are not well described. The purpose of this study was to assess the infarct characteristics at myocardial tissue level and subsequent clinical outcomes in SMuRF-less STEMI patients.

Methods: This multicenter, individual patient-data analysis included 2012 STEMI patients from four large magnetic resonance imaging (MRI) trials conducted in Austria, Germany, Scotland, and the Netherlands. SMuRF-less was defined as absence of hypertension, smoking, hypercholesterolemia, and diabetes. All patients underwent cardiac MRI at 3 (interquartile range [IQR]: 2–4) days after infarction to assess left ventricular (LV) volumes and ejection fraction, infarct size and microvascular obstruction (MVO). Clinical endpoints of interest were the occurrence of major adverse cardiovascular events (MACE), defined as composite of all-cause mortality, re-infarction and heart failure.

Results: No SMuRF was identified in 185 patients (9 %). These SMuRF-less patients did not show significant differences in LV ejection fraction (51 [IQR: 45–57] versus 50 [IQR: 43–57]%, $p = 0.124$), infarct size (17 [IQR: 9–25] versus 16 [IQR: 8–25]%, $p = 0.532$), and rates (48 versus 53 %, $p = 0.190$) as well as extent of MVO (0.0 [IQR: 0.0–1.8] versus 0.3 [IQR: 0.0–2.5]%, $p = 0.113$) compared to patients with SMuRFs. During a median follow-up time of 12 (IQR: 12–27) months, 199 patients (10 %) experienced a MACE event. No significant difference in MACE rates was observed between SMuRF-less patients and patients with SMuRF (8 versus 10 %, $p = 0.386$).

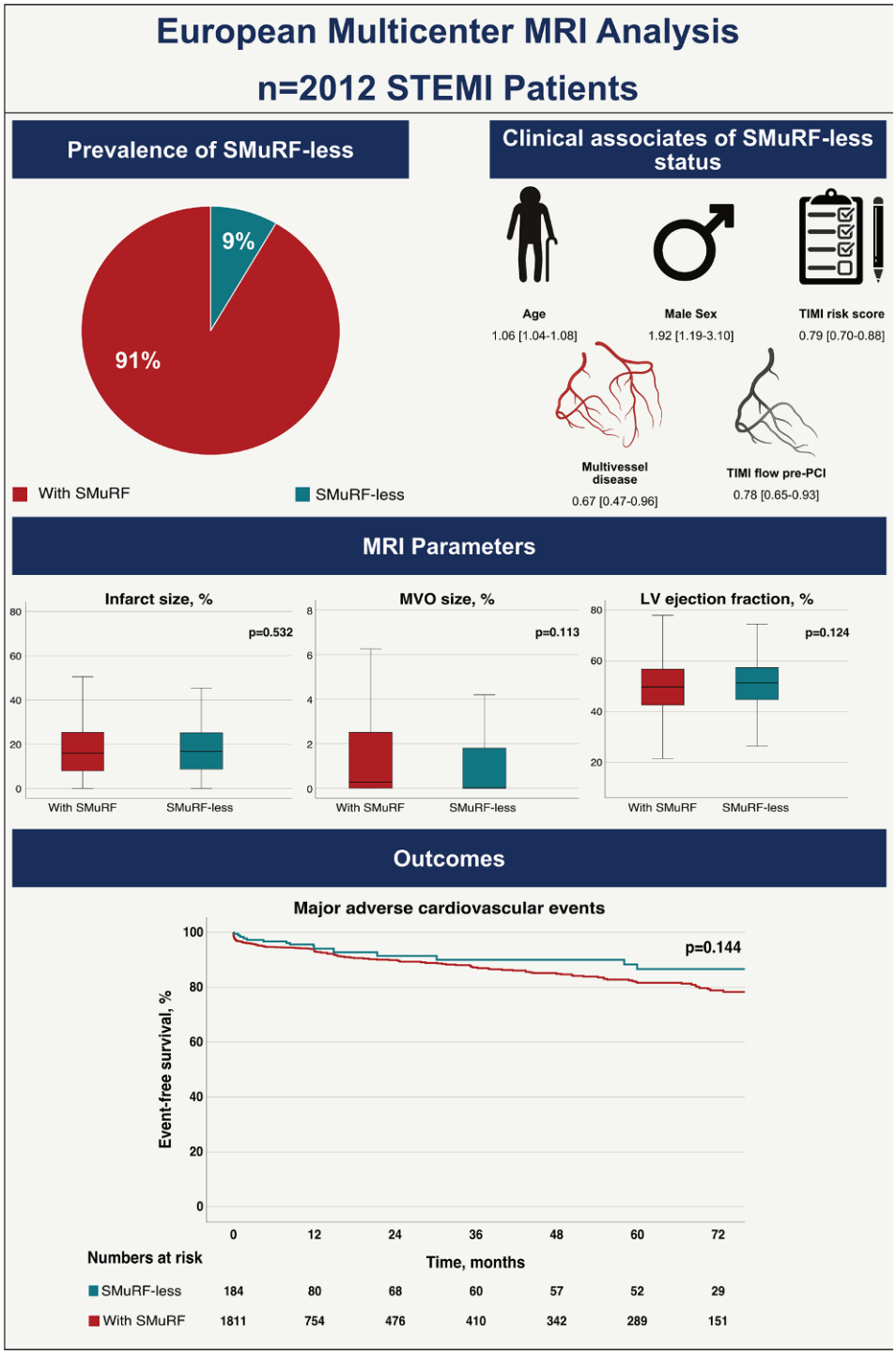
European Multicenter MRI Analysis
n=2012 STEMI Patients
Prevalence of SMuRF-less
9%
91%
With SMuRF
SMuRF-less
Clinical associates of SMuRF-less status
Age
1.06 [1.04-1.08]
Male Sex
1.92 [1.19-3.10]
TIMI risk score
0.79 [0.70-0.88]
Multivessel disease
0.67 [0.47-0.96]
TIMI flow pre-PCI
0.78 [0.65-0.93]
MRI Parameters
Infarct size, %
p=0.532
MVO size, %
p=0.113
LV ejection fraction, %
p=0.124
With SMuRF
SMuRF-less
Outcomes
Major adverse cardiovascular events
p=0.144
Event-free survival, %
Time, months
Numbers at risk
SMuRF-less 184 80 68 60 57 52 29
With SMuRF 1811 754 476 410 342 289 151

Fig. 1 | 15-5

Conclusion: In this large individual patient-data pooled analysis, SMuRF-less status was observed in 9 % of STEMI patients and was not associated with MRI infarct characteristics and subsequent MACE.

Postersitzung 16 – Vitien

16-1

Supervised learning-derived tailored risk-stratification in patients with severe secondary mitral regurgitation

Heitzinger G[1], Spinka G[1], Prausmüller S[1], Pavo N[1], Dannenberg V[1], Donà C[1], Koschutnik M[1], Kammerlander A[1], Nitsche C[1], Arfsten H[1], Kastl S[1], Strunk G[2], Hülsmann M[1], Rosenhek R[1], Hengstenberg C[1], Bartko P[1], Goliasch G[1]

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Introduction: Mitral regurgitation secondary to heart failure (sMR) has considerable impact on quality of life, heart failure (HF) rehospitalizations and mortality. A diverse burden of comorbidities suggests multifaceted aspects of individual risks. This risk-spectrum has never been studied but is essential to understand disease trajectories. The objective was to provide a comprehensive and structured decision-tree-like approach to risk-stratification in patients with severe sMR.

Methods: This large-scale, long-term observational study included 1317 patients with severe sMR from the entire HF spectrum (preserved, mid-range and reduced ejection fraction). Primary endpoint was all-cause mortality and survival tree analysis, a supervised learning technique, was applied to identify patient subgroups with excessive risk of mortality.

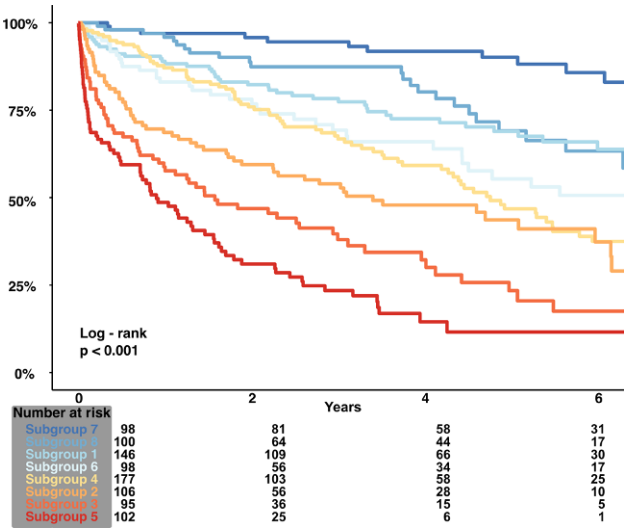

Fig. 2 | 16-1 Overall Kaplan Meier Analysis (derivation cohort). Kaplan Meier curves for each subgroup and according numbers of patients at risk. Subgroups are color-coded. Subgroup 7 (dark blue) displaying the lowest risk of mortality and subgroup 5 (dark red) depicting excessive risk of death

Results: Eight distinct subgroups that differed significantly in long-term survival were identified. Subgroup 7, characterized by younger age (≤66), higher hemoglobin (>12.7 g/dl) and higher albumin levels (>40.6 g/l) had the best survival. In contrast, subgroup 5 displayed a 20-fold risk of mortality (HR 95 %CI: 20.38 (10.78–38.52), $P < 0.001$) and presented with older age (>68 years) and low serum albumin (≤40.6 g/l) and higher NT-proBNP levels (≥9750 pg/ml). Results were consistent in internal and temporal validation.

Conclusion: Supervised machine learning reveals an unexpected heterogeneity in the sMR risk-spectrum, indicating the clinical challenges tied to severe sMR. A decision-tree-like

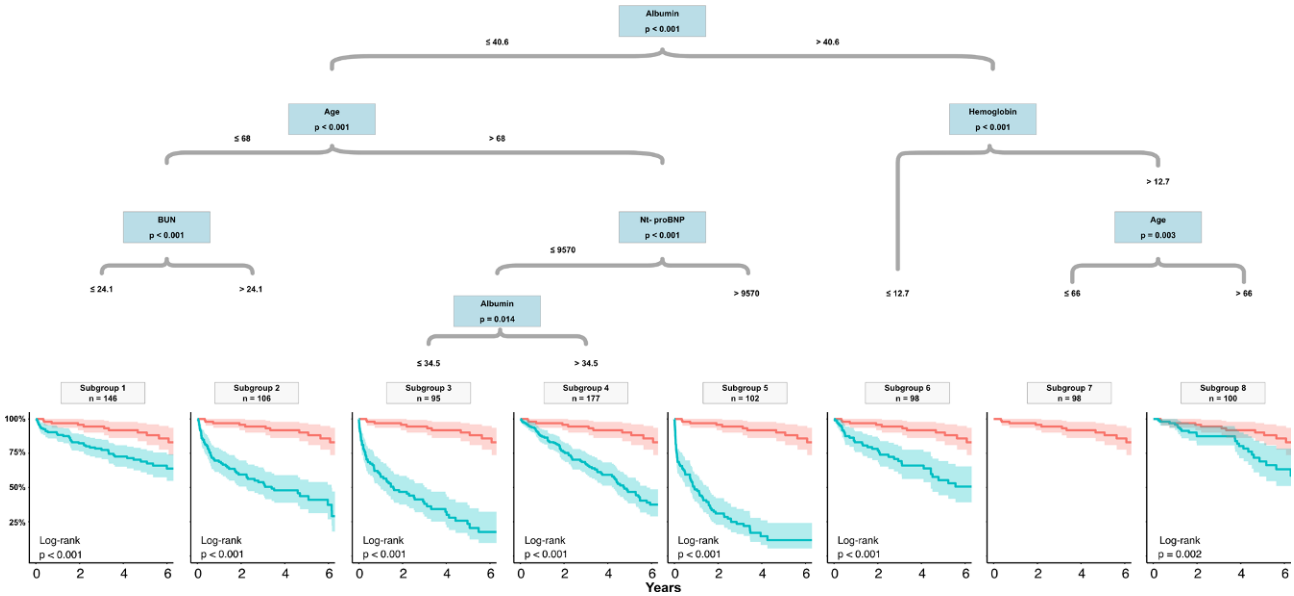

Fig. 1 | 16-1 Survival tree for all patients with severe sMR (derivation cohort). Subgroups of patient with severe sMR and heart failure identified by survival tree analysis. Predictors, cutoffs and the according Kaplan-Meier curve are depicted in tree nodes and final leaves. Subgroup 7 had the most favorable survival and served as the reference group (red color)

model can guide through the risk spectrum and provide tailored risk-stratification. This structured approach provides the foundation to generate hypotheses towards improved therapeutic strategies and optimized patient care.

16-2

Tricuspid regurgitation in cancer patients–A retrospective outcome analysis

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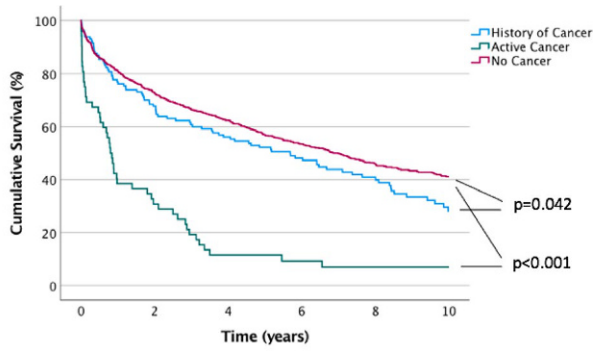
Introduction: Tricuspid regurgitation (TR) is a common condition associated with increased rates of hospitalization and death. It is known that TR may occur in oncologic patients as a consequence of chemotherapy or radiotherapy. Nevertheless, the prognostic impact of TR in oncologic patients is scarcely studied. The aim of this study is to investigate the survival rates of TR patients with different cancer types and status. The results will help to assess the prognosis before interventional or surgical tricuspid valve repair.

Methods: We included all patients diagnosed with at least moderate-to-severe TR at the Medical University of Vienna between 2003 and 2016 with normal left ventricular function and no other valvular lesions. Outcome analysis were performed according to cancer type, cancer history, and cancer status at last follow-up.

Results: A total of 973 patients were included, 182 patients had cancer, 52 active and 130 history of cancer according to the last records. Cancer patients were divided into subgroups of gastrointestinal, skin, glands, gynecological, breast, urogenital, lung and other cancer. Kaplan-Meier curves were calculated, and Log-rank tests performed. 10 years mortality of patients with cancer were higher than mortality of patients without cancer ($p < 0.001$). Mortality was borderline significantly higher in patients with a history of cancer compared with patients without cancer ($p = 0.042$).



No. at risk

History of Cancer	129	87	71	57	39	20
Active Cancer	51	15	5	3	0	0
No Cancer	790	574	491	381	276	153

Fig. 1 | 16-2

Table 1 | 16-2

Tumor subgroups and entities	Patients with cancer, $n = 182$ (100)
Gastrointestinal	31 (17)
Colon	11 (6)
Stomach	3 (2)
Rectum	7 (4)
Esophagus	8 (4)
Other	7 (4)
Skin	13 (7)
Melanoma	7 (4)
Other	5 (3)
Glands	38 (21)
Pankreas	1 (1)
Liver	3 (2)
Thyroid	8 (4)
Prostate	18 (10)
Other	8 (4)
Gynecological	15 (8)
Uterus	11 (6)
Cervix	1 (1)
Ovar	3 (2)
Breast	40 (22)
Urogenital	15 (8)
Kidney	10 (5)
Bladder	5 (3)
Lung	7 (4)
Pleura	1 (1)
Bronchus	4 (2)
Lung	2 (1)
Other	23 (13)
Brain	1 (1)
Blood	9 (5)
Lymph	7 (4)
Muscle	2 (1)
Bone	2 (1)
All patients	973 (100)
No cancer	791 (81)
Active cancer	52 (5)
History of cancer	130 (13)
All values are numbers (%)	

Conclusion: Mortality in patients with TR is very high and increased by active cancer or a history of cancer.

16-3

Beating heart mitral valve repair using the Edwards HARPOON system: A case report of the first implantation in Austria

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Introduction: Open-heart surgery is the gold-standard treatment for degenerative mitral regurgitation. However, minimal-invasive procedures are advancing rapidly as we progress into modern medicine. The HARPOON system by Edwards Lifesciences is a beating heart, off-pump, mitral valve repair technology that has been developed to treat posterior leaflet prolapse in degenerative mitral valve disease. The HARPOON system provides expanded polytetrafluoroethylene (ePTFE) neochords through a minimally invasive chest incision. The neochords are deployed by a hand-held device and anchored to the posterior mitral leaflet using self-forming knots. This echo-guided procedure allows real-time chordal adjustment (positioning, titration of the length of the artificial chords) favoring optimal leaflet coaptation and reduction of mitral regurgitation.Hereby we present the case of an 85-year-old male who was the first patient to undergo mitral valve repair using the HARPOON system in Austria.

Methods: n. a.**:**

Results: The patient was previously admitted to hospital with dyspnoea NYHA III due to severe mitral valve regurgitation caused by a prolapse with additional flail leaflet of the posterior leaflet segment P2. The patient was judged eligible by the local Heart Team. The decision was mainly based on the patient's comorbidities and mitral valve morphology (favorable posterior leaflet geometry and a tissue-to-gap ratio >2). On the day of the procedure, the 9 French shaft of the single-use device was introduced in the left ventricle approximately 2 cm basal from the true apex at the level of the papillary muscles. The tip of the device was advanced to the target area using transesophageal echo guidance to avoid entanglement with edge chords. After landing underneath the posterior leaflet, the ePTFE suture knots were deployed as close to the free edge of the posterior leaflet as possible. In total, 4 suture knots with a knot-to-knot mean distance of 3 mm were deployed. The 4 neochords were tensioned and apically attached to allow appropriate leaflet mobility with chordal relaxation during diastole. The postoperative course was unremarkable. The patient was discharged from the intensive care unit on the following day and was finally released from hospital one week after the procedure. Thirty-days echo follow-up revealed no residual mitral regurgitation with intact neochords.

Conclusion: The Harpoon mitral repair system is a novel, feasible technique to treat posterior mitral leaflet prolapse.

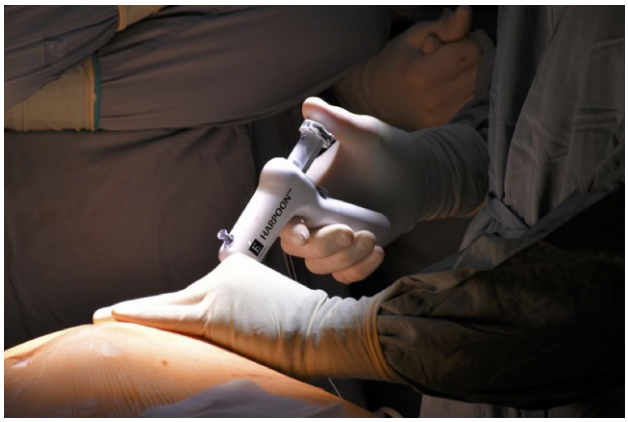

Fig. 1 | 16-3 The hand-held HARPOON mitral valve repair device

16-4

Effects of guideline directed medical therapy on secondary mitral regurgitation–Implications for compound sequencing

Spinka G[1], Bartko PE[1], Heitzinger G[1], Prausmüller S[1], Winter M[1], Arfsten H[1], Strunk G[2], Rosenhek R[1], Kastl S[1], Hengstenberg C[1], Pavo N[1], Hülsmann M[1], Goliasch G[1]

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Introduction: Guideline directed medical therapy (GDMT) is the recommended initial treatment for secondary mitral regurgitation (SMR), however supported by only little comprehensive evidence. This study therefore sought to assess the effect of GDMT titration on SMR and to identify specific substance combinations able to reduce SMR severity.

Methods: We included 261 patients who completed two visits with an echocardiographic exam available within one month at each visit. After comprehensively defining GDMT titration as well as SMR reduction, logistic regression analysis was applied in order to assess the effects of overall GDMT titration and specific substance combinations on SMR severity.

Results: SMR severity improved by at least one degree in 39.3 % of patients with subsequent titration of GDMT and was accompanied by reverse remodelling and clinical improvement.

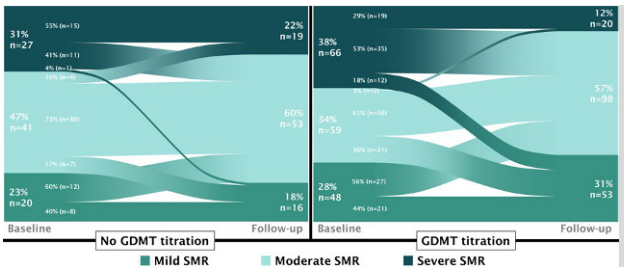

Fig. 1 | 16-4 Sankey diagram displaying the longitudinal evolution of SMR severity from baseline to follow-up according to GDMT titration group

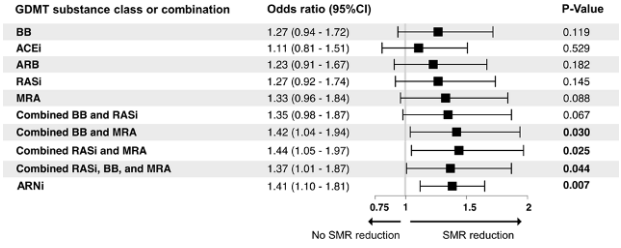

Fig. 2 | 16-4 Univariable logistic regression analysis assessing the impact of GDMT substance classes and combinations on the reduction of SMR displayed as forest plot

The effects of GDMT titration were significantly associated with SMR reduction (adj. OR 0.31, 95 %CI 0.13–0.71, $P = 0.006$). Moreover, ARNI as well as the combined dosage effects of (i) renin angiotensin system inhibitors (RASi) and mineralocorticoid-receptor antagonists (MRA), (ii) betablockers (BB) and MRA, as well as (iii) RASi, BB, and MRA were all significantly associated with SMR improvement ($P < 0.001$ for all).

Conclusion: The present study provides comprehensive evidence for the effectiveness of contemporary GDMT to specifically improve SMR. Our data indicates that GDMT titration conveys a threefold increased chance of reducing SMR severity. Moreover, the dosage effects of ARNI, as well as the combination of RASi and MRA, BB and MRA, and all three substances in aggregate are able to significantly improve SMR.

16-5

Prognostic impact of high-molecular-weight von Willebrand Factor multimer ratio in classical low-flow low-gradient aortic stenosis

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Introduction: High-molecular-weight (HMW) von Willebrand Factor (VWF) multimer deficiency occurs in classical low-flow, low-gradient (LF/LG) aortic stenosis (AS) due to shear force induced proteolysis. The prognostic value of HMW VWF multimer deficiency is unknown. Therefore, we sought to evaluate its impact on clinical outcome.

Methods: In this prospective research study, a total of 83 patients with classical LF/LG AS were included. All patients underwent dobutamine-stress-echocardiography to distinguish true-severe (TS) from pseudo-severe (PS) classical LF/LG AS. HMW VWF multimer ratio was calculated using densitometric Western blot band quantification. The primary endpoint was all-cause mortality.

Results: Mean age was 79 ± 9 years and TS classical LF/LG AS was diagnosed in 73 % ($n = 61$) and PS classical LF/LG AS in 27 % ($n = 22$) of all patients. Forty-six patients underwent aortic valve replacement (AVR) and 37 were treated conservatively. During a mean follow-up of 27 ± 17 months, 47 deaths occurred. Major bleeding complications after AVR (10/46; 22 %) were more common in patients with HMW VWF multimer ratio <1 (8/17; 47 %) in comparison to patients with a normal multimer pattern (2/29; 7 %) at baseline ($p = 0.003$). In a multivariable Cox regression analysis HMW VWF multimer deficiency was an independent predictor of all-cause mortality (hazard ratio [HR]: 3.02 [95 %CI: 1.31–6.96], $p = 0.009$).

Conclusion: This is the first study to demonstrate the predictive value of HMW VWF multimer ratio for risk stratification in patients with classical LF/LG AS. HMW VWF multimer deficiency was associated with an increased risk of all-cause mortality and major bleeding complications after AVR.

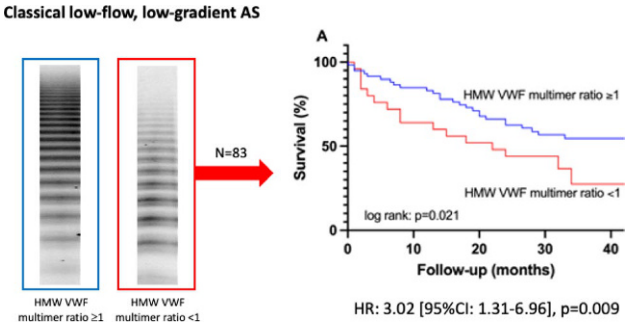

Fig. 1 | 16-5 Survival rates according to HMW VWF multimer ratio (<1 vs. ≥1) for the entire study population

16-6

Different calcification patterns of tricuspid and bicuspid aortic valves and their clinical impact

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Introduction: Mechanical strain plays a major role in the development of aortic calcification. We hypothesized that (a) valvular calcifications are most pronounced at the localizations subjected to the highest mechanical strain and (b) calcification

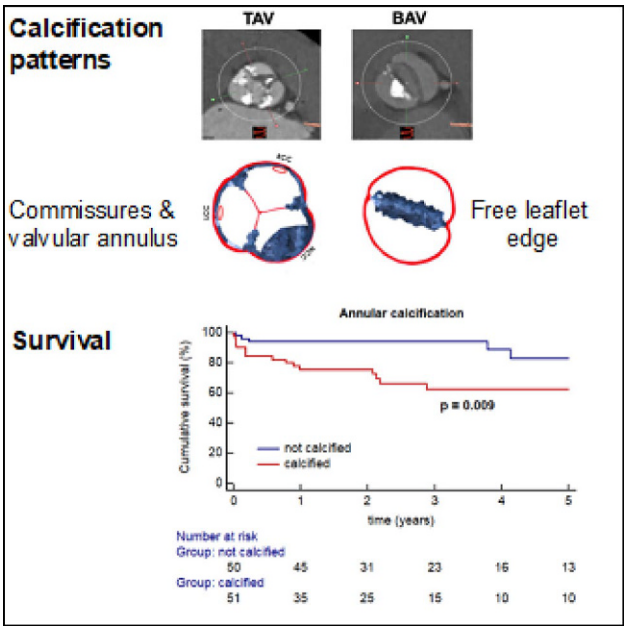

Fig. 1 | 16-6

patterns are different in patients with bicuspid (BAV) and tricuspid aortic (TAV) valves.

Methods: Multi slice computed tomography (MSCT) scans of 101 patients with severe aortic stenosis were analyzed using a 3D-post processing software to quantify calcification of TAV ($n=51$) and BAV ($n=50$) aortic valves after matching. Clinical follow-up for survival was assessed after a median of 2.3 years.

Results: BAV exhibited higher calcification volumes (1007 mm^3 vs. 825 mm^3, $p=0.014$) and increased calcification of the non-coronary cusp (NCC) (433 mm^3 vs. 341 mm^3, $p=0.018$) with significantly higher calcification of the free leaflet edge (529 mm^3 vs. 361 mm^3, $p<0.001$). The NCC showed the highest calcium load compared to the other leaflets (386 mm^3 vs. 270 mm^3 vs. 259 mm^3, $p=0.045$). Patients with annular calcification above the median had an impaired survival compared to patients with low annular calcification ($p=0.009$), whereas calcification of the free leaflet edge was not predictive ($p=0.53$).

Conclusion: Calcification patterns are different in aortic stenosis patients with BAV and TAV. Patients with high annular calcification but not free leaflet edge have an impaired prognosis.

16-7

Progranulin predicts intermediate recovery of systolic left ventricular fraction following transcatheter aortic valve implantation

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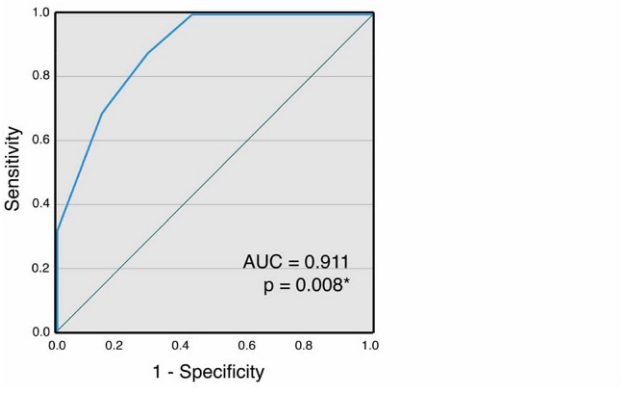
Introduction: Approximately one third of patients with severe aortic stenosis show reduced left ventricular ejection fraction (LVEF). Incidence and predictors for LVEF recovery following transcatheter aortic valve implantation (TAVI) have not been described sufficiently. Progranulin has been shown to be a promising biomarker for left ventricular reverse remodeling in patients with myocardial ischemia.

Methods: In this prospective cohort study, we included 53 consecutive individuals with severe symptomatic aortic stenosis admitted for TAVI. Patients underwent core laboratory echocardiographic assessment at baseline and follow-up at 6 and 12 months. Primary endpoint was LVEF recovery ≥10 % within 12 months after TAVI. Progranulin plasma levels were determined using BioVendor RMEE103R Human ELISA Kit prior TAVI. Correlation analysis and receiver operator characteristics were performed.

Results: Baseline characteristics are shown in Table 1. A total of 15 (28.3 %) out of 53 patients had LVEF ≤50 %. Of those 53.3 % showed LVEF recovery following TAVI at 6 and 12 months, respectively: 40.6 ±6.6 % vs. 47.1 ±14.1, $p=0.034$* and 48.5 ±10.8, $p=0.09$*. Progranulin plasma concentrations at baseline were increased in patients with severely impaired LVEF compared to patients with normal, mildly, or moderately reduced LVEF, $p=0.001$*, Table 2. Correlation analysis revealed an association between baseline Progranulin and LVEF recovery upon follow-up, r = 0.711, $p=0.001$*. Progranulin was able to predict intermediate recovery of systolic left ventricular fraction with an area under the curve of 0.911, $p=0.008$*.

Conclusion: In this pilot-study we found LVEF recovery in half of patients with prior reduced LVEF undergoing TAVI. Baseline Progranulin may be a promising biomarker for prediction of intermediate recovery of systolic LVEF.



Left ventricular ejection fraction recovery was defined ¤ 10 % upon follow-up. N = 15. * denotes p ¤ 0.05.

Fig. 1 | 16-7 ROC curve for Progranulin concentrations prior TAVI and post-procedural LVEF recovery

Table 1 | 16-7 Baseline patients' characteristics

	Overall	LVEF ≤50 %
Gender female n [%]	23 (43.4)	5 (33.3)
Age [years]	80.4 ± 5.1	81.4 ± 4.3
BMI [kg/m2]	28.6 ± 5.3	27.0 ± 4.8
NYHA functional class		
II n [%]	12 (22.6)	3 (20.0)
III n [%]	36 (67.9)	10 (66.6)
IV n [%]	5 (9.4)	2 (13.3)
Atrial fibrillation n [%]	26 (49.1)	10 (66.7)
CAD n [%]	41 (77.4)	14 (93.3)
PAD n [%]	13 (24.5)	6 (40.0)
Arterial hypertension n [%]	49 (92.5)	14 (93.3)
Stroke n [%]	6 (11.3)	1 (6.7)
Valve type		
Edwards Sapien 3 n [%]	16 (30.2)	6 (40.0)
Edwards Sapien XT n [%]	1 (1.9)	0 (0.0)
Medtronic Evolut R n [%]	25 (47.2)	0 (0.0)
Medtronic Evolut Pro n [%]	6 (11.3)	3 (20.0)
Medtronic Evolut Corevalve n [%]	5 (9.4)	6 (40.0)

LVEF left ventricular ejection fraction; *NYHA* New York Health Association; *CAD* coronary artery disease; *PAD* peripheral artery disease; *COPD* chronic obstructive pulmonary disease. * denotes $p \leq 0.05$.

Table 2 | 16-7 Progranulin concentration before TAVI

	LVEF ≥55 % $n=30$	LVEF <55–45 % $n=13$	LVEF <45–35 % $n=6$	LVEF <35 % $n=4$
Progranulin [pg/ml]	33.3 ± 7.9	35.3 ± 11.3 $p=0.507$	30.9 ± 5.4 $p=0.577$	50.4 ± 16.4 $p=0.001$*

LVEF left ventricular ejection fraction. All p-values compared to patients with LVEF >/= 55 %. * denotes $p \leq 0.05$.

16-8

A streamlined, machine learning-derived approach to risk-stratification in patients with moderate and severe secondary tricuspid regurgitation

Heitzinger G[1], Spinka G[1], Koschatko S[1], Baumgartner C[2], Dannenberg V[1], Halavina K[1], Mascherbauer K[1], Nitsche C[1], Donà C[1], Koschutnik M[1], Kammerlander A[1], Winter M[1], Strunk G[3], Pavo N[1], Kastl S[1], Hülsmann M[1], Rosenhek R[1], Hengstenberg C[1], Bartko P[1], Goliasch G[1]

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Introduction: Secondary tricuspid regurgitation (sTR) is the most frequent valvular heart disease and has significant impact on mortality. A high burden of comorbidities often worsens the already dismal prognosis of sTR, while tricuspid interventions remain underused and initiated too late. The objectives were to examine the most powerful predictors of all-cause mortality in moderate and severe sTR using machine learning techniques and to provide a streamlined approach to risk stratification using readily available clinical, echocardiographic and laboratory parameters.

Methods: This large-scale, long-term observational study included 3359 moderate and 1509 severe sTR patients encompassing the entire heart failure spectrum (preserved, mid-range and reduced ejection fraction). A random survival forest was applied to investigate the most important predictors, examine non-linear associations and group patients according to their number of adverse features, allowing for efficient risk-stratification. All results were consistent in internal validation.

Results: The most important predictors and investigated thresholds, that were associated with significantly worse mortality in moderate and severe sTR were age ≥75 years (≥70 years in severe sTR), NT-proBNP ≥4000 pg/ml, serum albumin <40 g/L, hemoglobin <13 g/dL and high sensitivity C-reactive protein ≥1.0 mg/dl. Additionally, grouping patients according to the number of adverse features yielded important prognostic information, as patients with 4 or 5 adverse features had a sevenfold risk increase in moderate sTR (7.11 [2.27–4.30] HR 95 %CI, P < 0.001) and fivefold risk increase in severe sTR (5.08 [3.13–8.24] HR 95 %CI, P < 0.001).

Conclusion: This study presents a streamlined, machine learning-derived approach to risk stratification in patients with moderate and severe sTR, that adds important prognostic information to aid clinical decision-making.

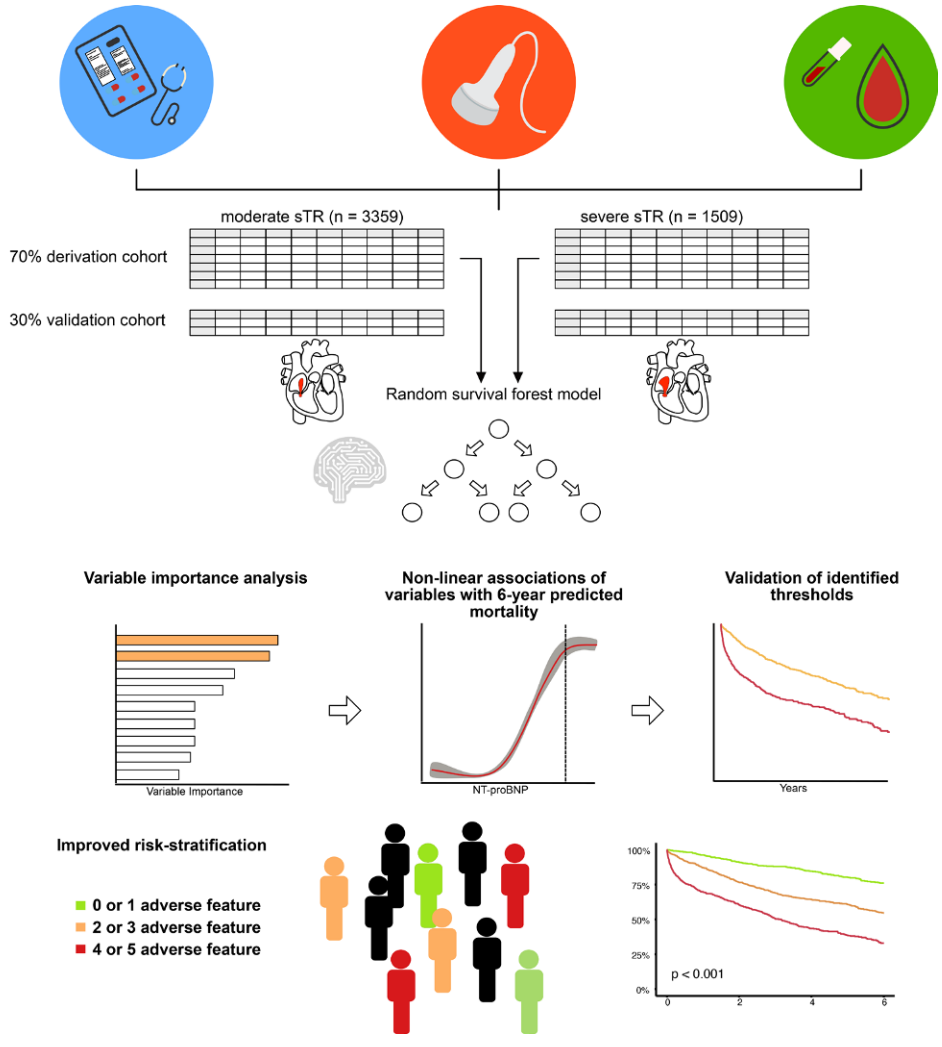

Fig. 1 | 16-8 A streamlined, machine learning derived approach to risk-stratification in secondary tricuspid regurgitation–4868 patients with moderate or severe secondary tricuspid regurgitation and heart failure, diagnosed in accordance with guideline recommendations were investigated using machine learning techniques. A broad spectrum of readily available clinical, echocardiographic and laboratory parameters were used to examine the most relevant predictors, assess non-linear associations and derive optimal thresholds. Stratifying patients according to the number of adverse features provided important prognostic information

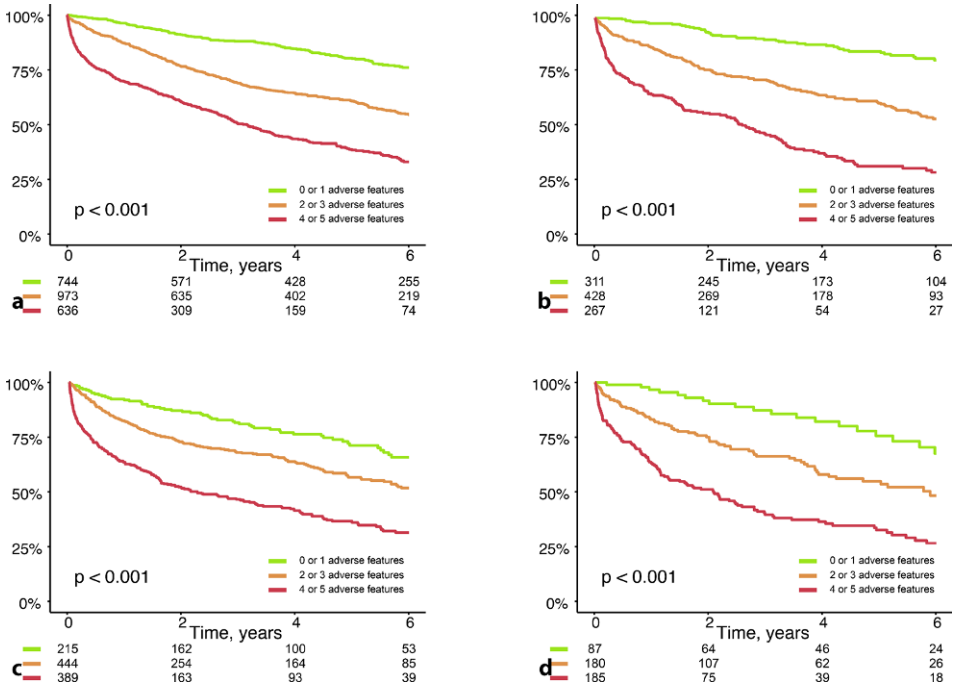

Fig. 2 | 16-8 Prognostic impact according to the number of adverse features–The most important predictors were identified and patients stratified according to their number of adverse features. Kaplan-Meier analysis shows excellent risk-stratification in patients with moderate sTR (derivation cohort (**a**), $n = 2353$, and validation cohort (**b**), $n = 1006$) and also in severe sTR (derivation cohort (**c**), $n = 1057$ and validation cohort (d), $n = 452$)

16-9

Reverse remodeling following valve replacement in coexisting aortic stenosis and transthyretin cardiac amyloidosis

Nitsche C[1], Koschutnik M[1], Donà C[1], Mascherbauer K[1], Kammerlander A[1], Heitzinger G[1], Dannenberg V[1], Halavina K[1], Winter M[1], Hacker M[2], Agis H[3], Rosenhek R[1], Bartko P[1], Hengstenberg C[1], Treibel T[4], Mascherbauer J[1], Goliasch G[1]

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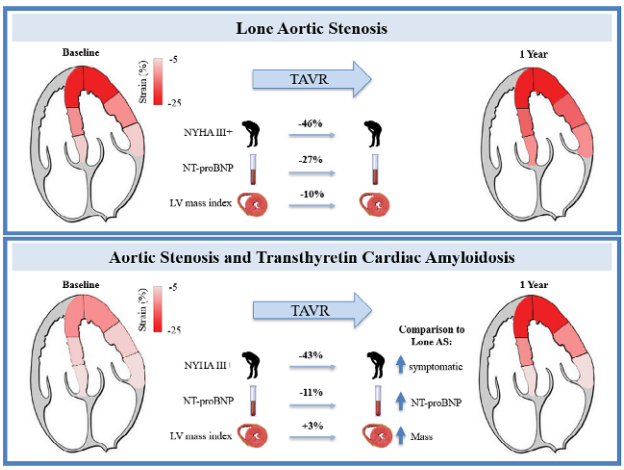

Fig. 1 | 16-9

Introduction: Dual pathology of severe aortic stenosis (AS) and transthyretin cardiac amyloidosis (ATTR) is increasingly recognized. Evolution of symptoms, biomarkers and myocardial mechanics in AS-ATTR following valve replacement is unknown. We aimed to characterize reverse remodeling in AS-ATTR and compare to lone AS.

Methods: Consecutive patients referred for transcatheter aortic valve replacement (TAVR) underwent ATTR screening by blinded 99mTc-DPD bone scintigraphy (Perugini Grade-0 negative, 1–3 increasingly positive) prior to intervention. ATTR was diagnosed by DPD and absence of monoclonal protein. Reverse remodeling was assessed by comprehensive evaluation before TAVR and at 1 year.

Results: 120 patients (81.8 ± 6.3 years, 51.7 % male, 95 lone AS, 25 AS-ATTR) with complete follow-up were studied. At 12-months (interquartile range [IQR] 7–17) following TAVR, both groups experienced significant symptomatic improvement by New York Heart Association (NYHA) functional class (both $p < 0.001$). Yet, AS-ATTR remained more symptomatic (NYHA ≥III: 36.0 % vs. 13.8, $p = 0.01$) with higher residual NT-proBNP levels ($p < 0.001$). Remodeling by echocardiography showed left ventricular mass regression only for lone AS ($p < 0.01$), but not AS-ATTR ($p = 0.5$). Global longitudinal strain (LS) improved similarly in both groups. Conversely, improvement of regional LS showed a base-to-apex gradient in AS-ATTR, whereas all but apical segments improved in lone AS. This led to the development of an apical sparing pattern in AS-ATTR only after TAVR.

Conclusion: Patterns of reverse remodeling differ from lone AS to AS-ATTR, with both groups experiencing symptomatic improvement by TAVR. Following AS treatment, AS-ATTR transfers into an ATTR cardiomyopathy phenotype likely amenable to specific treatment.

Postersitzung 17 – Diverse

17-1

Translation to German (Austrian) and qualitative linguistic validation of the Rapid Assessment of Physical Activity (RAPA) questionnaire

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Introduction: The Rapid Assessment of Physical Activity (RAPA) is a brief assessment tool for clinicians to capture physical activity (PA) levels in adults older than 50 years [1]. The RAPA was rated highest by the American Heart Association among 14 such available tools in a comparative evaluation. This evaluation took into account concurrent criterion validity, ability to assess compliance with the aerobic component of the PA guidelines, ability to assess compliance with the muscle strengthening component of the PA guidelines, test-retest reliability, and clinical feasibility [2]. The RAPA was developed in English at the Health Promotion Research Center of University of Washington, United States, in 2006. Translations of the RAPA from English to other languages have since been published. To date, however, a validated translation of the RAPA into German language has not been published. We therefore undertook a translation into German (Austrian) and a qualitative linguistic validation of the RAPA questionnaire.

Methods: To produce a rigorous translation of the RAPA, we applied the standard linguistic and cultural adaptation methodology of patient-reported outcome measures. This included (1) forward translation of the original English version (source questionnaire) to German by two independent translators, (2) reconciliation of the forward translations, (3) backward translation of the reconciled German version to English by an independent translator, and (4) comparison of the source questionnaire with the backward translation by an independent consultant. This resulted in a consolidated translation that was used for further qualitative linguistic validation by cognitive debriefing, an interview method in which participants are asked to verbalise their thought processes [3]. Thirteen adult Austrians (5 women and 8 men, age range 55–78 years, all native German-speakers) with heterogeneous cardiac, respiratory, neurologic and orthopaedic medical histories completed the translated RAPA and took part in cognitive debriefing interviews. The interviewer asked specific questions about the questionnaire content, wording, formatting and layout, and about participants' thought processes in completing the questionnaire. The questionnaire was amended iteratively after every two to three interviews, until five consecutive interviews raised no further issues requiring revision. The study was conducted according to standard ethical research guidelines. All interviewees gave written informed consent.

Results: In translating the RAPA from English to German (Austrian, Fig. 1), a number of decisions were made to match the wording and example activities to the local (Austrian) context, e. g., "Anstrengung" (effort/exertion) rather than "Intensität" (intensity), and "Hantel- und Krafttraining" (free weights and strength training) rather than "calisthenics". In the scoring instructions we adopted terminology from the official Austrian PA recommendations. Interviewees completed the RAPA on average in 2 min (range 1½ to 5½ min). Cognitive debriefing raised two main issues. Firstly, relating to the description of PA according to intensity levels, several participants made the point that, depending on the individual, the same example activity could be conducted at different intensity levels. However, when interviewees verbalised their thoughts in estimating their own intensity levels, it was apparent that they appropriately considered the physiological markers of intensity, rather than categorising strictly by type of activity. Secondly, relating to RAPA items 4 and 5, initial misunderstandings regarding the described amount and frequency of PA improved after several revisions of the wording. A tendency remained for interviewees to read items 4 and 5 repeatedly in order to grasp their intended meaning. However, this was considered adequate because

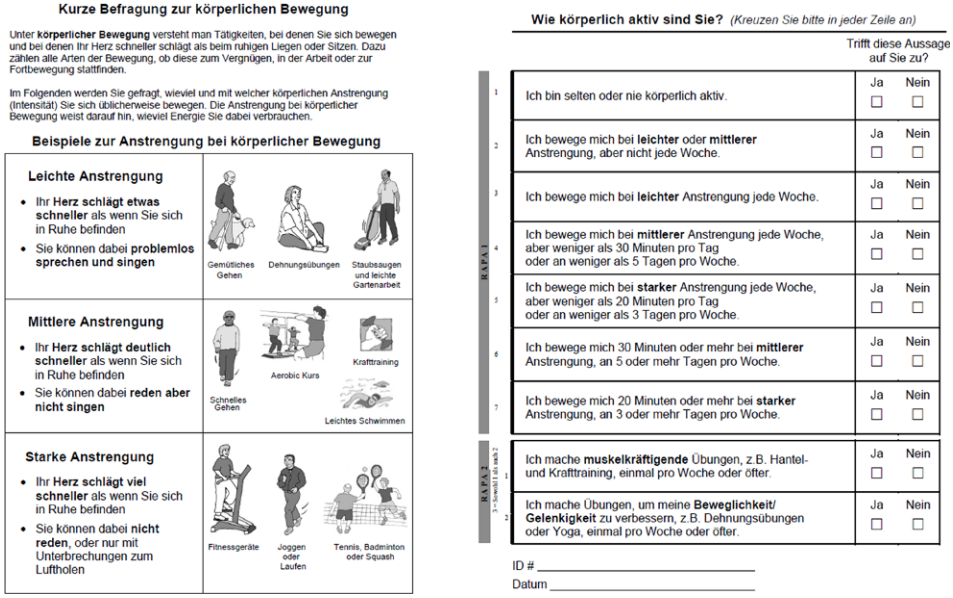

Fig. 1 | 17-1 German (Austrian) translation of the Rapid Assessment of Physical Activity (RAPA) questionnaire

interviewees demonstrated overall appropriate RAPA self-ratings when compared with accounts of their usual PA levels.

Conclusion: We have produced a German (Austrian) version of the RAPA questionnaire, applying a rigorous method of independent forward and backward translation and qualitative linguistic validation through cognitive debriefing with 13 older adults. Data collection for further psychometric validation of the new RAPA translation is currently underway. The translated version may be utilised in Austria to capture self-reported PA levels of German-speaking older adults in a standardised manner.

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17-2

RANTES and CD40L under conditions of long-term physical exercise, a potential connection to adaptive immunity

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Introduction: Exercising regularly was found to be associated with an improved immune response. RANTES and CD40L play a pivotal role in host defense, and individuals lacking adequate expression are prone to virus- and opportunistic infections. Therefore, we tried to illuminate a potential connection by measuring their serum levels under the conditions of long-term exercise.

Methods: 98 participants were enrolled in the study. The probands were asked to perform moderate physical activity for at least 150 min per week and/or vigorous-intensity exercise for at least 75 min per week. Bicycle stress tests were done at baseline and after 8 months of training to evaluate individual performance. Blood was drawn at baseline, after 2, 6, and 8 months to determine routine laboratory parameters and circulating serum levels of RANTES and CD40L.

Results: The study cohort consisted of 38.8 % female participants with an average age of 49.3 ± 6.7 years. RANTES and CD40L were found to increase by long-term physical exercise. In particular, probands with a performance gain of ≥3 % displayed a pronounced elevation of both markers, paired with a decrease in circulating IL6 levels and an improved lipid profile.

Conclusion: Conclusion: We were able to highlight rising levels of serum RANTES and CD40L under the conditions of physical exercise. Taking their role in host defense into account, conjunction of physical activity and the adaptive immune system could therefore be established. Furt

17-3

Der Zusammenhang zwischen kardiovaskulären Risikofaktoren und Depression einer erwachsenen Bevölkerung in Österreich: Implikationen für die Pflegeforschung und Pflegepraxis

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Einleitung: Herz-Kreislauf-Erkrankungen (HKE), die im Wesentlichen vermeidbar sind, und Depressionen treten oft gleichzeitig auf und gelten als schwerwiegende Gesundheitsprobleme mit den häufigsten Ursachen für Invalidität. Depression wurde als kardiovaskulärer Risikofaktor anerkannt [1–3]. Es gibt unterschiedliche Prävalenzen von kardiovaskulären Risikofaktoren und deren Behandlung und Prävention in Europa. Daher ist es wichtig zu erkennen, wo der Schwerpunkt der Prävention von HKE liegen sollte, um länderspezifische Programme zur Reduzierung der Belastung von HKE, insbesondere für Menschen mit Depressionen, umzusetzen [3]. Es wird davon ausgegangen, dass es in Österreich keine Studie gibt, die den Zusammenhang zwischen Depression und kardiovaskulären Risikofaktoren untersucht hat. Ziel dieser Studie ist es, den Zusammenhang zwischen den typischen kardiovaskulären Risikofaktoren Hypertonie, Body-Mass-Index (BMI) und Rauchen mit dem Schweregrad einer Depression bei einer erwachsenen österreichischen Bevölkerung zu untersuchen. Das zweite Ziel war es, geschlechtsbezogene Unterschiede zu identifizieren.

Methoden: Die vorliegende Arbeit ist eine Querschnittsstudie, die die Basisdaten aus dem BioPersMed (Biomarkers of Personalized Medicine) Kohorte der Medizinische Universität Graz, Österreich untersucht. Die erste nicht-wahrscheinliche Rekrutierung begann zwischen den Jahren 2011 und 2015 und führte zu einer Studienpopulation von 1022 erwachsenen Männern und Frauen, die im Großraum Graz lebten. Drei Risikofaktoren von HKE wurden für diese Arbeit untersucht und zur ersten Erhebung von geschulten Ärzten und Ärzt*innen und Pflegepersonal bewertet: selbstberichtete Diagnose von Bluthochdruck, selbstberichteter Raucherstatus und BMI (kg/m^2) durch die Messungen von Größe und Gewicht jedes Teilnehmers. Der PHQ-9 (Patient Health Questionnaire) wurde analysiert, indem der Gesamtscore-Wert in drei Gruppen umkodiert wurde. Deskriptive Statistik, statistische Korrelationsanalyse und eine ordinale Regressionsanalyse wurden durchgeführt.

Resultate: In der Datenanalyse wurden 977 Teilnehmer mit einem Durchschnittsalter von 57,3 Jahren eingeschlossen und mehr als die Hälfte der TeilnehmerInnen (56 %) waren weiblich. Die Ergebnisse zeigten einen signifikanten Zusammenhang zwischen Bluthochdruck und dem Schweregrad der Depression ($p < 0{,}01$), sowie bei allen drei kardiovaskulären Risikofaktoren für Männer und Übergewicht für Frauen. Menschen mit Bluthochdruck und erhöhtem BMI, angepasst an Alter, Geschlecht, Diagnose einer Depression und Rauchen, neigten mit höherer

Wahrscheinlichkeit in eine höhere Depressionsstufe zu fallen (BMI: ein Anstieg der „Log Odds“ um 0,033; $p < 0{,}01$; Hypertonie: Anstieg der „Log Odds“ um 0,339; $p < 0{,}5$). Darüber hinaus neigten Männer weniger häufiger in eine höhere Depressionsstufe zu fallen als Frauen (Senkung der „Log Odds“ von 0,435; $p < 0{,}5$).

Schlussfolgerungen: Dies ist die erste Querschnittsstudie, die den Zusammenhang zwischen kardiovaskulären Risikofaktoren und dem Schweregrad einer Depression bei einer erwachsenen österreichischen Bevölkerung, untersucht. Weitere Untersuchungen, bei denen Kausalität nachgewiesen werden kann, und Längsschnittanalysen in einer größeren Stichprobengröße, bei denen Generalisierbarkeit gegeben ist, werden empfohlen, um diese Ergebnisse gänzlich zu untersuchen und zu bestätigen. Die Wichtigkeit von Krankenpflegepersonen bei der Prävention von HKE ist feststehend, daher sollte ein Schwerpunkt auf die Verbesserung von Ressourcen, Programmen und Instrumenten zur Unterstützung der Interventionen von HKE in der Pflege gelegt werden. Des Weiteren sollte eine Agenda mit einer Orientierung und Prioritätensetzung in der kardiovaskulären Pflege für Pflegeforscher*innen, Förderagenturen und die Politik dienen. Die Ergebnisse liefern einen wichtigen Hinweis darauf, dass Präventionsprogramme von HKE in Österreich Screening und Management von kardiovaskulären Risikofaktoren in den Protokollen aufnehmen und entsprechend dem Schweregrad einer Depression anpassen sollen.

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17-4

Wearables zur Prävention und Überwachung in der Herzkreislaufmedizin

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Einleitung: Seit einigen Jahren finden Wearables für verschiedenste Indikationen als Lifestyle- (z. B. Fitnesskontrolle, Pulskontrolle bei Sport) und Medizinprodukte (z. B. Monitoring von Arrythmien/Vorhofflimmern, Heart Failure, Blutdruck, Schlafapnoe) zunehmend Verwendung. Von Laien für einzelne Indikationen oft kostengünstig käuflich, sind sie meist nicht als Medizinprodukt (Klasse I/Klasse IIa) zertifiziert und der Verordnung (EU) 2017/745 über Medizinprodukte und dem Medizinproduktegesetz unterliegend (EU) bzw. FDA cleared/approved (USA), doch werden Ärzte häufig auch mit hiermit erhobenen Daten konfrontiert. Wearables und Applikationen, die als Medizinprodukte zu Diagnostik und Behandlung dienen, sind seit 2019 im Rahmen der digitalen Gesundheitsanwendungen (DiGA) durch die Gesetzlichen Krankenkassen in Deutschland erstattungsfähig wodurch sich für Patienten die Kostensituation entspannt hat und die Verbreitung nochmals zugenommen hat.

Methoden: Ziel der Studie war die Betrachtung der in der Herzkreislaufmedizin derzeit gebräuchlichsten Wearables, der diesen zugrundeliegenden Technologien, der Indikationen zu deren Anwendung und deren regulatorischen Status.

Resultate: Bekannteste Wearables sind Smartwatches und Armbänder, Brustgurte und Pflaster. Diese erlauben eine kontinuierliche Messung verschiedener kardiologisch relevanter Parameter, z. B. physikalische Aktivität, Blutdruck, Herzfrequenz und EKG (QT-Zeit, ST-Hebung), arterielle Sauerstoffsättigung und Gewebeflüssigkeit über längere Zeiträume. Technisch erfolgt die Erfassung je nach Parameter über verschiedene Methoden, z. B. triaxiale Akzelerometer, Oszillometrie, Photoplethysmographie, Elektrokardiographie, Impedanzmessung. Die gewonnenen Daten können übertragen (z. B. durch Bluetooth und WLAN) und durch Algorithmen und künstliche Intelligenz ausgewertet und zur Diagnostik benutzt werden. Vorteile der Verwendung von Wearabels, z. B. in der Detektion kardialer Arrhythmien sind die kontinuierliche Erfassung und Auswertung hoher Datenmengen, die rasche Diagnosestellung mit hoher Sensitivität und Spezifität (z. B. für Vorhofflimmern >90 %), der mögliche Einsatz auch zur Therapiesteuerung von Patienten, die breite Verfügbarkeit ohne Notwendigkeit aufwendiger Hardware und die stärkere Einbindung von Patienten. Als potenzielle Nachteile stehen diesen jedoch z. B. die Abhängigkeit von Patientenschulung und Compliance, die Generierung einer hohen Anzahl auszuwertender Daten und die Datensicherheit der erhobenen Patientendaten, eine mögliche Überfrachtung durch die hohe Menge gewonnener Daten und, sofern keine Erstattung durch die Kostenträger erfolgt, die Kosten für die Patienten gegenüber.

Schlussfolgerungen: Die meisten Wearables in der Herzkreislaufmedizin dienen z. Zt. der Erfassung von physikalischer Aktivität, Herzfrequenz und Vorhofflimmern/EKG. Mit weiterer Verbreitung, besseren Algorithmen/künstlicher Intelligenz und niedrigeren Kosten sollten jedoch weitere Indikationen wie z. B. Heart Failure, Hyperkaliämie, Schlafapnoe und Führung von Patienten mit kardialen Erkrankungen und unter Rehabilitation hinzukommen. Entscheidend für die weitere Verbreitung der Wearables sind dabei die Einbindung der Handhabung und der erhobenen Daten in die klinische Diagnostik, die Datensicherheit und die Compliance der Patienten.

17-5

Safety and efficacy of starting a program for transfemoral catheter-based edge- to-edge tricuspid valve repair in high-risk patients with severe to torrential tricuspid regurgitation

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Introduction: Tricuspid regurgitation is a common disease associated with high morbidity and mortality mostly due to progressive right atrial and/or ventricular dilatation or dysfunction. Approaches to surgical reconstruction are limited because of high perioperative complication rate. Transcatheter transfemoral tricuspid repair was proposed to be safe and effective in patients of high perioperative risk due to multiple comorbidities and/or advanced age.

Methods: Quantitative and qualitative parameters of the first 20 patients including severity of tricuspid regurgitation before and after percutaneous tricuspid repair using MitraClipTM- or–after availability–the TriClipTM-System (Abbott) as well as periinterventional complications were retrospectively analyzed and recorded. Complications were defined as death, massive bleeding with the need of blood transfusion, myocardial infarction, hemorrhagic or ischemic stroke, acute kidney injury with the need of renal replacement therapy, prolonged intensive care unit stay and vascular injuries requiring surgical or radiological reconstruction. The last follow up echocardiography was accounted. Tricuspid regurgitation was graduated in mild, moderate, severe, massive and torrential. Demographic variables included age, previous percoutaneous mitral valve intervention, comorbidities such as atrial fibrillation, chronic coronary syndrome, diabetes, chronic kidney disease and arterial hypertension. Total amount of necessary clips was counted. TAPSE was assessed before intervention. Etiology of tricuspid regurgitation was documented. Implantation of MitraClipTM in tricuspid position was determined. Wilcoxon-Test was performed for statistical analysis.

Results: 20 Patients underwent a tricuspid edge-to-edge valve repair. 8 (40 %) patients had a torrential, 6 (30 %) patients a massive and 6 (30 %) patients a severe tricuspid regurgitation before intervention. Mean age was 79 years. 5 (25 %) patients had a previous or concomitant percutaneous mitral valve intervention. Atrial fibrillation was found in 19 (95 %), chronic coronary syndrome in 7 (35 %), diabetes in 3 (15 %), chronic kidney disease in 13 (65 %) and arterial hypertension in 16 (80 %) patients. Mean TAPSE before intervention was 18.3 mm, in 2 (10 %) patients a TAPSE <17 mm was assessed. 18 (90 %) patients suffered from secondary, 2 patients (10 %) from primary tricuspid regurgitation. In 7 (35 %) patients 1 clip, in 10 (50 %) patients 2 clips and in 3 (15 %) patients 3 clips were implanted. A MitraClipTM was implanted in 3 (15 %) patients, the other were treated with the TriClipTM-System. Tricuspid regurgitation was reduced in 8 (40 %) patients to moderate and in 12 (60 %) patients to mild early after intervention ($p < 0.001$). In the last follow up (mean duration 80 days) 1 (5 %) patient deteriorated from mild to moderate tricuspid regurgitation, all other patients showed stable results ($p = 0.317$). Efficacy was statistically persistent to the last follow up ($p < 0.001$). No periinterventional complications were documented.

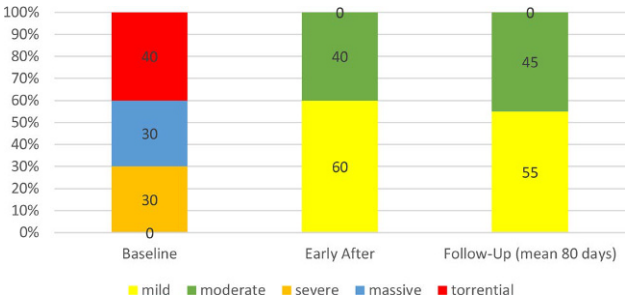

Fig. 1 | 17-5 Comparison of TR Severity at Baseline,Early After and Follow-Up (mean 80 days)

Conclusion: Starting a transfemoral catheter-based edge-to-edge tricuspid repair program in high-risk patients with severe to torrential tricuspid regurgitation appears to be safe and effective.

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17-6

Monozentrische Erfahrungen mit invasiver Diagnostik und Therapie der koronaren Mikrozirkulationsstörung

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Einleitung: Als INOCA (ischemia with non-obstructive coronary arteries) wird das Bestehen von Angina pectoris (AP) Sypmptomatik und nicht invasiv nachgewiesener myokardialer Ischämie ohne jedoch signifikanter angiographischer Stenose beschrieben. Als Ursache kommt unter anderem eine Dysfunktion der koronaren Mikrozirkukation (CMD) in Frage, welche mittels invasiver Diagnostik gemessen werden kann.

Methoden: Retrospektive Analyse einer Kohorte mit AP oder Dyspnoe, szintigraphisch positivem Ischämienachweis und fehlenden hämodynamisch wirksamen Koronarstenosen. Bei diesen Patienten wurde eine CMD-Messung mittels Druckmessdraht (PressureWire™ X Abbott, sowie Coroventis Software) durchgeführt und bei pathologischen Werten die medikamentöse Therapie individuell angepasst. Bei einem Teil der Patienten mit pathologischen Messwerten wurde nach Optimierung der medikamentösen Therapie sowohl eine invasive Nachmessung durchgeführt, als auch der klinische Therapieerfolg evaluiert.

Resultate: Im Zeitraum zwischen November 2020 und März 2022 wurde bei 37 Patienten aufgrund von therapierefraktärer AP Symptomatik oder Dyspnoe positivem Ischämienachweis und fehlender interventionspflichtiger Koronarstenosen eine Messung der koronaren Mikrozirkulation durchgeführt. 54 % dieser Patienten waren weiblich ($n = 20$), das mediane Alter betrug 63 Jahre (35; 83 Jahre). Bei 14 Patienten (38 %) wurde zumindest ein pathologischer Messwert (CFR und oder IMR) bei negativer FFR (>0,80) nachgewiesen. Von diesen Patienten hatten 9 eine pathologische IMR (24 %), einer isoliert eine erniedrigte CFR (3 %) und 4 Patienten eine kombinierte Störung von CFR und IMR (11 %). Drei Monate nach medikamen-

töser Optimierung wurde bei 6 Patienten (43 %) ein signifikanter Rückgang der zu Beginn pathologischen Werte gemessen (meist bis in den Normbereich). Dieses Ergebnis korrelierte mit deutlich rückläufigen klinischen Beschwerden.

Schlussfolgerungen: Die Diagnose der CMD ermöglicht einer Vielzahl von Patienten mit rezidivierender AP Symptomatik oder Dyspnoe bei fehlenden interventionspflichtigen Koronarstenosen eine individuell gesteuerte Optimierung der medikamentösen Therapie. Dadurch kann eine weitgehende Reduktion der Beschwerden bis zur Beschwerdefreiheit und somit Verbesserung der Lebensqualität erreicht werden.

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17-7

The importance of cardiovascular physiology in female carriers of duchenne muscular dystrophy

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Introduction: Duchenne muscular dystrophy (DMD) is a severe and progressive muscle-wasting disease. It is an X-linked recessive disorder caused by mutations in the DMD gene encoding dystrophin protein. Mutation prevents production of dystrophin which is a part of several protein complexes that function to strengthen muscle fibers, protecting them from injury as the muscles contract and relax. The cardiovascular manifestations include cardiac fibrosis, dilated cardiomyopathy, ventricular arrhythmia, and congestive heart failure. Heterozygous female DMD carriers are usually asymptomatic, however they may develop cardiovascular complications similar to homozygous male DMD patients at age of 40–60 years. However, female DMD carriers are underrepresented in existing studies and the cardiovascular complications are still not fully understood. This study aims to investigate the cardiovascular phenotype of heterozygous, 11-months old female Dmdmdx carrier rats and compare with age-matched (9 month old) wildtype female and male Dmdmdx rats.

Methods: The cardiac function, vascular endothelial function, cardiac fibrosis, expression of inflammatory markers, ACE and ACE2 activity, as well as protein expression of regulators of Ca2+ ion changes in the cardiomyocytes will be assessed. The methods involve echocardiography, wire myography, histological and immunohistochemical stains, qPCR, and western blotting, respectively.

Results: Preliminary results show that the endothelium-dependent vasodilation induced by the cumulative dosage of acetylcholine (Ach) was significantly impaired in aorta segments from female DMD carriers in comparison to wildtype female controls. In addition, close to 50 % carriers show sign of cardiac fibrosis and structural change of cardiac tissue. The evaluation of cardiac systolic and diastolic function, ACE activity and expression of markers of inflammation are in progress.

Conclusion: To our best knowledge we for the first time show that vascular endothelial dysfunction and cardiac fibrosis are present in female Dmdmdx carrier rat and may represent a promising small-animal model to elucidate mechanisms of cardiomyopathy development in the female dystrophic heart.

Postersitzung 18 – Interventionelle Kardiologie 2/Koronare Herzkrankheit

18-1

Left atrial appendage closure for stroke under oral anticoagulation

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Introduction: Transcatheter left atrial appendage closure (LAAC) is an established treatment option for patients with stroke despite adequate oral anticoagulation (OAC). However, there is no clear evidence regarding the post-interventional antithrombotic therapy and long-term outcome.

Methods: We analysed the baseline characteristics, the post-interventional antithrombotic regimen and long-term outcome of patients undergoing transcatheter LAAC for ischemic stroke despite oral anticoagulation from the Austrian LAAC Registry.

Results: Out of 372 patients undergoing LAAC between 2010 and 2021 at 9 centres, 23 patients with a history of stroke or thromboembolism under NOAC (78.3 %, $n=18$), VKA (4.3 %, $n=1$) or both (17.4 %, $n=4$) were identified. Mean±SD age was

71 ± 10 years and 17.4 % (n = 4) were female. CHA2DS2-VASc score was 4.7 ± 1.5 and HAS-BLED score was 2.7 ± 1.2. Patients received Amplatzer (26.1 %, n = 6), Amplatzer Amulet (56.5 %, n = 13) or Watchman (17.4 %, n = 4) devices. After LAAC, patients received either NOAC therapy (39.1 %, n = 9), NOAC plus clopidogrel or aspirin (30.4 %, n = 7), clopidogrel plus aspirin (13.0 %, n = 3), triple therapy (8.7 %, n = 2), single antithrombotic therapy or no antithrombotic therapy (4.3 %, n = 1 for both). Long-term antithrombotic therapy consisted of NOAC (60.8 %, n = 14), antiplatelet therapy (17.4 %, n = 4) or no antithrombotic therapy (21.7 %, n = 5). During long-term follow up (20 ± 16 months, total 463 person-months), no stroke, bleeding or death occurred; hospitalizations for other reasons occurred in 17.4 % (n = 4).

Conclusion: After LAAC, long-term NOAC therapy is discontinued in a considerable proportion of patients with increased stroke risk without contraindication to OAC. This approach is not associated with increased rate of ischemic events.

18-2

Influence of OFF-hours admission on outcome of CS-patients at a high-volume cardiology centre

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	admission time during ON-hours* (n=74)	admission time during OFF-hours** (n= 174)	
age [years], median (iqr 1;3)	72 (61;79)	69 (58;77)	p= 0.206
female gender, n (%)	16 (21.6)	54 (31.0)	OR 0.61; 95 % CI [0.31-1.21]; p= 0.165
body mass index, median (iqr 1;3)	26.7 (24.1;29.4)	26.9 (24.6;30.5)	p= 0.579
known renal insufficiency, n (%)	21 (28.4)	36 (20.7)	OR 1.52; 95 % CI [0.78-2.97], p= 0.192
known diabetes, n (%)	20 (27.0)	36 (20.7)	OR 1.42; 95 % CI [0.72-2.79], p= 0.320
known hypertension, n (%)	43 (58.1)	90 (51.8)	OR 1.29; 95 % CI [0.72-2.33], p= 0.405
known hyperlipidemia, n (%)	26 (35.1)	56 (32.2)	OR 1.14; 95 % CI [0.62-2.10], p= 0.661
known smoker/ex smoker, n (%)	18 (24.3)	41 (23.6)	OR 1.04; 95 % CI [0.53-2.06], p= 0.898
prior myocardial infarction, n (%)	16 (21.6)	30 (17.2)	OR 1.32; 95 % CI [0.64-2.74], p= 0.476
prior PCI/CABG, n (%)	22 (29.7)	38 (21.8)	OR 1.51; 95 % CI [0.78-2.92], p= 0.197
underlying disease, n (%)			
- STEMI/NSTEMI	47 (63.5)	127 (73.0)	OR 0.64; 95 % CI [0.35-1.20]; p= 0.172
- others	27 (36.5)	47 (27.0)	
location of index event, n (%)			
- out-of-hospital	38 (51.4)	111 (63.8)	OR 0.60; 95 % CI [0.33-1.08], p= 0.089
- in-hospital	36 (48.6)	63 (36.2)	
CPR, n (%)	47 (63.5)	114 (65.5)	OR 0.92; 95 % CI [0.50-1.68]; p= 0.773
lactate at admission [mmol/l], mean (± SD)	3.9 (± 3.2)	5.3 (± 4.8)	**p= 0.014**
pH at admission, median (iqr 1;3)	7.31 (7.20;7.41)	7.27 (7.14;7.36)	p= 0.146
syst. RR at admission [mmHg], median (iqr 1;3)	99 (85;115)	97 (80;116)	p= 0.453
norepinephrine dose at admission (12.5 mg/50 mL) [mL/hrs], median (iqr 1;3)	4 (3;7)	4 (2;7)	p= 0.991
intubated at arrival, n (%)	42 (56.8)	116 (66.7)	OR 0.66; 95 % CI [0.36-1.19], p= 0.151
treatment with MCS, n (%)	17 (23.0)	31 (17.8)	OR 1.47; 95 % CI [0.72-3.00], p= 0.289

*Monday-Friday, 8am to 4pm
**Saturday, Sunday; Monday-Friday 4pm to 8am

Fig. 1 | 18-2 Patient characteristics

	admission time during ON-hours* (n=74)	admission time during OFF-hours** (n= 174)	
syst. RR change after 24hrs [mmHg], median (iqr 1;3)	11 (-2;18)	11 (-6;20)	p= 0.919
lactate change after 24 hrs [mmol/l], median (iqr1;3)	-1.2 (-3.0;0.0)	-2.0 (-4.0;-0.4)	p= 0.196
pH change after 24 hrs, median (iqr 1;3)	0.11 (0.02;0.19)	0.12 (0.05;0.23)	p= 0.789
ventilation duration [days], median (iqr 1;3)	2 (0;7)	2 (0;6)	p= 0.451
ICU stay [days], median (iqr 1;3)	4 (1;12)	4 (2;9)	p= 0.471
total hospital stay [days], median (iqr1;3)	9 (3;18)	9 (2;19)	p= 0.769
complications (bleeding, sepsis), n (%)	9 (12.2)	24 (13.8)	OR 0.87; 95 % CI [0.35-2.09], p= 0.839
death within 24 hrs after admission, n (%)	18 (24.3)	42 (24.1)	OR 1.01; 95 % CI [0.51-1.99], p= 0.975
survival before intrahospital death [days], median (iqr1;3)	2 (0;7)	2 (0;6)	p= 0.736
intrahospital death, n (%)	41 (55.4)	84 (48.3)	OR 1.33; 95 % CI [0.74-2.39], p= 0.333

*Monday-Friday, 8am to 4pm
**Saturday, Sunday; Monday-Friday 4pm to 8am

Fig. 2 | 18-2 Outcome

Introduction: Outcome of patients, admitted during off-duty hours is an important quality measure of a medical service. This is especially true for critically-ill emergency diseases, in which proper and on-time therapy can have major impact on survival. The aim of our work was to compare in-hospital outcome of patients, presented with cardiogenic shock (CS) during on-duty hours versus off-duty hours at our centre.

Methods: All consecutive patients, who were admitted to a high-volume tertiary interventional cardiology centre with CS between 2019 and 2021 were enrolled in this prospective registry. Inclusion criteria was hemodynamic instability, requiring vasopressors. Patients were divided into two groups, according to being admitted during "ON-hours" (Monday-Friday, 8 am–4 pm) or during "OFF-hours" (Saturday, Sunday; Monday-Friday 4 pm–8 am). Data about patient characteristics, as well as the applied interventional- and intensive therapies were collected prospectively in a case report form. Primary endpoint was the in-hospital mortality.

Results: In total 248 patients were recruited. 174 (70 %) were admitted during OFF-hours, while 74 (30 %) during ON-hours. ON-hours-patients tended to exhibit a slightly more unfavourable risk profile compared to OFF-hours patients with regard to higher age (median 72 vs 69 years, $p=0.206$) and higher prevalence of known pre-existing vascular risk factors such as renal insufficiency (28.4 % vs 20.7 %, $p=0.192$), diabetes (27.0 % vs 20.7 %, $p=0.320$) and hypertension (58.1 % vs 51.8 %, $p=0.405$). While OFF-hours patients presented with markedly higher lactate levels compared to ON-hours patients (mean 5.3 ±4.8 vs 3.9 ±3.2 mmol/l, respectively; $p=0.014$), there was no difference either in the use of mechanical circulatory support between ON- and OFF-hours patients (23.0 % vs 17.8 %, respectively; $p=0.289$), or in mechanical ventilation (56.8 % vs 66.7 %, respectively; $p=0.151$) (see Fig. 1). In-hospital mortality was high, but comparable between ON- and OFF-hours patients (55.4 % vs 48.3 %, $p=0.333$). Mortality within the first 24 h after admission (24.3 % vs 24.1 %, $p=0.975$) as well as survived days before in-hospital death (median 2 vs 2 days, $p=0.736$) were similar between these groups (see Fig. 2).

Conclusion: Although patients, admitted with CS during OFF-hours, were in slightly worse hemodynamic status, their mortality was comparable with patients admitted during ON-hours. This finding suggests that quality of care can be maintained for 24/7 at our centre.

18-3

An unexpected cause of fever, night sweats and cough in times of COVID-19

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Introduction: Several days after receiving his third COVID-19 vaccination a 38-year-old man presented to a regional hospital due to persistent fever, night sweats and cough. In combination with increased inflammatory parameters and radiological signs of pneumonia in the left upper lobe and the lingula antibiotic therapy was initiated. A search for autoimmune diseases, immunosuppression and pathogens including SARS-CoV-2 were unremarkable. However, unexpectedly symptoms did not improve under this therapy. The patient was referred to our hospital, where in synopsis with persistent significantly elevated inflammatory parameters a computed tomography (CT) was performed. Besides progressive infiltrates and possible signs of bleeding into the lungs a subtotal stenosis of the left superior pulmonary vein (LSPV) could be found. This finding was almost certainly in context with a pulmonary vein isolation (PVI) performed for symptomatic therapy-refrac-

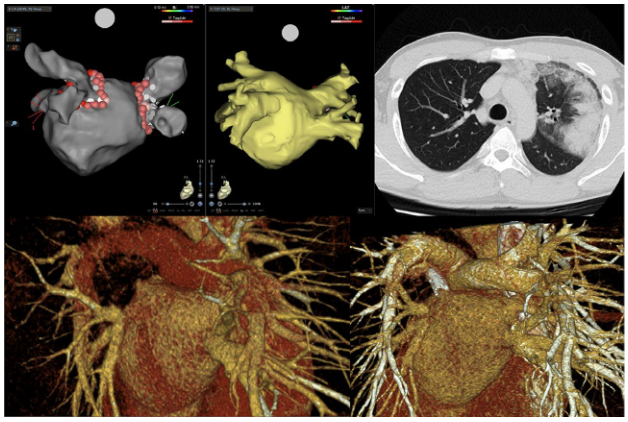

Fig. 1 | 18-3

tory atrial fibrillation 3 months ago. The report from the PVI recorded an unproblematic CARTO-procedure with the common variant of 2 × 2 pulmonary veins, a satisfying merge of the FAM-Map with the pre-interventional computed tomography scan and a first-pass isolation done with a 3.5 mm SmartTouch SF catheter using the CLOSE protocol (AI 550 anterior and 400 posterior, 45–50 watts). Taking everything in account the most likely cause of the pneumonia was a mechanical draining problem induced by pulmonary vein stenosis.

Methods: Therefore, we decided to perform a pulmonary vein dilatation. Under conscious sedation and fluoroscopy guidance a single transseptal puncture was performed. We then positioned a guidewire through a steerable sheath into the LSPV so we could pre-dilate the stenosis with a Boston Scientific NC 5.00 × 15 mm balloon (max. 8 atm). For the high probability of a re-stenosis we placed an Abbott Omnilink-Elite 10/19 mm vascular balloon-expandable stent system at the LSPV-ostium.

Results: We were able to document a nice result of the pulmonary vein dilatation with quite an impressive venous flow suggestive of the high pressure in the lung tissue caused by the congestion. Matching the highly gratifying clinical course CT confirmed a stable diameter of the LSPV as well as a significant reduction of the pneumonic infiltrates so the patient could be discharged in less than a week from the intervention. His medication at discharge consisted of aspirin 100 mg q. d. in combination with clopidogrel 75 mg q. d. over the course of the next 6 months.

Conclusion: All in all the probability of a symptomatic pulmonary vein stenosis after catheter ablation remains low (0.23–1.1 %) and in that same case the spontaneous course might sometimes even be favorable. When deciding to going for an interventional therapy approach stenting is preferred over percutaneous transluminal angioplasty for the lower recurrence-rates, yet keeping potential difficulties with the advanced anticoagulation regimen in mind [1].

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18-4

Significant reduction of scatter radiation exposure in interventional procedures for operator and assistant with a ceiling-suspended protection system–Data from the OSCAR Registry

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Introduction: Chronic exposure to scatter radiation (SCR) causes a significant degree of work-related damages in interventional cardiologists (IC), including cataracts, vascular alterations, and left-sided brain tumors. Conventional lead aprons provide no protection for the head. The openings for the arms leave a large entry for lateral radiation into the mediastinum. Even with protection glasses, the eye lenses are insufficiently protected. A ceiling suspended operator radiation protection system (Zero Gravity, CFI Medical Solutions, MI, USA), addresses these shortcomings with additional SCR protection for the head with a lead glass visor and for the mediastinum with additional lateral protectors, while being weightless for the operator. The ZG system has shown high efficacy in reducing scatter radiation for the operator in a limited number of trials. Currently, all larger studies collected data only with single digital dosimeters in one recording position.

Methods: We have created a prospective registry for Occupational SCAtter Radiation (OSCAR Registry; EK Nr. 1069/2021; clinicaltrials.org identifier NCT04945538) in order to (A) measure realistic per-procedure SCR doses at multiple critical anatomical locations of the IC (frontal head at eye level, left lateral head, left shoulder) and sterile assistant (Left head/neck) and (B) to study the impact of the ZG system on IC and sterile assistant (SA) SCR exposure when used in addition to the current standard of X-ray protection (SXP) in unselected all-comers cardiologic procedures. Methods: IC and SA were equipped with a total of 5 Unfors RaySafe i3 live-dosimeters (Unfors Raysafe Inc, Billdal, Sweden) at prespecified locations. 1125 consecutive cardiac procedures were recorded, in which either both IC and SA were using SXP (lead apron, thyroid shield) or the IC was using the ZG system and the SA was wearing SXP. In all procedures a suspended lead shield, patient lead cover, and an adjustable lead side-shield were present. Diagnostic angiographies (DA) and interventions (PCI) were grouped separately, the IC's and SA's SCR doses were compared. Statistic averages are shown as Mean±SEM. Groups were compared with the two-sample t-test or Mann-Whitney-U test. $p < 0.05$ was considered statistically significant.

Results: SCR doses were recorded in a total of 1125 procedures, 697 DA and 428 PCI. Compared to SXP, the use of the ZG device reduced the average SCR doses per procedure of the IC recorded at the left lateral head from 9.25 ± 0.56 µSv to 0.54 ± 0.06 µSv in DA (–94 %; $n = 445/252$, $p < 0.0001$) and from 22.00 ± 1.58 µSv to 1.23 ± 0.13 µSv for PCI (–94 %; $n = 269/160$, $p < 0.0001$). The IC's average frontal dose at eye level was reduced from 2.79 ± 0.15 µSv to 0.27 ± 0.03 µSv in DA (–90 %; $n = 445/252$, $p < 0.0001$) and from 6.56 ± 0.47 µSv to 0.49 ± 0.06 µSv in PCI (–92 %; $n = 269/160$, $p < 0.0001$). Consistently, the dose recorded immediately under the IC's left shoulder was reduced from 24.62 ± 1.40 µSv to 0.83 ± 0.14 µSv in DA (–97 %; $n = 445/252$,

$p = < 0.0001$) and from 65.30 ± 4.78 µSv to 1.68 ± 0.21 µSv in PCI (–97 %; $n = 269/160$, $p < 0.0001$). Furthermore, when the IC used the ZG system, the average SCR dose recorded at the SA's head was reduced from 2.17 ± 0.14 µSv to 1.18 ± 0.09 µSv in DA (–46 %, $n = 445/252$, $p = < 0.0001$) and from 9.53 ± 0.83 µSv to 3.83 ± 0.46 µSv in PCI (–60 %, $n = 269/160$, $p < 0.0001$). All SCR dose effects remained significant after correction for total dose-area product (µSv/Gy*cm2). Procedure duration, contrast use, procedural success rate and patient radiation dose were not affected by ZG use.

Conclusion: Consistent with preliminary data we had presented in 2021 [1], the current analysis of 1125 cases from the OSCAR Registry shows reference values for SCR exposure of IC and SA at multiple recording sites and confirms an impressive potential for SCR reduction when using the ZG system in daily cathlab routine. ZG provided significant protection for ICs in critical anatomical areas–even in a state-of-the-art cathlab inventory with multiple SCR reduction measures already in place. The current larger dataset also confirms a protective effect for the sterile assistant at the table wearing SXP. These findings, together with a growing number of clinical trial results, call for greater awareness of SCR protection in interventional cardiology and suggest a routine implementation of additional X-ray protection systems like ZG in order to drastically reduce cathlab staff SCR exposure.

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18-5

Prozeduren, Patientencharakteristika, Komplikationen und Patientenzufriedenheit an einer tertiären kardiologischen Tagesklink: Follow-Up des Innsbruckers kardiologischen Tagesklinikregister (IKTR)

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Einleitung: Das „Innsbrucker Kardiologische Tagesklinikregister" (IKTR) wurde mit Eröffnung der kardiologischen Tagesklinik im Oktober 2020 begonnen. Ziel ist es die Anzahl an Prozeduren, die Patientencharakteristika, sowie die Anzahl an Nulltagesaufenthalten konsekutiv zu erfassen. Eine telefonische Verlaufskontrolle nach einem Monat soll die Patientenzufriedenheit sowie die Verschiebung von Komplikationen in den extramuralen Bereich erfassen.

Methoden: Von Oktober 2020–August 2021 erfolgte eine retrospektive Auswertung der Patientendaten aus den elektronischen Patientenakten. Ab August 2021 wurden Patienten im Rahmen der Tagesklinischen Untersuchungen prospektiv in das IKTR eingeschlossen. Insgesamt wurden 757 tagesklinische Aufenthalte vereinbart. Davon mussten 2,5 % der Aufnahmen abgesagt werden. Von den 738 Aufnahmen wurden 666 Patienten für eine Koronarangiografie aufgenommen, davon waren 28 % weiblich. Ein telefonisches Follow-Up wurde bei allen Patienten nach mindestens einem Monat durchgeführt. Die Anzahl der gesamten elektiven Angiografien wurde anhand der MEL Kodierungen und ICD-10 Codes des Vergleichszeitraum 12.10.2020–11.10.2021 erhoben (DD0 ...).

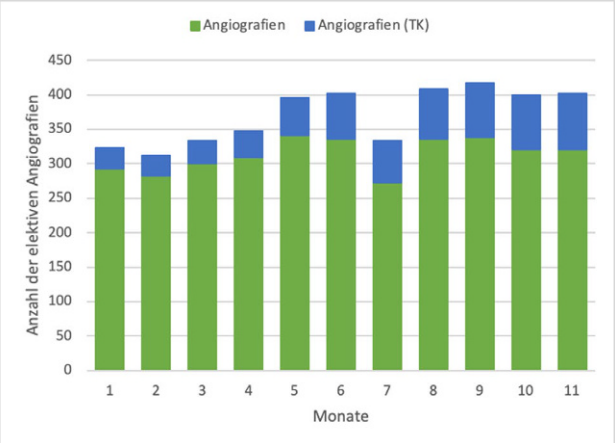

Abb. 1 | 18-5 Anteil der Angiografien an der Medizinischen Universität Innsbruck. 19,7 % der elektiven Koronarangiografien konnten über die Tagesklinik durchgeführt werden (*TK* Tagesklinik)

Resultate: Das mediane Alter der Patienten betrug 63 (IQR 50–76) Jahre und 30 % wiesen relevante Komorbiditäten auf. Im untersuchten Zeitraum wurden an der Kardiologie Innsbruck 3378 elektive diagnostische Links- und/oder Rechtsherzkatheteruntersuchungen durchgeführt. Es wurden 19,7 % der elektiven Koronarangiografien über die Tagesklinik durchgeführt. Es wiesen 46 % eine signifikante, interventionsbedürftige, Koronare Herzerkrankung auf. 34 % der Patienten erhielten eine Stentimplantation, 10,7 % der Patienten wurden für eine chirurgische Revaskularisierung vorgeschlagen. Eine Verlegung aufgrund einer periinterventionellen Komplikation wurde in 0,9 % der Fälle durchgeführt. Ein telefonisches Follow-Up wurde bei 574 Patienten nach Angiografie durchgeführt. Drei Patienten sind verstorben. Leichte Beschwerden im Bereich der Gefäßzugänge traten bei 35 % der Patienten nach Entlassung auf. Der Großteil davon (82 %) berichtete über ein Hämatom (53 %) oder Schmerzen (30 %) an der Einstichstelle. 37 Patienten (6,4 %) suchten wegen Komplikationen einen Arzt auf. 60 % davon traten ab dem zweiten Tag nach Entlassung auf. 18 (5,2 %) der Patient, die rein tagesklinisch betreut wurden, berichteten von einer Komplikation, die mit einem Arztbesuch verbunden war. Bei den Patienten, die noch stationär weiterversorgt wurden, waren es 8,3 % ($p = 0,17$). Insgesamt berichten die Patienten über eine sehr hohe Zufriedenheit mit dem tagesklinischen Aufenthalt (mediane Zufriedenheit 100/100, IQR 90–100).

Schlussfolgerungen: Die Einführung der kardiologischen Tagesklinik ermöglichte innerhalb eines Jahres den Anteil an tagesklinischen Angiografien in Tirol von 1.0 % im Jahr 2019, auf 12 % der elektiven Angiografien zu heben. Die Rate an Komplikationen mit Arztbesuch bei den tagesklinisch entlassenen Pateinten war nicht signifikant höher als bei den Patienten, die stationär weiterbehandelt worden sind. Die Zufriedenheit mit dem Tagesklinikaufenthalt war sehr hoch.

Eigenschaft	Alle (N=574)	TK (N=344)	STAT (N=230)
Alter (Jahre)			
Durchschnitt	63,0 ± 13,0	62,0 ± 8,8	63,0 ± 9,2
Geschlecht (n/%)			
weiblich	158 / 27,60	104 / 30,23	54 / 23,47
Beschwerden Einstichstelle (n/%)			
ja	199 / 37,40	123 / 35,75	76 / 33,04
Komplikationen (n/%)			
ja	37 / 6,40	18 / 5,23	19 / 8,26
Art der Komplikation (n/%)			
Hämatom (Punktionsstelle)	105 / 18,30	63 / 18,31	42 / 18,26
Blutung (Punktionsstelle)	6 / 1,04	2 / 0,58	4 / 1,73
Schmerzen (Punktionsstelle)	59 / 10,30	44 / 12,79	15 / 6,52
Apoplex/TIA	2 / 0,30	1 / 0,29	1 / 0,43
Myokardinfarkt	2 / 0,30	0 / 0	2 / 0,86
Blutung	3 / 0,50	1 / 0,29	2 / 0,86
Verschluss der Arteria radialis	4 / 0,70	2 / 0,58	2 / 0,86
Andere	18 / 3,10	10 / 2,90	8 / 3,47

Abb. 2 | 18-5 Es handelt sich hier um alle Patienten, bei denen eine Angiographie durchgeführt worden ist und die anschließend ein Follow-Up erhalten haben. Die Aufteilung erfolgt dann nach Patienten die tagesklinisch entlassen wurden und nach Patienten die noch stationär behandelt werden mussten. Die Rate an Komplikationen mit Arztbesuch bei den tagesklinisch entlassenen Pateinten war nicht signifikant höher als bei den Patienten, die stationär weiterbehandelt worden sind (*TK* Tagesklinik, *STAT* stationär)

18-6

Durchschnittliche Liegedauer nach einem kardiologischen Tagesklinikaufenthalt im Vergleich zur Normalstation: Eine Analyse nach Propensity Score Matching

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Einleitung: Die zunehmende Kostenerhöhung stellt eine der größten Herausforderungen für das österreichische Gesundheitssystem dar. Die Verlagerung der Patient:innenversorgung vom stationären in den tagesklinischen Bereich ist dabei ein zentrales Element zur Reduktion der Liegedauer und somit zur Kostensenkung. Die Tirol Kliniken als Betreibergesellschaft der Universitätsklinik für Innere Medizin III der Medizinischen Universität Innsbruck eröffneten im Jahre 2020 eine kardiologische Tagesklinik. Das Ziel dieser Studie war es, die Liegedauer an der Tagesklinik mit Patienten welche über die Normalstation aufgenommen wurden, zu vergleichen. Dies ist insofern relevant, da kardiologische Eingriffe, insbesondere Stentimplantationen, derzeit noch eine Überwachung über Nacht erfordern. Die Daten dieser Analyse sollen daher über den potenziellen Effektivitätsgewinn durch die Einführung einer Tagesklinik Aufschluss geben.

Methoden: Im Zuge einer retrospektiven Datenerhebung wurden im Zeitraum von der Eröffnung der kardiologischen Tagesklinik im Oktober 2020 bis einschließlich Mai 2021 654 Patient:innen der Normalstation sowie 332 tagesklinisch betreute Patient:innen erfasst. Alle Patienten wurden zur Durchführung einer diagnostischen Koronarangiographie (CAG) aufgenommen. Der primäre Effektivitätsendpunkt war ein Vergleich der Anzahl der Übernachtungen pro Patient:in. Dazu erfolgte ein Propensity Score Matching (PSM), um vergleichbare Kohorten zu bilden. Der sekundäre Endpunkt war die Anzahl der Nulltagesaufenthalte an der Tagesklinik. Für das PSM wurden Patientencharakteristika welche die Verweildauer potenziell beeinflussen können, vordefiniert: Alter, Geschlecht, kardiale Symptome, Anzahl der kardiovaskulären Risikofaktoren, Vorliegen einer bekannten Koronaren Herzerkrankung sowie relevante Komorbiditäten. Es konnten so 309 Patient:innen der Tagesklinik mit 309 der Normalstation verglichen werden. Der Unterschied in den Ausgangscharakteristika war zwischen beiden Gruppen nach PSM nicht signifikant.

Resultate: Die Liegedauer, in Nächten, nach durchgeführter CAG war an der neu eingerichteten Tagesklinik signifikant kürzer als auf der kardiologischen Normalstation. Dies war sowohl

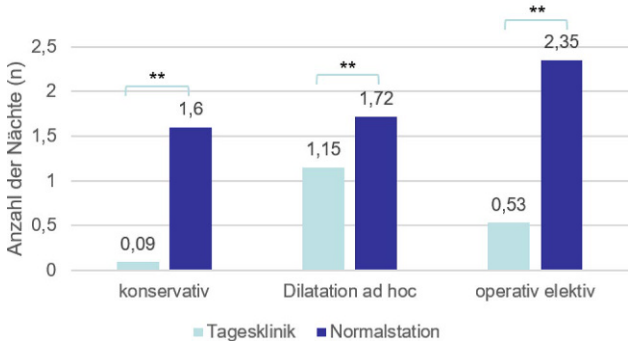

Abb. 1 | 18-6 Nach erfolgtem PSM berechnete durchschnittliche Liegedauer in Nächten. Im Vergleich die Werte aus der Tagesklinik und der Normalstation (Unterschied: *ns* nicht signifikant; *signifikant $p < 0{,}05$; **höchst signifikant $p < 0{,}001$; *PSM* Propensity Score Matching)

Tab. 1 | 18-6 Propensity score matching (PSM). Im Vergleich die Werte vor bzw. nach erfolgtem PSM. Eine standardized difference (SD) von unter 10 % für eine im Vergleich zwischen den beiden Kohorten gegebene Kovariate, spricht für ein geringes Ungleichgewicht. Der anfänglich durch die berechnete standardized difference (SD) dargestellte große Unterschied zwischen den Gruppen konnte im Sinne von vergleichbaren Kollektiven verringert werden (PSM Propensity score matching, TK Tagesklinik, NS Normalstation, SD standardized difference, KHK koronare Herzerkrankung)

Eigenschaft		vor PSM			nach PSM	
TK (N = 332)	NS (N = 647)	SD (%)		TK (*N* = 309)	NS (*N* = 309)	SD (%)
Alter (Jahre)						
Durchschnitt	61,5 ± 8,1	68,3 ± 9,4	55,0	62,4 ± 7,4	63,0 ± 8,4	4,8
Geschlecht (%)						
weiblich	24,10	36,30	26,8	25,60	28,50	6,5
männlich	75,90	63,70	26,8	74,40	71,50	6,5
Relevante Komorbiditäten (%)						
nein	76,50	63,20	29,3	75,40	75,40	0
ja	23,50	36,80	29,3	24,60	24,60	0
Aktive Symptomatik (%)						
nein	12,40	19,70	20,0	12,90	11,70	3,7
ja	87,60	80,30	20,0	87,10	88,30	3,7
bekannte KHK (%)						
nein	88,80	82,40	18,3	88,30	86,40	5,7
ja	11,20	17,60	18,3	11,70	13,60	5,7
Hochrisikopatient*in (> 2 Risikofaktoren; %)						
nein	61,10	66,80	11,9	41,1	38,5	5,3
ja	38,90	33,20	11,9	58,9	61,5	5,3
Liegedauer in Nächten						
(alle)	0,47 ± 0,92	1,88 ± 1,10	99,2	0,48 ± 0,95	1,72 ± 0,84	103

vor (0,47 ± 0,92 vs. 1,88 ± 1,10 Nächte, $p < 0{,}001$) als auch nach erfolgtem PSM (0,48 ± 0,95 vs. 1,72 ± 0,84 Nächte, $p < 0{,}001$) der Fall. Bei genauerer Betrachtung der Liegedauer je nach durchgeführter Prozedur, konnte nach PSM festgestellt werden, dass die Patient:innen der Tagesklinik jeweils eine signifikant kürzere Aufenthaltszeit aufwiesen als jene der Normalstation. Bei konservativem Procedere 0,09 ± 0,30 vs. 1,60 ± 0,67 Nächte ($p < 0{,}001$), bei „ad hoc" durchgeführter Koronarintervention 1,15 ± 1,14 vs. 1,72 ± 0,82 Nächte ($p < 0{,}001$) und bei einer in weiterer Folge geplanten elektiven Bypass-Operation 0,53 ± 1,37 vs. 2,35 ± 1,38 Nächte ($p < 0{,}001$). Der Anteil der Nullnachtaufenthalte belief sich auf 65,7 % aller tagesklinischen CAG-Aufnahmen.

Schlussfolgerungen: Bei ähnlichen Patient:innen kann durch eine tagesklinische Aufnahme die durchschnittliche Liegedauer – sowohl bei Patienten mit konservativem Procedere, als auch bei Patient:innen mit Koronarintervention oder Indikation zur Bypass OP – signifikant reduziert werden.

18-7

Long-term outcome in patients with chronic total occlusion–comparison between drug-eluting vs. bare-metal stents: a retrospective single center experience

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Introduction: The aim of the present study is to evaluate the outcome of patients receiving at least one drug-eluting stent (DES) in successful reopened chronic total occlusion (CTO) compared with bare-metal stents (BMS), in a real-world setting.

Methods: Three-hundred sixty-six consecutive patients were enrolled, and retrospectively subdivided in three group: DES new-generation (DESng; sirolimus-, everolimus-, zotarolimus- or biolimus-eluting stents), DES first-generation (DESfg) (sirolumus- and paclitaxel-eluting stents), as well as BMS in a retrospective analysis of a prospective registry from January 2003 until August 2020. The combined endpoint all-cause mor-

tality or target vessel revascularization (TVR) during a mean follow-up period of 5.61 ± 3.71-years was evaluated.

Results: Three-hundred fifteen (86.07 %) patients received a DES of which 238 a DESng (65.00 %), 77 a DESfg (21.00 %), while in 51 patients (13.93 %) a BMS was implanted. In total, 96/315 (30.4 %) patients reached the combined endpoint of all-cause mortality or TVR, 55/238 (23.11 %) in the DESng group, 25/77 (32.47 %) in de DESfg group, and 16/51 (31.37 %) in BMS group. The bivariable Cox-Hazard-Regression analysis shows a Hazard Ratio [HR] between DESng and DESfg of 1.115 (95 % CI: 0.685–1.814, p-value = 0.661). The HR for DESfg vs. BMS was 0.920 (95 % CI: 0.491–1.724, p = 0.796) and for DESng vs. BMS it was 0.889 (95 % CI: 0.504–1.568, p = 0.684).

Conclusion: In the present study, the long-term event rate for all-cause mortality or TVR in patients undergoing successful recanalization of a CTO was between 21 and 32 % without any. statistically significant difference between the different groups. CTO by use of new generation DES showed a tendency for a lower primary combined endpoint compared with first generation DES or BMS.

18-8

Short- and long-term outcome in patients with chronic total occlusion–comparison of successful intervention vs. failure: a retrospective single center experience

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Introduction: In this study we evaluated short- and long-term mortality in patients with one CTO who were successfully recanalized by percutaneous coronary intervention (PCI) and stent implantation versus unsuccessful interventions between January 2010 until August 2020.

Methods: Two-hundred ninety-one consecutive patients who underwent PCI and stent implantation for CTO were enrolled in this retrospective analysis of a prospective registry. CTO patients with successful recanalization were compared with a group of patients with unsuccessful recanalization at a mean follow-up duration of 4.22 ± 3.14-years. As combined primary endpoint all-cause mortality, or target vessel revascularization (TVR) were evaluated.

Results: In two-hundred thirty-seven (81.44 %) patients the intervention was successful, while in fifty-four (18.56 %) the intervention was unsuccessful. Fifty-eight (24.57 %) patients in the successful recanalization group, and 13 (24.07 %) in the unsuccessful PCI group reached the combined endpoint. The bivariable Cox-Hazard-Regression analysis showed a no significant difference between successful vs. failed CTO-PCI (HR = 1.20; 95 % CI: 0.550–2.617, p-value = 0.647) after 1-year. Also, after a 10-year follow-up there was no difference between groups (HR = 0.967; 95 % CI: 0.530–1.765, p = 0.912).

Conclusion: In our hands short-and long-term outcome with respect to all-cause mortality and target vessel revascularization was not different between successful and non-successful CTO-PCI.

Postersitzung 19 – Herzinsuffizienz 3/ Pulmonale Hypertension

19-1

Clinical and echocardiographic characteristics of female patients with breast cancer treated with anthracycline-based combined chemotherapy

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Introduction: The most common malignancy of women is the breast cancer. The therapy encompasses surgery, radiotherapy and systemic drug applications, depending on the tumor type (hormone receptor-positive (HRpos)/human epidermal growth factor receptor 2 (HER2)-negative (HER2neg), or HER-2pos or triple-negative BC, with or without metastases and the HER2-low-positive BC). Systemic treatment includes several types of drugs, including among others anthracyclines, Selective Estrogen Receptor Downregulators (SERDs), monoclonal antibody (such as trastuzumab), or immune checkpoint inhibitors (ICI). Anthracyclines and trastuzumab, especially their combination elicit cardiotoxic effects in dose-dependent manner, leading to myocardial fibrosis and activation of collagen synthesis, manifested as left ventricular dysfunction and heart insufficiency. The aim of our prospective registry was to evaluate the clinical and laboratory signs of cardiotoxicity and the cardiovascular outcome of female patients with breast cancer.

Methods: We have analyzed data of our prospective clinical registry (EC: 1534/2012), and selected female patients with breast cancer treated with combined chemotherapy including antracycline or its derivates. Further inclusion criteria were data availability of transthoracic echocardiography and laboratory investigations within 3-month before or after study inclusion. Exclusion criteria were any anticancer treatment without anthracycline drugs, second malignancy in the past or present, imaging and laboratory data out of the given time frame, insufficient data of therapy. Descriptive statistics was used to present the data as mean±standard deviation, or incidences given in percent (%).

Results: After exclusion of 25 patients, totally 30 patients have been included into this preliminary analysis. Mean age was 69.5 ± 12.5 year, 12 patients had relapse or metastases. The mean time between treatment start and study inclusion was 17.8 ± 14.9 months. The left ventricle was enlarged in 20 % of the patients, with mild/moderately/severely reduced left ventricular function in 40 %/13.3 %/6.7 %. Mild to moderate diastolic dysfunction was recorded in 26.7 % of the patients. Mild enlarged right ventricle with mild reduced function was observed in 6.7 % with mild to moderate tricuspid insufficiency in 36.7 % and estimated systolic pulmonary pressure of 40.3 ± 9.7 mm Hg. The median value of proBNP was 1891 pg/mL (137;1276; Interquartile Range, IQR), troponinT 89.8 ng/L (IQR: 9.8; 91.5), with normal CK and CKMB with moderate renal insufficiency (GFR: 54.9 ± 12.7 mL/min/1.73 m^2). We observed high frequency of concomitant cardiovascular disorders, as 16.6 % of patients had permanent atrial fibrillation, 26.7 % coronary artery disease and/or peripheral vascular diseases.

Conclusion: Female patients with breast cancer receiving combined chemotherapy with anthracycline have a high incidence of cardiotoxicity proven by laboratory or echocardio-

graphic imaging. Further investigations are ongoing including patients receiving novel anticancer treatments expected with less cardiotoxicity.

19-2

Validation of HF specific cut-offs of iron deficiency and natural course of parameters of iron status in stable HFrEF

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Introduction: Around 40–60 % of patients with chronic and acute HFrEF are affected by iron deficiency (ID) defined by HF specific cut-offs. The cut-offs for ID in HF were established on expert opinion and remain without validation. The causes for iron deficiency in HFrEF are unclear, whereas both reduced dietary intake and an inability of counterregulatory mechanisms as upregulation of gastrointestinal iron uptake have been discussed. ID is associated with more severe symptoms and reduced exercise capacity and is an independent predictor for adverse outcomes. Intravenous iron supplementation with ferric carboxymaltose (FCM) has been shown to increase exercise capacity and reduce hospitalizations and holds a class IIa recommendation in the most recent ESC guidelines. Data on the natural course of parameters of iron status, the clinical predictors for declining iron status and thereby enhanced risk to develop ID in HFrEF however are not available. The present study aims to assess ferritin, transferrin and transferrin saturation (TSAT), investigate their impact on outcome, validate the HF specific cut-offs and describe timely changes in iron status in patients with stable HFrEF.

Methods: Consecutive patients with stable chronic HFrEF and guideline directed medical therapy (GDMT) have been enrolled prospectively from the outpatient unit of heart failure between November 2010 and March 2021. Medical records and routine laboratory parameters including ferritin, transferrin and TSAT levels measured by the central laboratory have been documented for consecutive visits, i. e. at baseline (first available measurement), 6 ± 3 months, 12 ± 3 months, 18 ± 3 months

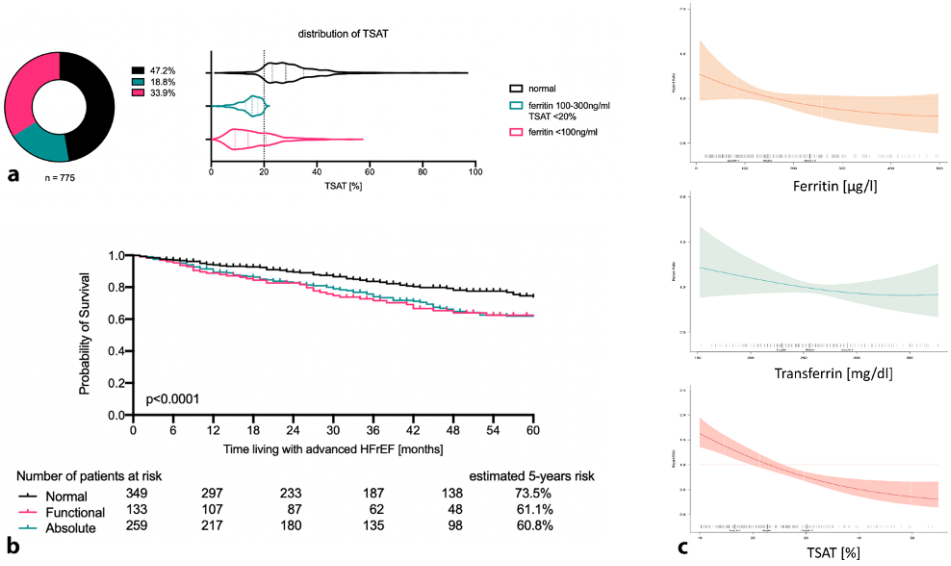

Fig. 1 | 19-2 Prevalence and types of ID in stable HFrEF and prognostic value. **a** The prevalence of normal iron status and ID as defined by guideline criteria in HFrEF are shown as a pie chart, distribution of TSAT for the different types of ID are displayed as violin plots. **b** Kaplan-Meier curves for HFrEF patients accroding to baseline iron status, comparison was done by the log-rank test. **c** Spline curve analysis for ferritin, transferrin and TSAT levels regarding all-cause mortality

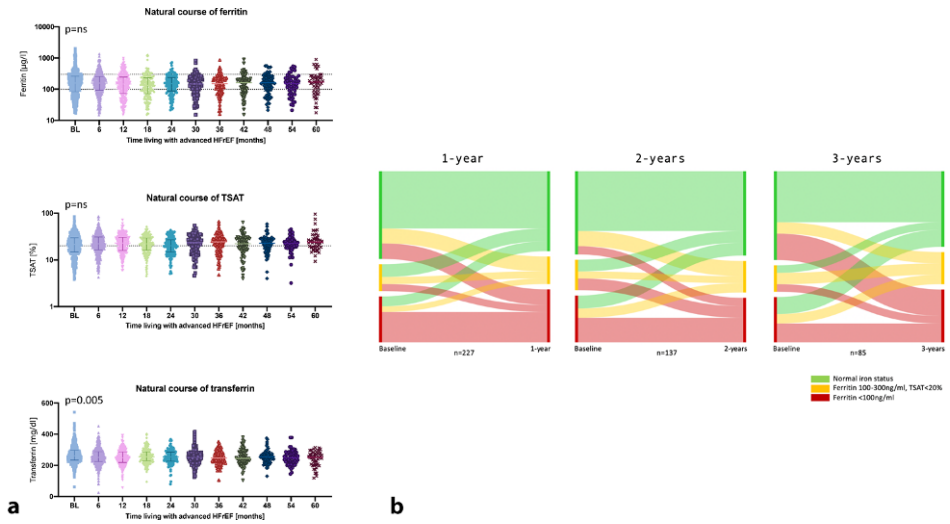

Fig. 2 | 19-2 Natural course of iron status in HFrEF. **a** Ferritin, transferrin and TSAT levels are shown as individual values and median (IQR) for different FUP timepoints. **b** Sankey diagrams for ID categories and paired data at 1-, 2- and 3-years FUP

etc., respectively. Changes in iron status were analyzed for the follow-up (FUP) timepoints. All-cause mortality was assessed as the primary outcome.

Results: A total of 775 patients were included into the study. Baseline iron status was analyzed for all patients. 61 patients received iv iron during the observation period and were therefor excluded from the analysis of changes of iron status. Median age was 62 years (IQR 53–72), 77 % were male and median NT-proBNP levels were 2031 pg/ml (IQR 856–4241). 47.2 % of patients showed normal iron status, whereas 18.8 % had ferritin 100–300 ng/ml and TSAT <20 % and 33.9 % had ferritin <100 ng/ml (Fig. 1a). 7.1 % of patients with normal iron status had TSAT <20 % and 28.1 % of patients with ferritin <100 ng/ml had TSAT >20 %. ID was associated with worse survival while survival curves for both types of ID were visually superimposable ($p < 0.0001$, log-rank test) (Fig. 1b). Spline curve analysis confirmed an increased mortality at values for ferritin <100 ng/ml and TSAT <20 % with surprising accuracy (Fig. 1c). TSAT seems to reflect risk best with a narrow confidence interval. Regarding the course of iron status there were no impressive changes during a FUP time of 5 years (p = ns for ferritin and TSAT, unpaired test across all timepoints) (Fig. 2a). When analyzing patients with 1-, 2- and 3-years FUP not only worsening of iron status but also improvement was apparent (Fig. 2b).

Conclusion: With 52.8 % ID is very common in stable HFrEF patients. ID is associated with worse outcome, whereas outcome is comparable for both HF specific ID categories, as probably TSAT <20 % and not ferritin levels is the key driver for bad outcome. Our data validate for the first time the HF specific cut-offs used to define ID with an increased morality at ferritin levels <100 ng/ml and TSAT <20 %, while TSAT seems more discriminative. Furthermore, iron status is subject for timely variation for both worsening and improvement without specific intervention. Identification of patients at risk for developing ID needs further analysis.

19-3

Geometrical structure alterations in coronary resistance artery network and the potential role of Tenascin C in diabetes

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Introduction: This study aimed to characterize the geometrical structure alterations in coronary resistance artery network and the potential role of Tenascin C (TNC), using diabetic mice.

Methods: Streptozotocin (STZ) induced diabetic mice models (n = 7–11 animals in each group) in Wild type (A/J) and Tenascin C KO (TNC KO) were used. 16–18 weeks post STZ injection, heart was dissected and micro-preparation of the whole subsurface network of the left coronary artery (down to branches of 40 µm outer diameter) was performed, followed by in situ pressure-perfusion and analysis using video-microscopy. Outer and inner diameters, wall thicknesses and bifurcation angles were measured on whole network pictures reconstructed into collages at 1.7 µm pixel resolutions.

Results: Data indicate that diabetic networks are significant associated with abnormal morphological alterations including trifurcations, sharp bends of larger branches, and branches directed in the retrograde direction ($p < 0.001$ by the $\chi 2$ test). Networks of TNC KO mice tended to form significant early divisions producing parallel running larger branches ($p < 0.001$ by the $\chi 2$ probe). Diabetic networks were substantially more abundant in 100–180 µm components, appearing in 2–5 mm flow distance from orifice. This was accompanied by thickening of the wall of larger arterioles (>220 µm) and thinning of the wall of a population of smaller (100–140 µm) arterioles ($p < 0.001$). Interestingly, diabetes model by STZ-injection did not induce further geometrical changes in TNC KO mice. Blood flow should cover larger distances in diabetic networks.

Conclusion: In diabetic mice, a combined network remodeling of the coronary vasculature was observed with hypertrophic and hypotrophic remodeling and vasculogenesis at well defined, specific positions of the network. TNC plays an important role in the formation of network geometry, and TNC knockout induces parallel fragmentation preventing diabetes-induced abnormal vascular morphologies.

19-4

Implantable pumps for treprostinil in pulmonary hypertension: Experience over more than a decade

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Introduction: Implantable pumps for intravenous treprostinil may overcome the limitations of administration via external pumps like painful site reactions for subcutaneous use or life-threatening catheter-related infection associated with the intravenous route. Since 2010 we have acquired vast experience with implantable pumps for treprostinil in pulmonary hypertension (PH).

Methods: We document all data of patients with PH in ELPHREG (ELisabethinen Pulmonary Hypertension REGistry). We evaluated all patients who underwent pump implantation until December 2021.

Results: We identified 106 patients (53 female and male each), mean age 66.8 years at time of pump implantation (range 16–87). The vast majority of patients was diagnosed with pulmonary arterial hypertension (PAH), other diagnoses included CTEPH, CpcPH and group V PH. All patients had been uptitrated subcutaneously to a mean dose of 26.4 ng/kg/min (range 7.4–120.8). Mean time on subcutaneous therapy was 9 months (range 1–78). Both planned and unplanned surgical interventions were exclusively performed by the dedicated team. Intraoperatively one case of ventricular tachycardia was observed, during postoperative stay one hypotensive episode, three cases of pneumothorax and one case of hematothorax in a patient with concomitant hematological malignancy and one case of pleural effusion were successfully managed. In 8 cases mild seroma were observed postoperatively, none of them requiring invasive treatment. 14 unplanned surgical interventions were performed during a follow-up of total 2915 patient-months mainly related to mechanical issues with the system like dislocation of the catheter or rupture of pump fixation but also one case of skin necrosis requiring change of the pump had to be managed. A suspected damage of the pump refill septum in one patient was under investigation at the manufacturer at the time of evaluation. An important finding in the long-term treatment was the increase of the flow-rate of the pump leading to shortening of the refill-interval from four to three weeks and finally to pump replacement in meanwhile 14 patients. During meanwhile more than 3000 refill procedures at our outpatient clinic we have observed a single complication leading to hospitalization of the patient for overdosing. No catheter related infection was observed.

Conclusion: The use of implantable pumps is associated with a complication rate of below 0.5 per 1000 patient-days at our center. The increase of flow-rate over time needs careful observation. Development of improved devices is warranted.

19-5

The one-minute sit-to-stand test (1-min STST) and echocardiographic findings in patients with heart failure with preserved ejection fraction (HFpEF)

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Introduction: Heart failure with preserved ejection fraction (HFpEF) is a common disease associated with poor outcome [1]. Close clinical-follow up and early diagnosis are crucial in the management of these patients. Echocardiography plays a pivotal role in the diagnostic process of HFpEF and is a good tool to evaluate cardiac function using left ventricular ejection fraction, estimated systolic pulmonary artery pressure (ePASP) and right ventricular tricuspid annular plane systolic excursion (TAPSE). High ePASP and low TAPSE as well as TAPSE/ePASP ratio are associated with higher mortality in HFpEF patients [2–4]. Despite echocardiographic markers, precise risk stratification remains challenging. The one-minute sit-to-stand-test (1-min STST) is a quick and objective test of functional capacity as was shown in previous studies [5–7] and can be potentially used besides echocardiography for risk stratification in HFpEF. Objective: The aim of this investigation was to prospectively examine whether there are any differences in echocardiographic parameters between patients with limited 1-min STST performance and those with preserved 1-min STST performance.

Methods: We evaluated 39 HFpEF with the 1-min STST. All patients underwent standard transthoracic echocardiography including measurements of left ventricular systolic function, ePASP and TAPSE. Patients were divided into two groups based on their number of 1-min STST repetitions using the age- and sex-stratified norm-reference values developed by Strassmann et al. [8] for healthy people. Limited 1-min STST performance was defined as ≤50 % of predicted 1-min STST repetitions (group I, $n=24$), preserved 1-min STST performance as >50 % of predicted 1-min STST repetitions (group II, $n=15$) Fig. 1).

Variable	All patients N = 39	Group I: Limited exercise capacity N = 24 (62%)	Group II: Preserved exercise capacity N = 15 (38%)	*p*-value
Demographics				
Age, years	71 ± 11	70 ± 13	77 ± 8	0.547
Female, sex	24 (62%)	13 (68%)	7 (54%)	0.403
BMI, kg/m²	30.17 ± 7.97	31.79 ± 8.40	27.56 ± 6.70	0.108
NT-proBNP, pg/mL	2462.62 ± 2661.59	2969.24 ± 3151.38	1652.03 ± 1329.89	0.080
Comorbidities				
Hyperlipidemia	31 (80%)	18 (75%)	13 (87%)	0.380
Systemic hypertension	29 (74%)	21 (88%)	8 (53%)	**0.017**
Atrial fibrillation	26 (67%)	14 (58%)	12 (80%)	0.163
COPD	11 (28%)	9 (38%)	2 (13%)	0.103
Diabetes mellitus	18 (46%)	10 (42%)	8 (53%)	0.477
Echocardiographic parameters				
Left ventricular ejection fraction, %	52.57 ± 1.74	51.89 ± 0.33	53.24 ± 3.15	0.218
Left ventricular size, mm	42.51 ± 5.73	41.96 ± 6.44	43.40 ± 4.42	0.452
Right ventricular size, mm	39.21 ± 7.41	40.63 ± 6.95	36.93 ± 7.78	0.132
Left atrial size, mm	60.55 ± 10.43	59.00 ± 9.57	62.40 ± 11.62	0.460
Right atrial size, mm	62.33 ± 10.91	62.58 ± 11.12	61.93 ± 10.93	0.859
Estimated pulmonary artery systolic pressure, mmHg	64.55 ± 22.19	70.52 ± 23.59	55.40 ± 16.69	**0.038**
Peak tricuspid regurgitation velocity, m·s⁻¹	3.55 ± 0.81	3.81 ± 0.69	3.18 ± 0.84	**0.018**
TAPSE, mm	17.46 ± 3.81	15.75 ± 3.40	19.73 ± 3.13	**0.001**
Tissue Doppler imaging of the RV, m/s	0.17 ± 0.16	0.17 ± 0.17	0.18 ± 0.15	0.880
TAPSE/ePASP ratio	0.31 ± 0.13	0.25 ± 0.97	0.38 ± 0.12	**< 0.001**

Notes. Continuous variables are presented as mean ± standard deviation; categorical variables are given as numbers (percentages).
Abbreviations: BMI indicates body mass index; NT-proBNP: N-terminal pro-brain type natriuretic peptide; COPD: chronic obstructive pulmonary hypertension; TAPSE: tricuspid annular plane systolic excursion; RV: right ventricle.

Fig. 1 | 19-5 Basic characteristics of our study population

Results: Consistent with known characteristics of the HFpEF patients our study sample presents with a relatively high mean age of 71 ±11 years, female predominance ($n=24$, 62 %) and elevated body mass index (grade I obesity on average). Patients with limited 1-min STST performance (group I) showed worse echocardiographic parameters with a higher ePASP ($p=0.038$), higher tricuspid regurgitation velocity (TRV) ($p=0.018$) and more reduced TAPSE ($p=0.001$) as well as TAPSE/ePASP ratio ($p<0.001$) compared to patients in group II. There were no statistically significant differences in remaining echocardiographic parameters, demographic data and comorbidities between the two groups, aside from arterial hypertension ($p=0.017$).

Conclusion: Patients with worse 1-min STST performance had worse echocardiographic parameters. Because impaired ePASP, TAPSE and TAPSE/ePASP ratio are associated with higher mortality and because in our investigation patients with worse echocardiographic parameters performed worse in the 1-min STST, we postulate that the 1-min STST may be used as an additional diagnostic tool for identifying vulnerable HFpEF patients.

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19-6

Chronic thromboembolic pulmonary hypertension and left ventricular filling pressures

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Introduction: Chronic thromboembolic pulmonary hypertension (CTEPH) is characterized by chronic obstruction of major pulmonary arteries with organized thrombi and is classified as pre-capillary pulmonary hypertension (PH) by the current hemodynamic definition of the guidelines. However, clinical risk factors for PH due to left heart disease (LHD) including features of the metabolic syndrome, left-sided valvular heart disease and stable ischemic heart disease can be frequently observed in patients with CTEPH. The aim of this study was to investigate the prevalence, mechanisms and prognostic implications of elevated left ventricular filling pressures (LVFP) in patients with CTEPH.

Methods: 394 consecutive CTEPH patients undergoing a first diagnostic right and left heart catheterization were included in this study. mPAWP and LVEDP were utilized for assessment of LVFP. Two cutoffs were applied to identify patients with elevated LVFP: (1) mPAWP and/or LVEDP >15 mm Hg as recommended by the current PH guidelines and (2) mPAWP and/or LVEDP >11 mm Hg, which is the upper limit of normal in healthy subjects. Clinical and echocardiographic features as well as long-term mortality data were assessed.

Results: LVFP was >15 mm Hg in 41 (10.4 %) and >11 mm Hg in 155 patients (39.3 %). Univariable logistic regression analysis identified age, body mass index, systemic hypertension, diabetes, atrial fibrillation, mitral regurgitation and left atrial volume as significant clinical predictors of elevated LVFP. Systemic hypertension, atrial fibrillation, mitral regurgitation and left atrial volume remained independent determinants of LVFP in adjusted analysis. LVFP >11 mm Hg was associated with worse long-term survival (p-logrank = 0.020).

Conclusion: Elevated LVFP is common in patients with CTEPH at the time of diagnosis. Elevated LVFP in CTEPH appears to be due to comorbid left heart disease. CTEPH patients with LVFP >11 mm Hg have worse outcomes.

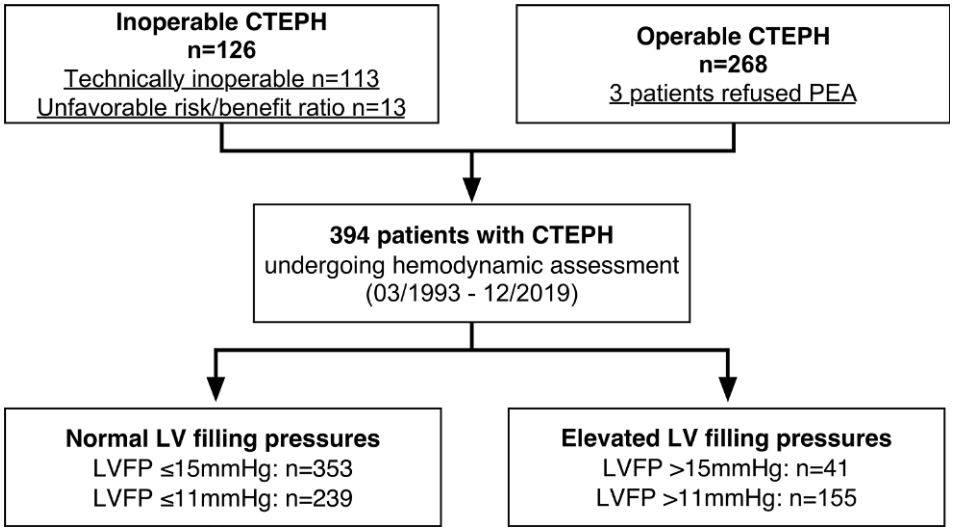

Fig. 1 | 19-6 Patient disposition

19-7

The influence of hydration status on hemodynamics in patients with pulmonary hypertension

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Introduction: Pulmonary Hypertension (PH) is a severe and progressive disease characterized by elevated blood pressure in the pulmonary circulation, with an increase in right ventricular (RV) afterload ultimately causing RV failure and death. Right heart catheterization (RHC) is the gold standard to diagnose PH, and hemodynamic measurements play a critical role to validate the success of treatment. RV failure has been associated with increased total blood volume, venous congestion and systemic fluid retention. We assessed the influence of volume overload and body composition on hemodynamics in patients with PH to further elucidate if hydration status impacts important diagnostic parameters like mean pulmonary arterial pressure (mPAP) or pulmonary vascular resistance (PVR).

Methods: 39 patients who underwent RHC at the Department of Cardiology, Medical University of Vienna were included in this study. Body composition (fluid status as well as fat and muscle content) was measured by bioelectrical impedance analysis using the body composition monitor (BCM, Fresenius

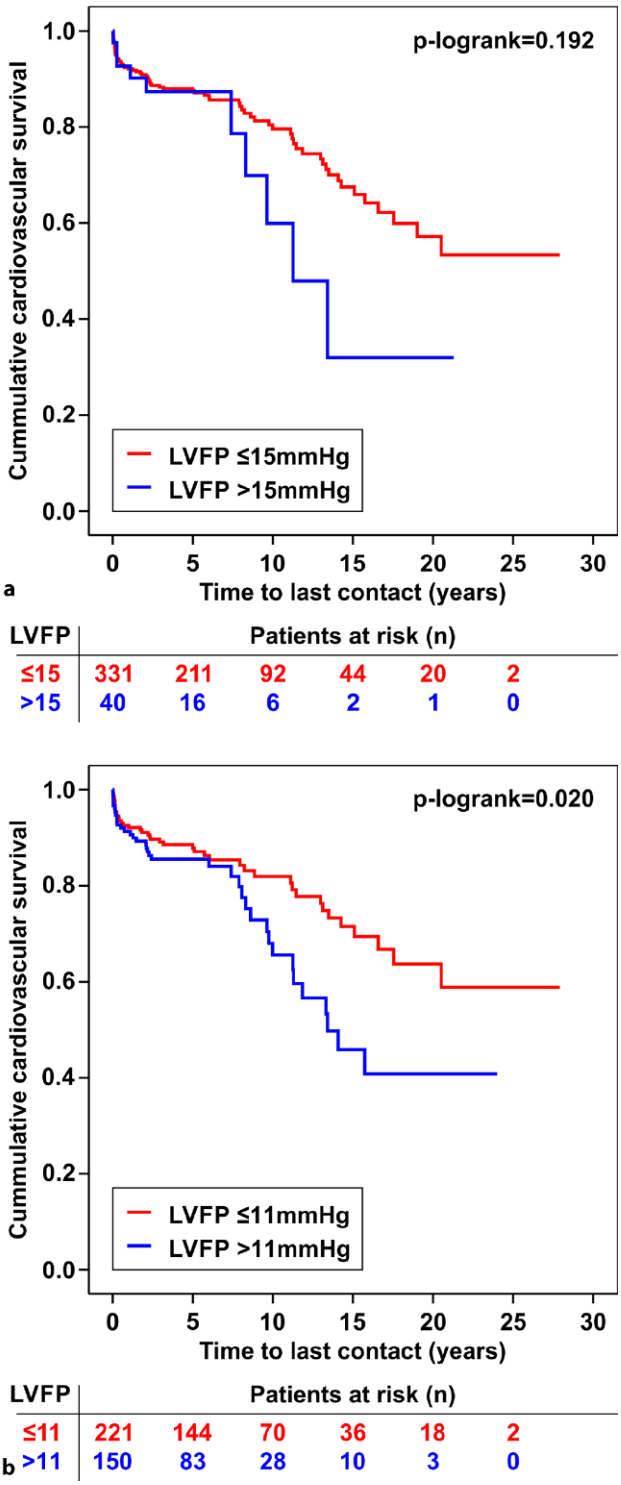

Fig. 2 | 19-6 Survival

Medical Care) immediately before the RHC. Statistical analysis of BCM measurements and RHC data were performed using GraphPad Prism 9.0 (GraphPad Software Inc.) and SPSS 26.0 (IBM). P-values of <0.05 were considered statistically significant.

Results: Patients were 71 years (51–78) old, had a BMI of 25.6 kg/m² (22.1–31.2) and an mPAP of 38.8 ±13.8 mm Hg. Hydration measurement in patients ranged from –8.4 L to +4.0 L, and 46 % of the patients were overhydrated. Fluid overload significantly correlated with enddiastolic RV pressure (rS = 0.3484, $p = 0.0346$). Extracellular water (ECW) significantly correlated with cardiac output (CO; rS = 0.6745, $p \leq 0.0001$). In the multivariate analysis, after adjusting for age and gender, fluid overload and ECW were independent predictors of enddiastolic RV pressure and CO ($p = 0.049$ and $p \leq 0.001$). No significant impact of the hydration status on mPAP or PVR could be observed in patients with PH.

Conclusion: Hydration status significantly impacts RV pressure and CO in patients with PH. In our study population no effect on mPAP or PVR could be observed, however further analysis in a bigger cohort is ongoing.

19-8

Pulmonary arterial hypertension in a patient with transaldolase deficiency–An uncommon case

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Introduction: Transaldolase deficiency is a rare genetic disease, caused by mutation in the transaldolase gene. Currently, 34 patients are known worldwide. Transaldolase is a key enzyme in the pentose phosphate pathway. This pathway provides NADPH und Ribose-5-Phosphat. By that, it maintains the mitochondrial trans-membrane potential and a correct apoptosis [1]. The patients show a wide range of symptoms which can vary in severity. The most frequently mentioned pathologies are hepatosplenomegaly, hydrops fetalis, dysmorphia, liver cirrhosis, haemolytic anaemia and congenital heart disease [2].

Methods: We report on a 35-year-old woman with ASD in childhood, now PFO, unclear hepatosplenomegaly, bicytopenia, ovarial dysgenesis and mild facial dysmorphia since birth. A cause or mechanism cannot be found in frequent hepatologic and haematologic check-ups. At the age of 28, the patient sustains a severe bleeding of oesophageal varices. After recompensation of anaemia the patient shows respiratory distress signs of cardiac decompensation. In echocardiography the patient shows signs of a moderately severe right heart failure with elevated TRPG-Values around 85 mm Hg. So we started immedi-

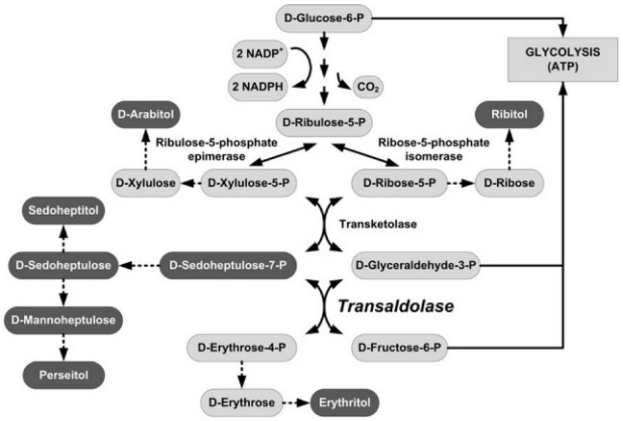

Fig. 1 | 19-8 A schematic representation of the pentose phosphate pathway [4]

Timeline

January 1986	Birth, Detection of ASD II, hepatosplenomegaly of unclear genesis, bicytopenia, ovarian dysgenesis, mild facial dysmorphia
July 1997	Heart catheter without signs of a significant shunt volume
Until 2011	Two bone marrow biopsies – negative
July 2014	Genetic diagnostics of fertility because of primary amenorrhoea and wish for child, biochemical diagnostics of lysosomal storage disease (Mb. Gaucher and Mb. Pompe) – negative
September 2014	Bleeding of oesophageal varices with haemorrhagic shock and endoscopic band ligation
	PRIND with hemiparesis on the left side and a recent ischaemia frontotemporal on the right side (MRI) (caused by a crossed embolism in presence of PFO)
	Remarkable pulmonary hypertension in echocardiography and right-heart-catheter-examination (PA 35/23/29 mmHg, PCW 13/8/9 mmHg, PVR 4,4 Wood units, CO 4,5 l/min)
	PFO in contrast echocardiography
October 2014	MRI of the liver: no signs of a Budd-Chiari-syndrome, known hepatosplenomegaly
	Liver elastography: degree of fibrosis F2-F3 possible, limited interpretation
	HVPG - measurement: no signs of portal hypertension
	Transjugular liver biopsy: histological no sign of liver cirrhosis
November 2014	Head MRI because of hyperprolactinaemia and primary amenorrhoea: no pathological process in the region of the sella turcica
November 2014 to September 2016	Regular cardiac and gastroenterological controls
September 2016	MRI-Angiography and diagnostic angiography: no relevant AV-Shunt in the liver
	Gastroscopy: no new onset oesophageal varices, but signs of hypertensive gastropathy
October 2018	Genetic examination with whole exome sequencing: homozygous mutation of the TALDO1- gene — c.574C>T (p.Arg192Cys)
February 2020	Gastroscopy: no new onset oesophageal varices
Until September 2021	Frequent echocardiographic checkups

Fig. 2 | 19-8 Timeline

ately a therapy with iloprost inhalation and a oral application of macitentan. After stabilisation and under therapy the right heart catheter investigation showed elevated arterial pressure values (PA 35/23/29 mm Hg, PCW 13/8/9 mm Hg, CO 4.5 l/min, PVR 4.4 Wood units) Because of the still unclear hepatosplenomegaly a further evaluation with a transjugular liver biopsy is initiated. The histology is negative, there are no signs of fibrosis. A HPVG-pressure measurement shows normal values without a hint for portal hypertension. After an appropriate observance, the patient is discharged. Regular controls in our cardiac and gastroenterological ambulance are following. Under therapy with Macitentan, the patient is in NYHA level I without any new onset oesophageal varices. Four years later, transaldolase deficiency was diagnosed through a genetic test due to an unfulfilled wish for child.

Results: An accurate clinical phenotyping of this disease, due to rare incidence, is very difficult. We report about this case, to promote this procedure. Most of the unclear symptoms of our patient can be explained by transaldolase deficiency. But following things are uncommon in this case: First, in available literature, there is only one patient described who reached adulthood [3]; however, our patient was 32 years old, when she was diagnosed. Further, this is the first case of transaldolase deficiency in combination with pulmonary arterial hypertension. Although there are clinical signs of portal hypertension in several HVPG-measurements, there are no elevated pressure values. Under therapy with Macitentan, there are satisfying TRPG-values, the patient is in functional class I, new onset oesophageal varices cannot be detected. So, we recommend considering transaldolase deficiency in patients with pulmonary arterial hypertension in combination with unclear hepatosplenomegaly, clinical signs of portal hypertension and congenital heart disease–even when they are elder then 25 years.

Conclusion: So we recommend considering transaldolase deficiency in patients with pulmonary arterial hypertension in combination with unclear hepatosplenomegaly, clinical signs of portal hypertension and congenital heart disease–even when they are elder then 25 years.

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Postersitzung 20 – Risikofaktoren/ Stoffwechsel/Lipide 2

20-1

Value of blood pressure measurement earlier versus later in life to predict cardiovascular mortality

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Introduction: We here aimed at comparing the value of systolic blood pressure (BP) earlier versus later in life to predict cardiovascular mortality.

Methods: In a cardiovascular observation study (OS) we prospectively recorded fatal cardiovascular events over up to 19 years in 1282 patients of whom 570 had the Metabolic Syndrome (MetS) at baseline. These patients had participated in a health survey (HS) 15 years prior to the OS baseline. BP was measured both at the HS and at the baseline of the OS.

Results: We found that the increase in cardiovascular mortality matched the increase of BP in the HS in a linear way but this is not the case for BP assessed at the OS (Fig. 1). A cox regression analysis revealed that each millimeter of mercury (mm Hg) increased the risk for cardiovascular death by 2 % (HR = 1.02 [1.01–1.03], $p < 0.001$). Applying a stratification for the presence of MetS, we found that in both groups BP was a significant predictor of cardiovascular mortality (HRMetS = 1.02 [1.01–1.02], $p < 0.001$ and HRnoMetS = 1.02 [1.01–1.03], $p < 0.001$). In contrast, BP as measured at the baseline of the OS was not significantly associated with cardiovascular death during follow-up neither in the total population nor in any subgroup (HR = 1.00 [0.99–1.01], $p = 0.652$; HRMetS = 1.00 [0.99–1.01], $p = 0.468$ and HRnoMetS = 1.00 [0.99–1.01], $p = 4.66$).

Conclusion: We thus conclude that BP assessed earlier in life is a better predictor of cardiovascular mortality than BP assessed later in life.

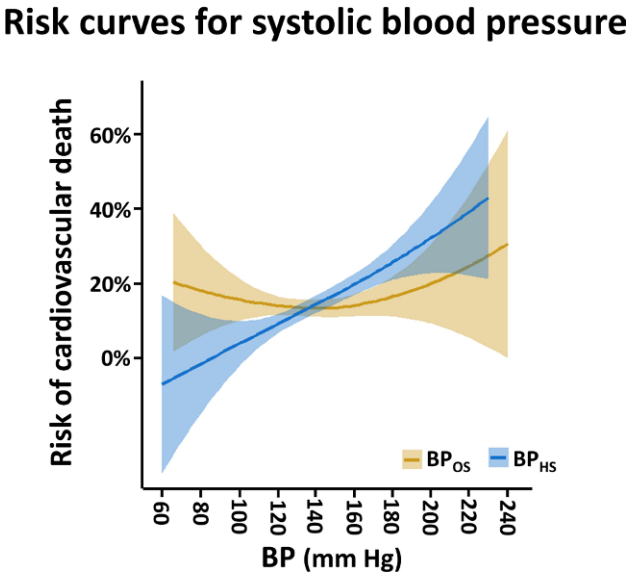

Fig. 1 | 20-1 Risk curves for systolic blood pressure. Risk curves are calculated for blood pressure (BP) assessed at the health survey (HS) and at the baseline of the cardiovascular observation study (OS) according to loess (LOcally WEighted Scatter-plot Smoother) fitting with 95 % confidence intervals for cardiovascular death during follow up.

20-2

Ceramide-based lipid profiles and the prevalence of Type 2 diabetes differ between patients with coronary artery disease and those with peripheral artery disease

Leiherer A[1,2,3], Muendlein A[2,1], Brandtner E[2], Saely CH[1,2,4], Vonbank A[4,1,2], Mader A[4,2], Sprenger L[2,1,4], Maechler M[2,1,4], Larcher B[4,2], Jylha A[5], Laaperi M[5], Laaksonen R[5], Maerz W[6], Fraunberger P[3,1], Kleber M[7], Drexel H[2,1,8,9]

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Introduction: Serum lipids and metabolic diseases, in particular type 2 diabetes (T2D) and non-alcoholic fatty liver disease (NAFLD), predict the atherosclerotic diseases coronary artery disease (CAD) and peripheral arterial disease (PAD). However, it is not known in how far a more detailed characterization including serum lipids improves discrimination of PAD from CAD.

Methods: A cohort of 274 statin-naïve patients with either PAD ($n = 89$) or stable CAD ($n = 185$) were referred to metabolic screening and were characterized using nuclear magnetic resonance- and liquid chromatography-tandem mass spectrometry based advanced lipid and lipoprotein analysis. Results were validated in an independent cohort of 1239 patients with PAD or CAD.

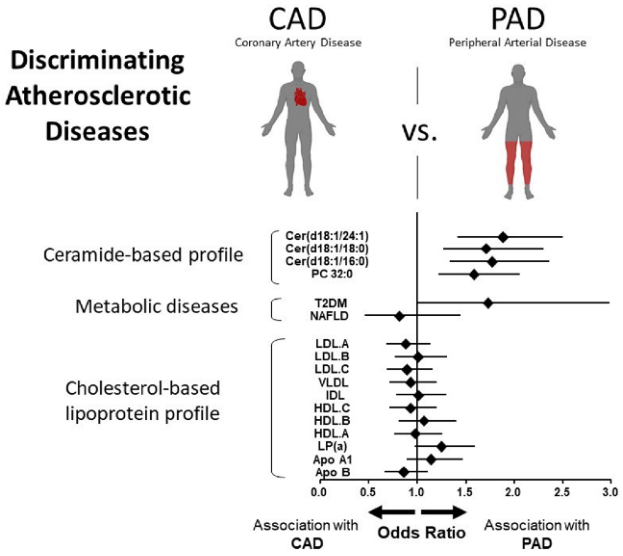

Fig. 1 | 20-2 Association of lipid parameters and metabolic diseases with the prevalence of periphal arterial disease (PAD) instead of coronary artery disease (CAD). The forest plots represent the adjusted odds ratios (OR) of logistic regression for the association of the lipid parameters with the prevalence of PAD (QR > 1) or CAD (OR < 1). The OR is fiven for 1 standard deviation (SD) together with the 95 % confidence interval

Results: We found a significant difference in T2D prevalence and in the ceramide-based lipid profile between PAD and CAD patients. However, neither cholesterol-based markers (including LDL-C, HDL-C) and detailed lipoprotein profiles nor the NAFLD status differed significantly between PAD and CAD patients (Fig. 1). The difference between ceramide-based lipid profiles of CAD and PAD remained significant also after adjusting for body composition, smoking, inflammatory parameters, and T2D.

Conclusion: We conclude that PAD and CAD differ in ceramide-based lipid profiles and T2D status, but not in other lipid characteristics or metabolic diseases.

20-3

Cystatin C predicts major cardiovascular events in patients with coronary artery disease both among patients with Type 2 diabetes and in non-diabetic individuals

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Introduction: Cystatin C is an established biomarker for renal function, and, given the close association of chronic kidney disease and cardiovascular disease might indicate new-onset or deteriorating cardiovascular disease. However, evidence for cystatin C as a predictor of cardiovascular events is limited and controversial. We therefore aimed at investigating the role of Cystatin C as a predictor of future major adverse cardiovascular events (MACE) in a high risk-cohort of patients with coronary artery disease (CAD).

Methods: Cystatin C was measured in 1098 patients with angiographically proven CAD. Vascular events were recorded over a mean follow-up of 8.0 ± 5.0 years.

Results: At baseline, 239 patients had T2 DM and 859 did not have diabetes. During follow-up, 30.0 % of our patients suffered MACE. Cystatin C proved to be a strong and independent predictor of vascular events in the total study cohort (standardized adjusted HR 1.20 [1.12–1.28], $p < 0.001$). When diabetes status was taken into account, cystatin C significantly predicted major cardiovascular events in non T2 DM patients (HR = 1.16 [1.08–1.26], $p < 0.001$) and in patients with T2 DM (HR = 1.34 [1.13–1.60], $p = 0.001$).

Conclusion: We conclude that cystatin C predicts major cardiovascular events in patients with coronary artery disease both among patients with type 2 diabetes and in non-diabetic individuals.

20-4

Type 2 diabetes mellitus and congestive heart failure in women are mutually independent predictors of non-alcoholic fatty liver disease

Maechler M[1,2,3], Sprenger L[1,2,3], Mader A[3,1], Larcher B[3,1], Vonbank A[3,2,1], Leiherer A[2,1,4], Muendlein A[1,2], Drexel H[1,2,5,6], Saely CH[2,1,3]

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Introduction: Non-alcoholic fatty liver disease (NAFLD) is associated with both type 2 diabetes mellitus (T2 DM) and congestive heart failure (CHF), and T2 DM is highly prevalent in CHF patients, in particular among women. However, the single and joint associations of T2 DM and CHF with NAFLD in women have not been investigated yet. This issue therefore is addressed in the present study.

Methods: We investigated 76 female patients with CHF and 321 female controls who did not have signs or symptoms of CHF and in whom significant coronary artery disease was ruled out angiographically. The presence of NAFLD was determined using the validated fatty liver index (FLI).

Results: The prevalence of T2 DM was 39.5 % in women with CHF and 22.1 % in controls ($p = 0.002$). FLI values and prevalence rates of NAFLD (FLI ≥60) in non-CHF women without

T2 DM were 43 ± 28 and 31.6 %, respectively. They were significantly higher in non-CHF, but T2 DM patients (65 ± 28, $p < 0.001$ and 64.8 %, $p < 0.001$, respectively), in CHF patients without T2 DM (67 ± 25, $p < 0.001$ and 63.0 %, $p = 0.002$, respectively) and in CHF patients with T2 DM (66 ± 31, $p < 0.001$ and 66.7 %, $p < 0.001$, respectively). In multivariate analysis of covariance, T2 DM and CHF proved to be mutually independent predictors of FLI after adjustment for age, sex, BMI, LDL-C, history of smoking and hypertension and use of statins ($F = 7.25$; $p = 0.007$ and $F = 46.89$; $p < 0.001$, respectively); concordantly, T2 DM and CHF independently predicted the presence of NAFLD in logistic regression analyses, with adjusted odds ratios of 3.22 [1.38–7.50]; $p = 0.007$ and 21.71 [6.58–71.59]; $p < 0.001$, respectively.

Conclusion: We conclude that CHF and T2 DM are mutually independent predictors of NAFLD in women.

20-5

Remnant cholesterol predicts major cardiovascular events in patients with coronary artery disease both among patients with Type 2 diabetes and in non-diabetic individuals

Mader A[1,2], Sprenger L[2,3,1], Vonbank A[1,3,2], Larcher B[1,2], Maechler M[2,3,1], Leiherer A[3,2,4], Muendlein A[2,3], Drexel H[2,3,5,6], Saely CH[3,2,1]

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Introduction: Remnant cholesterol, which is calculated as total cholesterol minus LDL cholesterol minus HDL cholesterol has attracted interest as a marker of cardiovascular event risk. The power of remnant cholesterol to predict major cardiovascular events (MACE in patients with established coronary artery disease is unclear and is addressed in the present study).

Methods: We enrolled 1472 consecutive patients with established coronary artery disease. Prospectively, cardiovascular events were recorded over a mean follow-up period of 8.0 ± 5.03 years.

Results: At baseline, remnant cholesterol was significantly higher in patients with T2 DM ($n = 446$) than in non-diabetic subjects (27 ± 24 vs. 21 ± 23 mg/dl; $p < 0.001$). During follow-up, 493 of our patients suffered MACE; the event rate was significantly higher in patients with T2 DM than in non-diabetic subjects (62.5 vs. 37.5 %; $p < 0.001$). Remnant cholesterol in Cox regression models adjusting for age, sex, hypertension, smoking, body mass index and LDL cholesterol independently predicted MACE in the total study population (standardized adjusted HR 1.17 [1.09–1.28], $p < 0.001$), and in patients with T2 DM as well as in non-diabetic subjects (standardized adjusted HRs 1.24 [1.07–1.44], $p = 0.005$ and 1.14 [1.02–1.26], $p = 0.017$, respectively).

Conclusion: From our data we conclude that remnant cholesterol in patients with established coronary artery disease predicts MACE both among patients with T2 DM and among non-diabetic subjects.

20-6

The LIPL Study Lipid panels and platelet activity in coronary heart disease

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Introduction: Measuring lipid panel in the fasting state can be inconvenient for patients and may aggravate their compliance. Moreover, people spend most of the day in the nonfasting state. Hence, it suggests that the changes in the process of atherosclerosis happen mainly under the influence of nonfasting lipids [1–3]. Up to date, the studies in the postprandial state were primarily performed in healthy subjects. This exploratory, cross-sectional study investigates the change in lipid profile and platelet activity in patients with different cardiovascular risk profiles in the postprandial state.

Methods: The studied population consists of 66 patients with different cardiovascular risks: patients with coronary artery disease (CAD) and diabetes mellitus type 2 (DM2) ($n = 20$), CAD without DM2 ($n = 25$), and a healthy control group ($n = 21$). Lipid variables and markers of platelet function were assessed during the fasting state (baseline) and 3 and 5 h after a standardized fat meal using a standardized oral fat tolerance test (OFTT), a milkshake with 90 g of fat. The platelet activity was measured with a Multiplate test using ADP, ASPI and TRAP reagents.

Results: Patients with CAD and DM2 were significantly older and had the highest BMI. All patients with CAD were on acetylsalicylic acid, and 95 % were on high-dose statins. Total cholesterol, LDL-c, Apolipoprotein A1, and Apolipoprotein B did not change during the OFTT, irrespective of the group. HDL-c decreased statistically significantly three and five hours after the fat loading with a peak after five hours (3.46 ± 0.4 mg/dL., $p < 0.001$). Triglycerides (TG) increased significantly during the OFTT, with a peak after 5 h (130.2 ± 14.5 mg/dL., $p < 0.001$) irrespective of the group. There was no difference in TG concentration between the groups. All differences stayed statistically significant after adjustment for age and BMI. Platelet activity increased, as shown by a significantly increased thrombocyte count after three hours ($p < 0.001$) and increased platelet activity measured by multiplate test with ADP (7.16 ± 2.17 AU, $p = 0.005$), ASPI (4.60 ± 1.40 AU, $p = 0.005$), and TRAP (11.41 ± 3.10 AU, $p = 0.001$) reagents three hours after the fat loading. The platelet activity measured by all three reagents remained increased even after the adjustment for age and BMI (ADP: 7.14 ± 2.10 AU, $p = 0.004$, ASPI: 4.60 ± 1.40 AU, $p = 0.006$, TRAP: 11.40 ± 3.10 AU, $p = 0.002$). Moreover, the platelet activity measured by ADP (–13.12 ± 4.93, $p = 0.030$), and TRAP (–14.6 ± 15.51, $p = 0.031$) reagents was lower in the control group. This difference was not statistically significant after adjusting for age and BMI.

Conclusion: This study showed that fatty meal causes worsening of lipid profile and leads to increased platelet activity in subjects irrespective of cardiovascular risk profile.

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20-7

Pilot Study: The LIPL-PLATELET Study LIPid paneLs And PLATELET activity in coronary heart disease

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Introduction: Introduction: Statins represent the main group within lipid-lowering therapy and are known to exert pleiotropic effects that might be caused by LDL-C reduction and/or direct influence of the lipid-lowering agent [1–3]. Whether PCSK9-I bear a pleiotropic potential is unclear. This hypothesis-generating study aimed to investigate the change in the lipid profile and its potential influence on platelet reactivity after standardized oral fat tolerance testing (OFTT) under an optimized lipid-lowering therapy (combination of statin plus ezetimibe) alone or after a 3-months treatment period with the pro-protein convertase subtilisin/Kexin type 9 inhibitor (PCSK9-I), alirocumab.

Methods: In this pilot project we investigated ten patients with chronic coronary syndrome (CCS) and hyperlipidaemia with an indication for a PCSK9-I. Lipid variables and markers of platelet function were assessed during the fasting state (baseline) and 3 and 5 h after a standardized fat meal by use of a standartized OFTT using a milkshake with 90 g of fat. Measurements were performed in the same CCS patients under dual lipid lowering therapy alone and after three months of therapy with alirocumab.

Results: The mean age of the population was 58.9 (±12.13) years, and 80 % of the population were males. All CCS patients were on acetylsalicylic acid and P2Y12 inhibitors during the whole course of the study. OFTT caused a statistically significant increase of triglycerides ($p < 0.001$). All other parameters of the lipid profile remained unchanged after fat loading. However, there was a statistically significant decrease in total cholesterol ($p < 0.001$), non-HDL cholesterol ($p < 0.001$), and apolipoprotein B ($p < 0.001$) after initiation of PCSK9-I. Postprandial inflammatory reaction after OFTT was reflected by a statistically significant increase of leucocyte ($p = 0.002$) and neutrophil counts ($p < 0.001$). There was no difference in OFTT induced postprandial inflammation after treatment with alirocumab. The multiplate electrode aggregometry (MEA) test with ADP ($p = 0.002$) and ASPI reagents ($p = 0.002$) showed a paradoxically increased platelet reactivity 5 h after OFTT only in patients on PCSK9-I. However, platelet reactivity remained unchanged during OFTT in CCS patient before or after alirocumab therapy.

Conclusion: Alirocumab significantly improved the lipid profile in CCS patients and led by trend to decreased postprandial inflammation. This finding is of potential interest but deserves further investigation as also the unexpected increase in platelet reactivity five hours after OFTT.

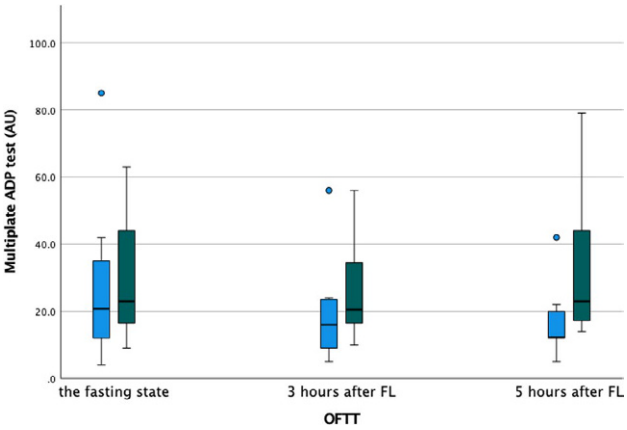

Fig. 1 | 20-7 Platelet activity measured by multiplate ADPtest during the oral fat tolerance test. b Boxplots showing mean values and error bars (AU aggregation units, OFTT oral fat tolerance test, FL fat loading, Blue boxplot at baseline, no therapy with PCSK9-inhibitors, and solid line connecting each time point during the OFTT, Green boxplot after three months of therapy with PCSK9-inhibitors, and dashed line connecting each time point during the OFTT)

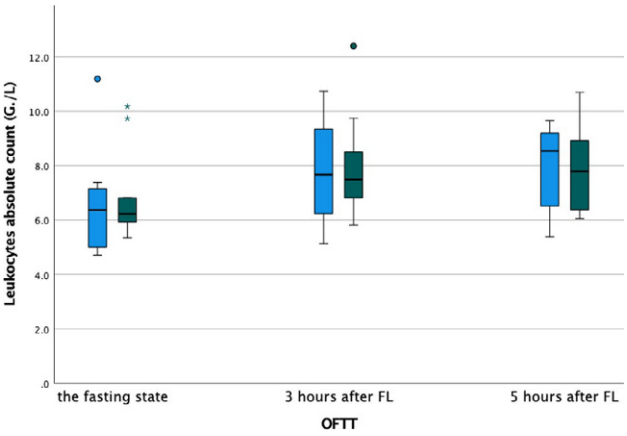

Fig. 2 | 20-7 Absolute leukocyte count during the oral fat tolerance test. b Boxplots showing mean values and error bars (OFTT oral fat tolerance test, FL fat loading, Blue boxplot at baseline, no therapy with PCSK9-inhibitors, and solid line connecting each time point during the OFTT, Green boxplot after three months of therapy with PCSK9-inhibitors, and dashed line connecting each time point during the OFTT)

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Postersitzung 21 – Rhythmologie 3

21-1

Healthcare utilization, employment, and all-cause mortality in atrial fibrillation patients treated by drug therapy versus catheter ablation

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Introduction: Atrial fibrillation (AF) is the most prevalent arrhythmia, associated with increased mortality and morbidity and causing relevant hospitalization rates per year. Its impact on healthcare expenditure is approximately 1 % in western countries. Treatment options for symptomatic AF consist of rate and rhythm control drugs (non-PVI) as well as catheter ablation of the pulmonary veins (PVI). This method is recommended in the current guidelines of the European Society of Cardiology for drug-refractory AF or as first line therapy at patient's preference. Published health economic data on the impact of PVI mainly consist of model assumptions. Direct comparisons of actual expenditures, labour market force, and mortality between drug therapy and PVI are scarce.

Methods: We analyze effective healthcare expenditures, labour market data, as well as mortality and morbidity based on inpatient and outpatient data from the Upper Austrian Health Insurance Fund social security system. The data on patients with a first hospitalization for AF in the years 2005 to 2018 were examined. Propensity score matching (PSM) using all CHADS2-VA2Sc variables and working collar for the socio-economic status was used to create comparable groups.

Results: Out of 21,791 patients identified by their first hospitalization for AF 1624 (7.4 %) were treated with at least one PVI. PSM identified 1013 well-matching pairs (non-PVI and PVI) for a total of 2026 AF patients. In short-term over 2 years both, in- and outpatient expenditure were significantly higher in the PVI group with a total increase of € 4100 per year, including medication. Most of this cost excess is gained by the PVI procedures during this period which also creates significantly higher hospital days (1.6 days per year). Long-term data up to 10 years still reveal a significantly higher health care utilization concerning hospital days, inpatient as well as outpatient costs. PVI patients utilize € 2200 more per year then non-PVI patients. A positive effect of PVI is seen in a significantly higher employment rate (+5.1 %), due to reduced retirements (–7.6 %), which is a highly relevant factor concerning economic impact of an intervention. Sick leave days are roughly 3 days more per year in PVI patients. Utmost important is the 5.8 % all-cause mortality reduction over 10 years in PVI patients with most difference in the first 5 years.

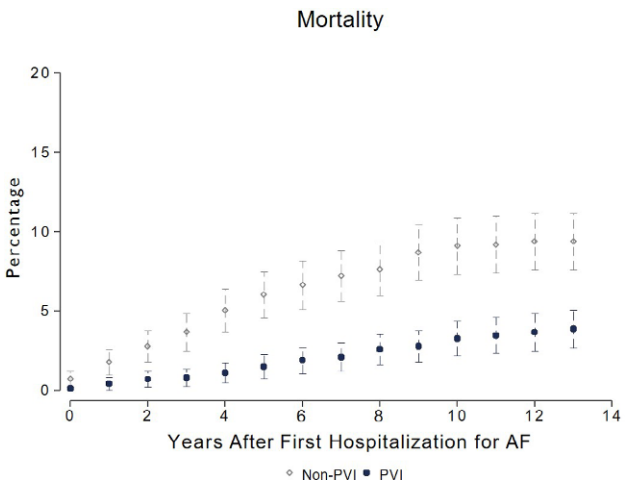

Fig. 1 | 21-1

	Ø Non-PVI	Ø PVI	Diff.	p-value	95% CI
Health Care Utilisation					
Hospital Days	4.592	5.421	0.829***	0.000	[0.430, 1.228]
LKF Points	2,381.206	4,014.588	1,633.382***	0.000	[1,350.966, 1,915.798]
LKF Turnover	3,121.271	5,297.225	2,175.954***	0.000	[1,806.870, 2,545.039]
Drug Expenditure	706.593	682.043	-24.550	0.550	[-105.048, 55.948]
Outpatient Medical Care	630.563	674.044	43.480***	0.001	[18.350, 68.611]
Mortality					
All-cause Mortality	0.091	0.033	-0.058***	0.000	[-0.079, -0.037]
In-Hospital Death	0.674	0.564	-0.110	0.244	[-0.295, 0.075]
Cardiovascular Death	0.344	0.545	0.202	0.105	[-0.043, 0.446]
Neoplasm Death	0.359	0.318	-0.041	0.727	[-0.275, 0.192]
Other Death Cause	0.312	0.182	-0.131	0.203	[-0.333, 0.072]
Pacemakers and Cardioversion					
Pacemaker	0.028	0.052	0.025***	0.005	[0.008, 0.042]
Implantable Cardioverter-Defibrillator (ICD)	0.022	0.014	-0.008	0.179	[-0.019, 0.004]
Cardioversion	0.333	0.522	0.190***	0.000	[0.147, 0.232]
Labour Market Outcomes					
Employed	0.708	0.759	0.051***	0.009	[0.013, 0.090]
Unemployed	0.150	0.139	-0.011	0.496	[-0.042, 0.020]
Retired	0.568	0.492	-0.076***	0.001	[-0.119, -0.032]
Blue Collar	0.408	0.408	0.001	0.982	[-0.051, 0.052]
Sick Leave Days	14.881	17.895	3.014***	0.000	[1.333, 4.695]
Risk Factor Diagnoses					
Heart Failure	0.125	0.102	-0.024*	0.093	[-0.051, 0.004]
Hypertension	0.314	0.309	-0.005	0.811	[-0.045, 0.035]
Diabetes	0.088	0.075	-0.013	0.291	[-0.037, 0.011]
TIA/Stroke	0.054	0.044	-0.010	0.305	[-0.029, 0.009]
Coronary Heart Disease	0.192	0.256	0.064***	0.001	[0.028, 0.100]
Peripheral Artery Disease	0.026	0.011	-0.015**	0.013	[-0.026, -0.003]
Hyperlipidemia	0.169	0.280	0.112***	0.000	[0.075, 0.148]
Renal Failure	0.018	0.016	-0.002	0.730	[-0.013, 0.009]
Dementia	0.004	0.001	-0.003	0.179	[-0.007, 0.001]
Relevant Medication					
Prescription Probability					
Anticoagulants (B01*)	0.716	0.954	0.238***	0.000	[0.207, 0.269]
Antiarrhythmic (C01*)	0.497	0.777	0.280***	0.000	[0.240, 0.320]
Antihypertensive (C02*)	0.110	0.100	-0.010	0.468	[-0.037, 0.017]
Antidiabetic (A10*)	0.134	0.099	-0.036**	0.013	[-0.063, -0.008]
Lipid-Lowering Drugs (C10*)	0.399	0.471	0.072***	0.001	[0.029, 0.115]

Fig. 2 | 21-1

Conclusion: Analyzing a cohort of 2026 PSM AF patients comparing drug therapy vs PVI, we found significantly higher in- and outpatient expenditure including medication in short-term. Most of this cost excess is produced by the PVI procedures during this period. Long-term data over 10 years still show higher health care utilization in PVI patients concerning hospital days, inpatient as well as outpatient costs. The benefit of PVI is seen in significantly higher employment status in the PVI group, which is crucial for the gross economic benefit. Most important we can show a significant reduction in all-cause mortality in PVI patients.

21-2

Single-center outcome after ablation of atrial fibrillation using very high-power short duration pulmonary vein isolation

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Introduction: Catheter ablation of atrial fibrillation is (AF) an established second line therapy for patients with symptomatic paroxysmal (PAF) and persistent AF (persAF). Novel abla-

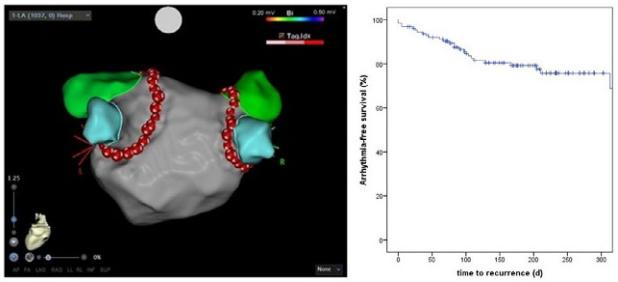

Fig. 1 | 21-2 Left panel: Sample image of a vHPSD-PVI, posterior view of the left atrium. Right panel: Single procedure arrhythmia-free survival after vHPSD-PVI

tion catheters with integrated thermocouples allow fast application of radiofrequency lesions with powers up to 90 W. We aimed to describe primary and secondary outcomes after very high-power short duration (vHPSD) ablation.

Methods: 126 consecutive patients (78 PAF, 43 persAF, 5 longstanding persistent AF) underwent pulmonary vein isolation (PVI) using the QDOT Micro Catheter (Biosense Webster) with the ablation mode QMODE+ (90 W, 4 s, interlesion distance ≤4 mm anterior, ≤6 mm posterior).

Results: Mean age was 62 ±9 years, 33 % were female, median CHA2DS2-VASc Score was 2 (0, 7). Median follow up duration was 204 (14, 461) days. 30 % of patients had additional ablation of typical right atrial flutter. Primary success rate to achieve pulmonary vein isolation was achieved in all patients, no catheter-related complications (e. g., charring, steam pop) occurred. First pass isolation of all 4 PVs was achieved in 48 % of patients, re-ablations were necessary in the carina regions (right: 37 % of cases, left: 29 %) and ridge (14 %). Median procedure for PVI only were 102 (45–210) minutes. Arrhythmia-free survival was 79.6 % (see Fig. 1). Eight patients underwent re-do procedures during follow-up showing most commonly showing gaps in the right inferior PV (63 %) and ridge (50 %).

Conclusion: Very high-power short duration ablation allows safe and quick pulmonary vein isolation. However, first pass isolation rate is low due to gaps in the carina regions. Arrhythmia-free survival is comparable to other pulmonary vein isolation techniques.

21-3

Photoplethysmography telemonitoring during the first week after atrial fibrillation ablation: Feasibility and clinical implications

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Introduction: The incidence of early atrial fibrillation (AF) recurrence within the first week after AF ablation and its predictive value for late AF recurrences are unclear. TeleCheck-AF is a remote on-demand mobile health (mHealth) infrastructure,

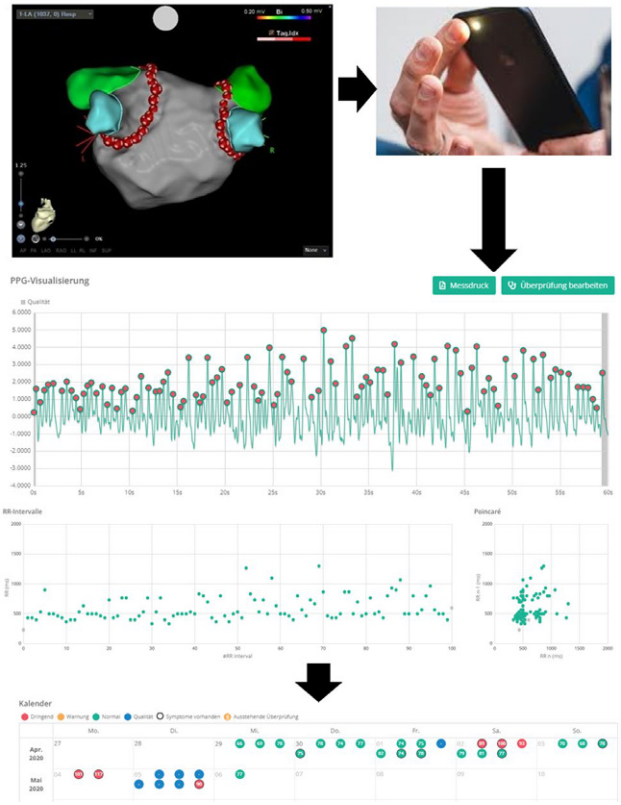

Fig. 1 | 21-3 Schematic overview of the telemonitoring process. After the ablation, the patient measures PPGs for one week, dashboard view for clinician shows regular rhythm (*green*), atrial fibrillation (*red*) and unclear tracings (*blue*)

which is based on a mobile phone app using photoplethysmography (PPG) technology (Fibricheck) allowing rate and rhythm monitoring through teleconsultations. The feasibility and clinical implications of PPG telemonitoring specifically during the first week after atrial fibrillation ablation is unknown

Methods: Within the TeleCheck-AF project, the University Hospital Graz offered a total of 382 consecutive patients undergoing AF ablation (between June 1st 2020 and December 15th 2021) photoplethysmography (PPG) telemonitoring with "FibriCheck" during the first week after the ablation procedure. Patients received a QR code for activation of the software on their smartphone and were connected to the clinician's telemedicine portal. They were instructed to perform rhythm monitoring three times per day and in case of symptoms. Clinicians assessed the tracings and contacted the patients if therapeutic steps were indicated.

Results: In total, 119 patients (31 %) agreed to perform telemonitoring after ablation. Patients undergoing telemonitoring were younger compared to those who visited the clinic/did not? (58 ± 10 years vs. 62 ± 10 years, $p < 0.001$). 34 % were female, median CHA2DS2-VASc-Score was 1 (0–6). 62 % of patients had paroxysmal AF and 37 % had persistent AF. One of four patients (24 %) had already undergone previous ablations. Most index ablations were radiofrequency ablations (89 %; 7 % cryo; 4 % pulsed field ablation). Median follow up duration was 281 (16–620) days. 27 % of patients had tracings suggestive of AF in the week following the index ablation. Telemonitoring resulted in clinical interventions ins 24 % of patients: amiodarone was started in 8 %, class I antiarrhythmic drugs were up titrated in 7 %, cardioversion was scheduled in 5 %, antiarrhythmic drugs were reduced due to symptomatic bradycardia in 3 % of patients. During follow-up, 22 % of patients had ECG-documented AF

recurrences. PPG recordings suggestive of AF in the week after ablation were predictive of late recurrences ($p < 0.001$).

Conclusion: Rhythm monitoring with a PPG-based mHealth application was feasible and often resulted in clinical interventions. Due to its high availability, PPG-based follow-up actively involving patients after AF ablation may close a diagnostic and prognostic gap and increase active patient-involvement.

21-4

Cardiac relapse of extranodal NK/T cell lymphoma manifesting as incessant ventricular tachycardia

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Introduction: Cardiac tumours are rare, but patients may present with symptoms well known from common cardiac diseases like heart failure, arrhythmias, or embolic complications. We report one of the first cases of a cardiac metastasis from an ENKTL-NT presenting with an incessant ventricular tachycardia. A 39-year-old man presented at the general practitioner for a routine follow-up due to the history of an extranodal NK/T Cell Lymphoma of the nasal type (ENKTL-NT) in clinical remission. The physical examination detected the presence of a tachycardia. The electrocardiogram showed a rhythmic broad complex tachycardia interpreted as a hemodynamically tolerated ventricular tachycardia (VT, Fig. 1). An immediate transfer to our emergency department was organized. As the patient arrived with the still ongoing VT, the hemodynamic situation deteriorated towards cardiogenic shock with the need for electrical cardioversion.

Methods: A single synchronized shock with 100 J was not successful and induced ventricular fibrillation (VF) followed by cardiopulmonary resuscitation. After three successful defibrillations the VF converted into an instable sinus rhythm degenerating again into a hemodynamically tolerated VT. An intravenous antiarrhythmic therapy with ajmaline, landiolol, and electrolyte substitution stabilized the ventricular rate at 120–150 beats per minute with only short periods of sinus rhythm. Transthoracic echocardiography revealed a hyperechogenic zone of 3 × 2 cm in the apex of the left ventricle of unknown aetiology as well as a hypokinesia in this region and a small pericardial effusion. Coronary artery disease as an ischemic cause of the rhythmic instability was ruled out by coronary angiography. Therefore urgent electrophysiological examination with VT-ablation was discussed but rejected due to the apical tumour. Intensification of the antiarrhythmic therapy by adding intravenous amiodarone established a bradycardic sinus rhythm with short interruptions caused by VTs and even VFs with the need of recurrent delivery of electrical shocks, which led us to establish intermittent overdrive pacing with temporary transvenous pacemaker.

Results: After having excluded ischaemic cause, further cardiac imaging (cMR, PET-CT) was planned. Cardiac magnetic resonance imaging revealed in line with the echocardiography a 5.5 × 2.5 × 3.5 cm sized tumour (Fig. 1) and PET-CT scan showed increased tracer uptake in the apical region, both highly suspicious of a metastatic relapse of the ENKTL. Histological workup of an endomyocardial biopsy of the left ventricular apical region confirmed this diagnosis. An immediately chemotherapy was performed consisting of a six-day run-in phase with dexamethasone followed by methotrexate and PEG-Asparaginase. After initiation of this therapy, no more rhythmic events were detectable and the antiarrhythmic therapy regimen could be deescalated to low-dose bisoprolol and amiodarone peroral.

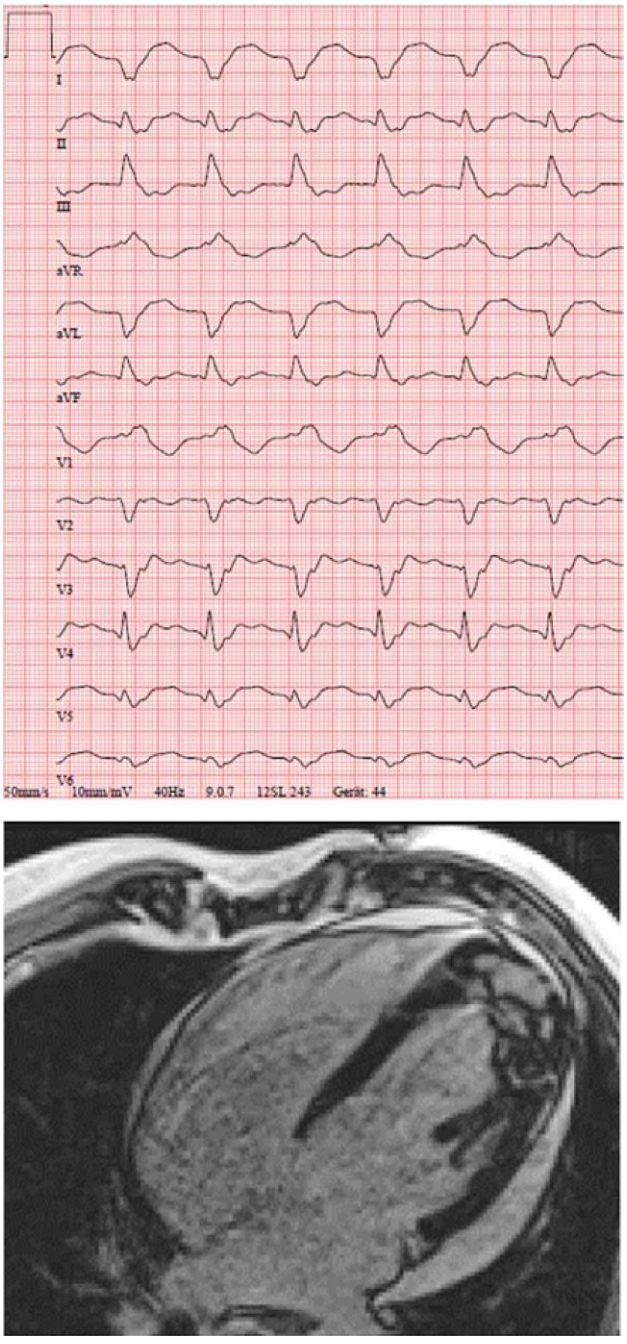

Fig. 1 | 21-4

Conclusion: After 18 days at CCU and having initiated the chemotherapy successfully the patient could be transferred to the haematology ward with stable sinus rhythm. To prevent sudden cardiac death the patient was supplied with a wearable defibrillator and left the hospital three days later. Three months later, an MRI, showing no left ventricular scarring, and an EP study, demonstrating no inducible ventricular arrhythmia, were successfully performed. This case empasises that cardiac metastases may cause acute and severe arrhythmias, especially in young patients without obvious cardiovascular risk factors and with a recent history of malignancy.

21-5

Vergleich unterschiedlicher Ablationstechniken bei Patienten mit persistierendem Vorhofflimmern

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Einleitung: Die linksatriale Katheterablation ist eine hochwirksame Behandlung für Patient*innen mit paroxysmalem und persistierendem Vorhofflimmern (VHF). In den meisten Zentren werden linksatriale lineare Ablationslinien (LARL) und eine Ablation des rechtsatrialen Isthmus (CTI) zusätzlich zur Pulmonalvenenisolation (PVI) durchgeführt, um den Ablationserfolg bei persistierendem Vorhofflimmern zu verbessern. In dieser Studie wurden die Langzeit-Ergebnisse der Patient*innen, die mittels PVI und/oder LARL behandelt wurden, verglichen.

Methoden: 141 konsekutive Patient*innen, die an unserem Zentrum zwischen 2016 und 2020 einer Ablation von persistierendem VHF unterzogen wurden, wurden im Österreichischen Ablationsregister identifiziert und in die retrospektive Studie eingeschlossen. Insgesamt erhielten 60/141 (43 %) Patient*innen eine PVI, 47/141 (33 %) eine PVI plus CTI, 29/141 (21 %) eine PVI+ CTI+LARL und 5/141 (4 %) wurden einer PVI+LARL unterzogen. Die Nachbeobachtungsdauer betrug zwischen 1 und 5 Jahre mit einem von Mittelwert von 3 Jahren. Die primären Endpunkte waren die Freiheit von Vorhofflimmern in seriellen 12-Kanal-EKGs und 7-tägigen Holter EKGs, Hospitalisierungen wegen VHF, Re-Ablationen sowie das subjektive Wohlbefinden der Patient*innen nach einem blanking-Zeitraum von sechs Monaten nach dem Eingriff.

Resultate: Insgesamt zeigten 116/141 (82 %) der Patient*innen kein Rezidiv von VHF in der Nachbeobachtungszeit. In den verschiedenen Gruppen betrug die Freiheit von VHF 48/60 (80 %) bei alleiniger PVI, 39/47 (83 %) bei PVI+CTI, 25/29 (86 %) bei PVI+CTI+LARL und 4/5 (80 %) bei PVI+LARL. Die Rehospitalisierungsraten waren in der PVI+LARL Gruppe am niedrigsten (20 %) Die höchste Rate zeigte sich in der PVI+CTI+LARL-Gruppe (45 %), im Vergleich dazu Patient*innen mit alleiniger PVI (37 %) befinden sich im Mittelfeld. Kardioversionen mussten seltener in der PVI+CTI Gruppe (9 %), jedoch auch häufiger in der PVI+CTI+LARL (34 %) Gruppe durchgeführt werden. Ebenso zeigt sich hier die PVI Gruppe im Mittelfeld (23 %). Re-Ablationen wurden allerdings häufiger in der PVI+CTI-Gruppe durchgeführt (34 %) als in der PVI+LARL-Gruppe (20 %). Die niedrigste Rate zeigte sich bei den Patient*innen in der Gruppe mit alleiniger PVI (13 %). Bei allen 141 Ablationen traten 3 Komplikationen auf, diese wurden allesamt in der PVI-Gruppe beobachtet. Zwei Patient*innen entwickelten eine Perikardtamponade und bei einem Patient*innen wurde ein Aneurysma spurium festgestellt.

Schlussfolgerungen: Die vorliegenden Daten zeigen, dass LARL und CTI zusätzlich zur PVI den klinischen Erfolg der Katheterablation bei Patient*innen mit persistierendem Vorhofflimmern erhöhen. Zusätzliche LARL und CTI waren mit einer erhöhten Rate an Re-Hospitalisierungen, Kardioversionen und Re-Ablationen, aber nicht mit vermehrten Komplikationen verbunden.

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Hemondynamic and rhythmologic effect of push-dose landiolol in critical care patients–A retrospective cross-sectional study

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Introduction: Landiolol as a highly cardioselective ultrashort acting β1-blocker has only been sparsely used as a bolus formulation in critical care patients so far [1, 2]. Therefore, the hemodynamic and rhythmologic effects of push-dose landiolol in critical care are yet to be fully evaluated.

Methods: We retrospectively included patients with non-compensatory supraventricular tachycardia treated with push-dose landiolol at an intensive care unit (ICU) in Vienna, Austria. Hemodynamic data was derived from invasive blood pressure monitoring.

Results: Thirty patients (63 [55–72] years) with sudden onset of non-compensatory supraventricular tachycardia were investigated. These patients had received 49 bolus landiolol applications (7 [6–13] mg; 22 rhythmic and 27 arrhythmic). Successful rate control was accomplished in 20 (40.8 %) cases, rhythm control was achieved in 13 (26.5 %) episodes, and 16 (32.7 %) applications showed no effect. The heart rate was significantly lower after the application (145 [130–150] vs. 105 [100–125] bpm, $p < 0.001$) in a 90-minute observational period. While the systolic blood pressure slightly declined by 4.6 % (135 [123–156] vs. 132 [106–152] mm Hg), no clinically relevant drop in hemodynamics, no other adverse events were observed.

Conclusion: Push-dose landiolol was safe in critically-ill ICU patients without significant hemodynamic effects. An algorithm for bolus application landiolol in critically-ill and emergency department patients could be implemented in the in- and prehospital setting after further investigation.

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