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Abstracts

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BEST ABSTRACTS – VORTRÄGE – BASIC SCIENCE

BA Basic – Vortrag

SIRT4 deficiency protects the heart from ischemia reperfusion injury

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Introduction: Mitochondrial dysfunction causally contributes to myocardial ischemia reperfusion (IR) injury, including defects in electron transport, increased opening of the mitochondrial permeability transition pore (mPTP), and increased ROS generation, including succinate-driven ROS production via reverse electron transport. Sirtuin 4 (SIRT4) is a mitochondrial NAD+-dependent deacylase which suppresses energy metabolism and increases ROS generation. Since mitochondrial NAD+ depletion during IR may compromise mitochondrial sirtuin activity, we aimed to evaluate the role of SIRT4 in the pathogenesis of myocardial IR injury.

Methods: Mice with deletion (SIRT4–/–) or cardiomyocyte selective overexpression of SIRT4 (SIRT4tg) were investigated. IR injury was evaluated in vivo using transient LAD ligation, and ex vivo using Langendorff perfusion. ATP synthesis was measured using bioluminescence, mPTP opening was evaluated by measuring calcium retention capacity, and metabolite profiling was performed using MS/MS-based metabolomics. Oxidative stress was evaluated by quantifying 4-hydroxy-2-nonenal (4-HNE)-modified proteins. Gene expression was measured using RT-PCR.

Results: Cardiac function was unchanged between wildtype (WT) and SIRT4–/– mice using echocardiography and Langendorff perfusions. However, cardiac infarct size was markedly reduced (–50 %; $p < 0.05$) in SIRT4–/– mice following transient LAD ligation, and recovery of developed pressure following global no-flow ischemia in the Langendorff model was also improved by 45 % ($p < 0.05$). While mitochondrial ATP synthesis and calcium retention capacity were mainly unchanged between groups following IR, the IR-induced increase in mitochondrial 4-HNE levels in WT mice was completely blunted in SIRT4–/– mice (–50 %; $p < 0.05$). Metabolomics revealed decreased accumulation of succinate and depletion of several TCA cycle intermediates, and restoration of succinate and other TCA cycle intermediates using treatment with epigallocatechin gallate prevented cardioprotection in SIRT4–/– mice. Furthermore, SIRT4–/– hearts displayed concerted upregulation of genes encoding for mitochondrial antioxidant proteins. In contrast, infarct size following transient LAD ligation was unchanged in SIRT4tg mice compared to WT mice.

Conclusion: Lack of SIRT4 protects the heart from IR injury, possibly by prevention of mitochondrial oxidative stress via attenuation of succinate-mediated ROS production and increased detoxification of ROS. Suppression of SIRT4 activity may represent a promising therapeutic strategy to attenuate myocardial IR injury.

BA Basic – Vortrag

Proteomic and lipidomic profile of neutrophil extracellular traps

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Introduction: Neutrophil extracellular traps (NETs) are formed by decondensed chromatin, histones, and neutrophil granular proteins and were found to be critically involved in various diseases. Although NETs formation and degradation has been intensively investigated, the underlying mechanisms of NETosis are still largely unknown. Therefore, the aim of this study was to use advanced state-of-the-art proteomic and metabolomic techniques to understand the underlying mechanisms of NETosis in more detail.

Methods: NET formation was initiated in polymorphonuclear cells, isolated from healthy donors, by ionomycin. Nuclear extract, cytoplasm and secretome fractions were collected at different stages of NETosis. Specimens were digested filter assisted in solution and shotgun liquid chromatography mass spectrometry analysis were performed. Furthermore, metabolites of the secretome were extracted on solid-phase columns and analyzed with Vanquish coupled high-field orbitrap.

Results: Sixteen intracellular neutrophil proteins were found significantly upregulated during NET formation and were distinguished between their location either in the nucleus or the cytoplasm compartment. Furthermore, we identified 11 proteins and 8 metabolites that were expelled into the extracellular space during NETosis. Strong alterations in several cell compartments were detected in the expression of neutrophil gelatinase-associated lipocalin (NGAL, see Fig. 1) and matrix-metalloproteinase-9. In addition, the metabolites hepoxilin B3, 12-epi-LTB4 and 6-trans-LTB4 show strong changes of their expression levels in the extracellular space.

Conclusion: The present study identified the expression of novel proteins and metabolites during NETosis. These molecules could lead to a better understanding of fundamental NET formation mechanisms and may be used as biomarkers to quantify NET burden.
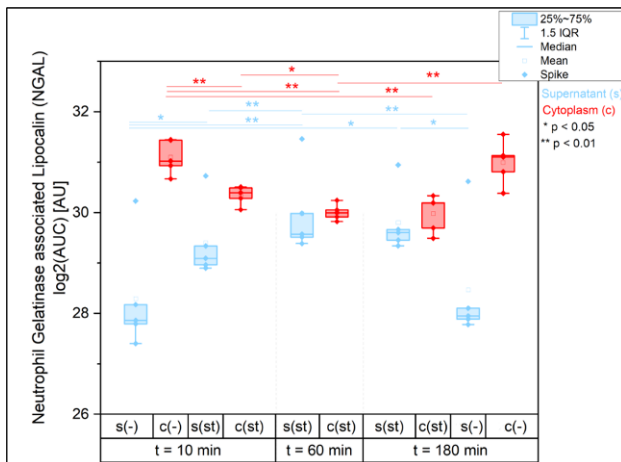
Fig. 1 | BA Basic – Vortrag

BA Basic – Vortrag

Tenascin-C deficiency improves cardiac and vascular function in diabetic cardiomyopathy in mice

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Introduction: Diabetic cardiomyopathy is known for cardiac and vascular dysfunction in lack of structural heart disease accompanied by myocardial remodeling with cardiomyocyte apoptosis, fibrosis and as endothelial dysfuntion. More recently, Tenascin-C (TN-C) upregulation in the myocardium and serum has been linked to worse outcome in diabetic and heart failure patients. However, the causative role of TN-C in the development of diabetic cardiomyopathy has not been known.

Methods: AJ and TNC-KO adult male mice were repeatedly injected with streptozotocin (50 mg/kg) to induce diabetes. Cardiac function was measured by echocardiography at baseline and at 18-20 weeks follow-up. Vascular endothelial function was assessed by using wire myography in isolated abdominal aorta segments. Cardiac fibrosis and coronary network geome-try were assessed. In addition, the hemodynamic effects of purified human TN-C (phTN-C) on isolated working rat hearts were evaluated. At the end of the experiment LV myocardial biopsy was taken in order to measure high energy phyophates by HPLC. To clarify the potential source of TNC, a cellular model of diabetic cardiomyopathy using human induced pluripotent stem cell-derived cardiomyocytes (hiPSC-CMs) was established. Moreover, human ventricular cardiac fibroblasts (HCF) were cultured, then starved and treated with 1) TGF-β; 2) phTN-C (10 µg/ml) and TLR4 inhibitor in combination with TN-C and subsequently mRNA expression of α-SMA, TN-C, Col-1, Col-3 and ACE1 were assessed by RT-qPCR. Finally, human umbilical vein endothelial cells (HUVEC) were treated either with phTN-C (10 µg/ml) or combination with TLR-4 inhibitor (TAK-242, 50 nM) and analysed the expression of NADPH oxidase 1 and 4 (NOX1, NOX4), and interleukin-6 (IL-6).

Results: Blood glucose levels of diabetic AJ and TNC-KO animals did not show difference. TN-C deficiency was accompanied by preserved ejection fraction ($p < 0.05$) and preserved endothelium-dependent relaxation (at 18 weeks, $p < 0.05$ and $p < 0.001$, respectively). Histology revealed less cardiac and perivascular fibrosis in TNC-KO diabetic animals than in the AJ diabetic group ($p < 0.01$). Notably, larger coronary arteries showed multiple branching distally and thicker arterial walls in diabetic animals, while TNC-KO diabetic mice had richer branching systems, suggesting better left ventricular perfusion. In addition, cumulative dosage of rhTN-C (80 ng/ml) resulted in a significant reduction in cardiac output ($p < 0.01$) and LV systolic pressure ($p < 0.05$) in isolated rat hearts. These hemodynamic changes were accompanied by the reduction in ATP and Pcr levels in comparison with saline treatment, respectively ($p < 0.01$). Mechanistically, hiPSC-CMs under diabetic conditions did not upregulate TN-C. In contrast, TGF-β treatment markedly upregulated TN-C expression in HCF ($p < 0.01$). Notably, HCF exposed to rhTN-C promoted both α-SMA and ACE1 mRNA expression, respectively ($p < 0.05$). In addition, HUVEC incubated with rhTN-C showed increased expression of IL-6 and oxidative stress-related markers (NOX4) and TLR-4 inhibitor pre-treatment markedly reversed these changes.

Conclusion: These findings highlight the underlying mechanisms of the role of TN-C in cardiovascular dysfunction in diabetes. TN-C creates an intracellular environment that facilitates fibrosis and oxidative stress, which, leads to cardiomyocyte and endothelial cell dysfunton. Thus, TN-C may be a critical mediator of the progression of cardiovascular dysfunction in diabetes as well as a potential target for therapy.

BEST ABSTRACTS – VORTRÄGE – CLINICAL SCIENCE

BA Clinical – Vortrag

The impact of electrocardiogram transmission and direct cath lab transfer on time to reperfusion in patients with acute ST-segment elevation myocardial infarction

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Introduction: Door-to-balloon time is prognostic and an important quality indicator in the management of patients with ST segment elevation myocardial infarction (STEMI). Shortening the time from artery occlusion to reperfusion by reducing system delays is associated with improved outcomes. The
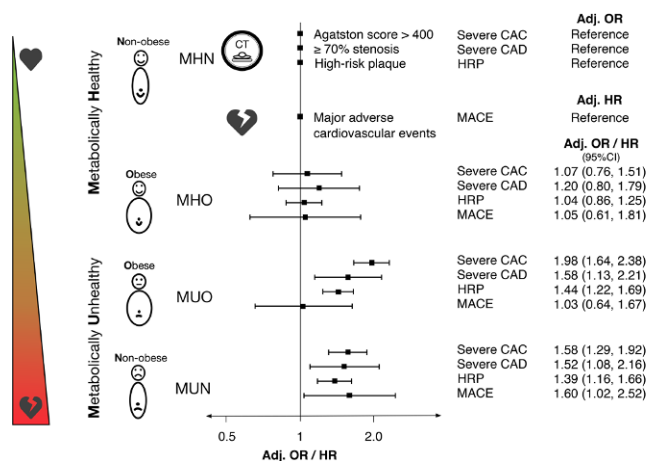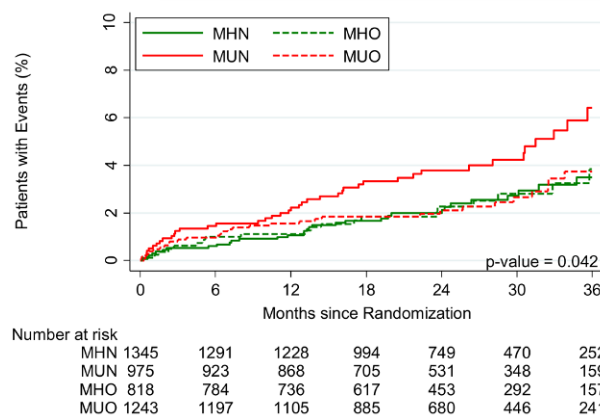
Fig. 1 | BA Clinical – Vortrag

objective of our study was to evaluate whether the electronic transmission of the electrocardiogram (ECG) from the emergency medical services (EMS) to the hospital and implementation of the policy to transfer patients with ST segment elevation directly to the cath lab–bypassing the emergency room and the intensive care unit–improves reperfusion times.

Methods: In August 2018 ECG transmission from the EMS to our hospital, a primary PCI center in Upper Austria, was implemented and a policy was released to transfer STEMI patients directly to the cath lab, whenever it was ready. Reperfusion times of all STEMI patients were prospectively assessed. We compared door-to-balloon times of STEMI patients who were admitted to our center in the 12 months preceding August 2018 to those in the year following the change of the clinical pathway.

Results: From September 2017 until August 2019, 477 consecutive STEMI patients underwent coronary angiography at our center. In the year before the implementation of ECG transmission and direct cath lab transfer (group 1) 209 patients were admitted and in the year after the policy change (group 2) 268 patients. In the latter group 47.8 % (128 patients) could be directly transferred to the cath lab while in group 1 all patients were initially assessed in the emergency room or intensive care unit. Baseline characteristics such as gender, age or diabetes did not differ between groups. In group 1, mean door-to-balloon time was 47.5 min (standard deviation 21.1 min, 95 % confidence interval 44.6–50.3), whereas in group 2 mean door-to-balloon time was 40.8 min (standard deviation 23.8 min, 95 % confidence interval 38.0–43.7; $p=0.0016$). Patients who directly went to the cath lab had a mean door-to-balloon time of 27.8 min (standard deviation 14.4 min, 95 % confidence interval 25.3–30.3) while patients who were not directly transferred to the cath lab, had a mean door-to-balloon time of 52.7 min (standard deviation 24.5 min, 95 % confidence interval 48.6–56.8). These patients were more likely to arrive out of regular working hours ($p=0.001$). The reduction in reperfusion time after implementation of the new policy was about 20 min for patients who had their ECG transmitted and who were directly transferred to the cath lab.

Conclusion: Transmission of the ECG from the EMS to the hospital and the policy to transfer patients with ST-segment elevation directly to the cath lab impressively reduced door-to-balloon times. A 20-minute reduction in reperfusion delay is of prognostic importance in STEMI patients.

BA Clinical – Vortrag

Association of metabolic health and obesity with coronary artery disease phenotype

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Introduction: Metabolic health state is associated with major adverse cardiovascular events (MACE). However, whether metabolic health states differ in coronary artery disease (CAD) phenotype and whether this association is modified by obesity is unknown.

Methods: We included stable chest pain patients from the Prospective Multicenter Imaging Study for Evaluation of Chest Pain (PROMISE) who underwent coronary computed tomography (CT) angiography. Being metabolically healthy was defined as maximum of one metabolic syndrome component except diabetes. Obesity was defined as a body mass index ≥30 kg/m^2. Four metabolic health sates were defined: metabolically healthy non-obese and obese (MHN, MHO) and metabolically unhealthy non-obese and obese (MUN, MUO). Differences in coronary CT phenotype (severe coronary artery calcification [CAC score ≥400], severe CAD [≥70 % stenosis], high-risk plaque [HRP]) and MACE (composite of death, myocardial infarction, unstable angina) between the four groups were assessed using logistic and Cox regression models, adjusted for age, sex, and smoking.

Results: Of 4381 patients (48.4 % male, 60.5 ±8.1 y/o), 49.4 % were metabolically healthy (30.7 % MHN; 18.7 % MHO) and 50.6 % unhealthy (22.3 % MUN; 28.4 % MUO). MHO had a
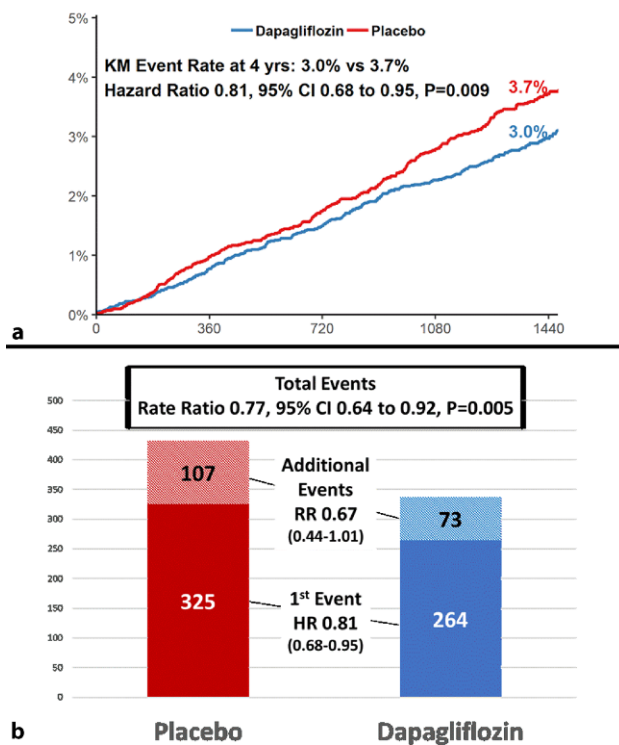
Fig. 1 | BA Clinical – Vortrag

similar CAD phenotype as compared to MHN (severe CAC (OR 1.07 [0.76–1.51]), severe CAD (OR 1.20 [0.80–1.79]), HRP (OR 1.04 [0.86–1.25])). Among metabolically unhealthy individuals, obese patients had a similar CAD phenotype as compared to non-obese (severe CAC (OR 1.26 [0.96–1.61]), severe CAD (OR 1.06 [0.76–1.47]), HRP (OR 1.04 [0.86–1.24])). However, both MUN and MUO had an unfavorable CT phenotype as compared to MHN (severe CAC (OR 1.86 [1.40–2.46] and 2.33 [1.78–3.05]), severe CAD (OR 1.52 [1.08–2.16] and 1.58 [1.13–2.21]), HRP (OR 1.39 [1.16–1.66] and 1.44 [1.22–1.69]), see Fig. 1A). During a median follow-up of 26 months, a total of 130 (3 %) events occurred. Obesity did not increase the risk among metabolically healthy but only MUN were at higher risk for MACE (see Fig. 1B).

Conclusion: In stable chest pain patients, metabolic health states exhibit distinctly different CAD phenotypes and risk for subsequent MACE, independent of obesity.

BA Clinical – Vortrag

Effect of dapagliflozin on atrial fibrillation/flutter in patients with Type 2 Diabetes mellitus: insights from the DECLARE-TIMI 58 trial

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Introduction: Atrial fibrillation (AF) and atrial flutter (AFL) are associated with diabetes and its comorbidities including hypertension, obesity, and heart failure (HF). Sodium-glucose cotransporter 2 inhibitors (SGLT2i) have been shown to lower blood pressure, reduce weight, have salutary effects on left ventricular remodeling and reduce hospitalization for HF and cardiovascular death in patients with type 2 diabetes (T2D). We therefore hypothesized that SGLT2i may reduce the risk of AF/AFL.

Methods: DECLARE-TIMI 58 studied the efficacy and safety of the SGLT2i dapagliflozin in 17,160 patients with T2D and either multiple risk factors for (MRF, $n=10,186$) or known atherosclerotic cardiovascular disease (ASCVD, $n=6974$). Here, we explore the effect of dapagliflozin on the first and total number of AF/AFL events using Cox and negative binomial models, respectively. AF events were identified using a MedDRA preferred term search ("Atrial Fibrillation", "Atrial Flutter") in the safety database.

Results: Dapagliflozin reduced the risk of incident AF/AFL by 19 % (264 versus 325 events; hazard ratio 0.81, 95 % CI 0.68 to 0.95, $P=0.009$, Fig A). The reduction in AF/AFL events was consistent regardless of presence or absence of a history of AF/AFL at baseline (Prior AF/AFL ($n=1116$): HR 0.79, 95 % CI 0.58 to 1.09, No AF/AFL: HR 0.81, 95 % CI 0.67 to 0.98; P-INT 0.89). Similarly, presence of ASCVD (HR 0.78, 95 % CI 0.62 to 0.99) versus MRF (HR 0.83, 95 % CI 0.66 to 1.04; P-INT 0.72), or a history of HF (HF: HR 0.78, 95 % CI 0.55 to 1.11, No HF: HR 0.81, 95 % CI 0.68 to 0.97; P-INT 0.88) did not modify the reduction in AF/AFL events observed with dapagliflozin. Dapagliflozin also reduced the total number of AF/AFL events (337 versus 432; rate ratio 0.77, 95 % CI 0.64 to 0.92, $P=0.005$; Fig B).

Conclusion: Dapagliflozin appears to reduce both incident AF/AFL as well as the total number of AF/AFL events in patients with T2D. This effect was consistent regardless of the patients' prior history of AF, ASCVD, or HF.

Covid-Vorträge

Myocardial ACE2 activity is independent from the use of ACEi/ARB/ARNi in severe heart failure, a COVID-19 high-risk population

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Introduction: Patients with heart failure (HF) are a high-risk population for severe SARS-CoV 2 (COVID-19) infections. This population is especially dependent on HF therapy including RAS-inhibitors, such as ACEi, ARB or ARNi. ACE2 is the main enzyme of the beneficial Ang1-7/ACE2/MasR axis that physiologically counteracts classical RAS. ACE2 is a membrane-bound aminopeptidase present in the lungs, kidney and heart exerting cardioprotective properties. ACE2, however, similarly

functions as a receptor for SARS-CoV 2 and facilitates viral entry predominantly into alveolar cells. There has been concern that the use of ACEi/ARB results in an upregulation of ACE2 enhancing SARS-CoV 2 infectivity, which could contribute to the high-mortality of SARS-CoV 2 in patients with CV comorbidities.[1] Recent analysis of retrospective data seems to be reassuring with regards to the safety of ACEi/ARB. Heart failure remains nevertheless associated with adverse outcomes with a 2.5-fold increased risk for in-hospital mortality. [2] A considerable proportion of patients develops cardiac injury associated with an increased in-hospital mortality. [3] Cardiac ACE2 has been proposed to be involved in SARS-CoV 2 related myocardial injury, [4] whereas enhanced cardiac ACE2 in patients with cardiovascular disease, especially HF, might be particularly detrimental. This study aimed to assess myocardial ACE2 and RAS enzyme activity in patients with end-stage HF, depending on the use/non-use of ACEi/ARB/ARNI.

Methods: We have measured ACE2 activity alongside related enzymes of the RAS, i. e. tissue chymase, ACE, neprilyin (NEP), prolyl-endo-peptidase (PEP) and prolyl-carboxy-peptidase (PCP), in left-ventricular myocardial samples of 52 end-stage HF patients receiving heart transplantation. Briefly, enzymatic activities have been determined by measuring angiotensin formation rates in tissue homogenates after spiking the samples with AngI, AngII or Ang1-7, respectively, followed by incubation in the presence or absence of selected RAS enzyme inhibitors. Samples were analyzed by LC-MS/MS (liquid chromatography-mass spectrometry/mass spectrometry). Specific activity of each enzyme was calculated by determining the inhibitor sensitive fraction (control minus inhibitor) of product formation related to control.

Results: We present for the first time data on myocardial RAS enzymatic activity in humans with HF––with contrast to data on human circulating ACE2 or ACE2 expression and protein concentrations in rodents–in order to investigate whether there is an upregulation of cardiac ACE2 under contemporary guideline recommended HF therapy. The results for subgroups according to background RAS-inhibitor therapy (no RASi: $n=9$, ACEi: $n=28$, ARB: $n=8$, ARNi: $n=7$) are displayed in Fig. 1. ACE2 activity was below the detection limit for all samples, suggesting a quite minimal functional role under these conditions. The presence and mode of RAS-inhibitor therapy was not related to alterations in the myocardial enzymatic profile with comparable activities of all investigated enzymes in patients without RASi or taking ACEi, ARB or ARNi.

Conclusion: These data do not support the hypothesis of RASi-mediated cardiac tissue RAS-alteration and ACE2 upregulation but rather encourage the continuation of RASi therapy in severe heart failure in order to minimize cardiac complications. Fig. 1. Myocardial tissue RAS metabolites and enzymatic

regulation of the failing heart. Mass spectrometry based determination of A. angiotensin metabolites and B. enzymatic activities for the main RAS enzymes involved in the metabolization of Ang1-7, AngI and AngII, i. e. ACE, Chymase, NEP, PEP, ACE2 and PCP according to background RAS-inhibitor therapy (no RASi: $n=9$, ACE-I: $n=28$, ARB: $n=8$ and ARNI: $n=7$). For RAS-fingerprints numbers in brackets indicate the specific angiotensin peptides. Size of spheres and numbers beside represent absolute concentrations of angiotensins (pg/ml, median value) analyzed by mass spectrometry. Enzymatic activities for the respective reaction are shown as boxplots, comparisons were made by the Kruskal-Wallis-test.

Cardiac injury in severe COVID-19 is similar to pneumonias of other origin in a multicenter study

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Introduction: COVID-19, a respiratory virus causing severe pneumonia, also affects the heart and other organs. Whether its cardiac involvement is a specific feature consisting of myocarditis, or simply due to microvascular injury and systemic inflammation, is yet unclear and presently debated. Because cardiac injury is also common in other kinds of pneumonias, we investigated and compared such occurrence in severe pneumonias due to COVID-19 and other causes.

Methods: We analysed data from 156 critically ill patients requiring mechanical ventilation in four European tertiary hospitals, including all $n=76$ COVID-19 patients with severe disease course requiring at least ventilatory support, matched to $n=76$ from a retrospective consecutive patient cohort of severe pneumonias of other origin (matched for age, gender and type of ventilator therapy). Cardiac injury, defined as elevated serum hs-troponin, CK-MB and NT-proBNP.

Results: Cardiac injury (78.1 %) and newly onset systolic dysfunction (20.8 %) were high in critical COVID-19. However, when compared to the non-COVID-19, mortality (COVID-19 = 38.2 % vs. nonCOVID-19 = 51.3 %, $p=0.142$) and impairment of systolic function were not significantly different. Surprisingly, cardiac injury was even more frequent in nonCOVID-19 (96.4 %). Although inflammatory activity (CRP and interleukin-6) was indifferent, D-dimer and thromboembolic incidence (COVID-19 = 23.7 % vs. nonCOVID-19 = 5.3 %, $p=0.002$) driven by pulmonary embolism rates (COVID-19 = 17.1 % vs. non-COVID-19 = 2.6 %, $p=0.005$) were higher.

Conclusion: Cardiac injury was high but similar to other pneumonias in severe COVID-19 requiring mechanical ventilatory support, indicating that cardiac involvement may not be its specific feature. While mortality was also similar, COVID-19 is characterized with increased thrombogenicity and high pulmonary embolism rates.
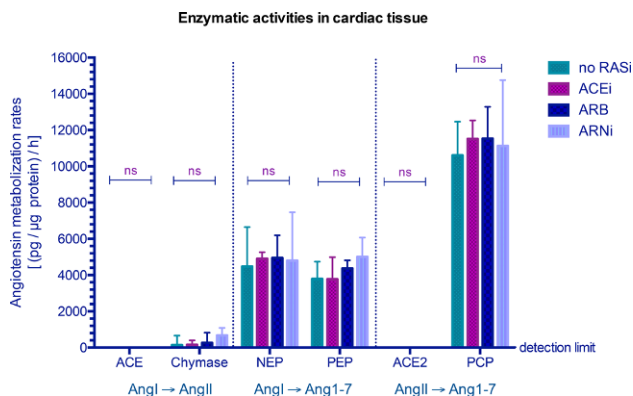

Fig. 1 | Covid-Vorträge

| | COVID-19 (n=76) | | NonCOVID-19 (n=76) | | |
	n	Mean±SD, Median (Q3-Q1) or %	n	Mean±SD, Median (Q3-Q1) or %	p-value
Gender (female)	23/76	30.3%	23/76	30.3%	>0.999
Age (years)	76	66.8±13.4	76	65.3±13.4	0.480
BMI (kg/m²)	76	27.5(6.0)	72	26.0(7.8)	0.159
Aetiology of pneumonia					
Bacterial	0/76	0%	51/76	67.1%	
Viral	76/76	100%	22/76	28.9%	
Toxic	0/76	0%	3/76	3.9%	
Bacterial superinfection if viral or toxic	28/76	36.8%	19/76	25.0%	
Required respiratory therapy					
Non-invasive ventilation	13/76	17.1%	13/76	17.1%	>0.999
Invasive ventilation	63/76	82,9%	63/76	82.9%	>0.999
Medical History					
Arterial hypertension	43/76	56.6%	41/76	53.9%	0.870
Coronary artery disease	10/76	13.2%	14/76	18.4%	0.505
Peripheral vascular disease	4/76	5.3%	4/76	5.3%	>0.999
Diabetes mellitus	20/76	26.3%	17/76	22.4%	0.706
Current smoking	13/76	17.1%	22/76	28.9%	0.123
Heart failure	7/76	9.2%	14/76	18.4%	0.157
Valvular heart disease	3/76	3.9%	5/76	6.6%	0.719
Atrial fibrillation	9/76	11.8%	16/76	21.1%	0.189
Pulmonary arterial hypertension	4/76	5.3%	2/76	2.6%	0.681
Obstructive lung disease	11/76	14.5%	17/76	21.1%	0.295
Restrictive lung disease	3/76	3.9%	7/76	9.2%	0.327
Malignancy	11/76	14.5%	17/76	22.4%	0.295

Baseline characteristics of the investigated cohorts. *p<0.05.

Fig. 1 | Covid-Vorträge Baseline characteristics of investigated cohorts

| | COVID-19 (n=76) | | NonCOVID-19 (n=76) | | |
	n	Median(Q3-Q1) or %	n	Median(Q3-Q1) or %	p-value
Cardiac injury	57/73	78.1%	54/56	96.4%	0.004*
Cardiac laboratory markers					
Initial hs-Tn (%)	73	178.6(481.1)	56	317.9(398.2)	0.003*
Max. hs-Tn (%)	73	354.3(1409.6)	56	550.0(1108.9)	0.021*
Initial CK (U/L)	74	174.5(320.8)	76	103.0(350.8)	0.231
Max. CK (U/L)	74	518.0(856.3)	76	490.5(949.0)	0.864
Initial CK-MB (U/L)	44	19.1(16.6)	51	27.3(26.2)	0.001*
Max. CK-MB (U/L)	44	22.0(28.8)	51	38.0(42.0)	0.002*
Initial NT-proBNP (pg/mL)	44	811.0(2849.8)	56	3890.0(6926.3)	<0.001*
Max. NT-proBNP (pg/mL)	44	2217.1(4481.3)	56	6625.5(13920.0)	0.001*
Functional parameters on TTE	48/76	63.2%	72/76	94.7%	
Reduced LVEF	14/48	29.2%	18/72	25.0%	0.676
Newly onset of reduced LVEF	10/48	20.8%	9/72	12.5%	0.307
LVEF (%)	48	55.0(10.0)	72	55.0(8.8)	0.277
LV dilatation	1/44	2.3%	2/66	3.0%	>0.999
RV dilatation	10/44	22.7%	11/68	16.2%	0.460
Pericardial effusion	3/47	6.4%	8/71	11.3%	0.522
Radiology findings					
Cardiomegaly	35/76	46.1%	35/76	46.1%	>0.999
Cardiomegaly during FU	15/76	19.7%	13/76	17.1%	0.835
Pulmonary venous congestion	26/76	34.2%	56/76	73.7%	<0.001*

Cardiac outcome of patients´ during ICU (intensive care unit) stay. *p<0.05.

Fig. 2 | Covid-Vorträge Parameters of cardiac outcome/ function during ICU stay

Complications and mortality of cardiovascular emergency admissions during COVID-19 associated restrictive measures

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Introduction: While hospital admissions for myocardial infarction (MI) and pulmonary embolism (PE) are decreased during the COVID-19 pandemic, controversy remains about respective complication and mortality rates. This study evaluated admission rates, complications, and intrahospital mortality for selected life-threatening cardiovascular emergencies (MI, PE, and acute aortic dissection (AAD)) during COVID-19-associated restrictive social measures (RM) in Styria, Austria.

Methods: By screening a patient information system for International Statistical Classification of Diseases and Related Health Problems (ICD) diagnosis codes covering more than 85 % of acute hospital admissions in the state of Styria (~1.24 million inhabitants), we retrospectively identified patients with admission diagnoses for MI (I21, I22), PE (I26), and AAD (I71). Rates of complications such as cardiogenic shock and cardio- pulmonary resuscitation, treatment escalations (thrombolysis for PE), and mortality were analyzed by patient chart review during 6 weeks following onset of COVID-19 associated RM, and during respective time frames in the years 2016–2019.

Results: 1668 patients were included. Cumulative admissions for MI, PE and AAD decreased (RR 0.77; $p < 0.001$) during RM compared to previous years. In contrast, intrahospital mortality increased by 65 % (RR 1.65; $p = 0.041$), mainly driven by mortality following MI (RR 1.80; $p = 0.042$). PE patients received more frequently thrombolysis treatment (RR 3.63; $p = 0.006$), while rates of cardiogenic shock and cardiopulmonary resuscitation remained unchanged. Of 226 patients hospitalized during RM, 81 patients with suspected COVID-19 disease were screened for SARS-CoV-2 infection with only 5 testing positive.

Conclusion: Cumulative hospital admissions for cardiovascular emergencies decreased during COVID-19 associated RM while intrahospital mortality increased.

FEATURED POSTER SESSION 1

FPS 1/1-1

αβT-cells partially block neonatal cardiac regeneration

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Introduction: Cardiac remodeling and subsequent heart failure remain critical issues after myocardial infarction (MI). We and others demonstrated complete cardiac regeneration in a neonatal mouse model of MI. This cardiac regenerative potential is limited to the first few postnatal days and its decline parallels with the maturation of the adaptive immune system. Herein, we hypothesized that the T-cell maturation status critically impacts the myocardial healing outcomes in neonates and contributes to the shift from regenerative to scarring phenotype observed shortly after birth.

Methods: The post-MI immune responses were characterized in postnatal day one (P1, regenerative) compared to seven-day old (P7, scarring) mice subjected to permanent left anterior descending artery (LAD) ligation. The myocardial leukocyte infiltrate was phenotyped by flow cytometry at 36 h and five days after LAD ligation. Next, we studied neonatal post-MI repair in lymphocyte-deficient Rag2 knock-out (KO) mice subjected to LAD ligation. Moreover, we adoptively transferred syngeneic splenic Thy 1.1 + T-cells obtained from adult donors into P1 recipients subjected to LAD/SHAM surgery and then assessed their impact on post-MI healing.

Results: LAD ligation induced a robust early inflammatory response (36 h post-MI) in both age groups. The in situ inflammation was, nevertheless, rapidly resolved in P1-, but not in P7-infarcted animals. The distinct age groups showed a similar profile of cardiac myeloid cell infiltration but showed remarkable differences in the lymphoid compartment. P1-infarcted mice showed an early recruitment of γδT-cells, whereas P7-infarcted mice exhibited a prominent infiltration of αβT-cells. Of note, neonatal cardiac regeneration was not altered in neonatal lymphocyte-deficient (Rag2 KO) animals. However, the adoptive transfer of adult T-cells had several consequences in neonatal mice subjected to ischemic injury. P1-infarcted mice transferred with adult T-cells showed an adult-like healing phenotype, marked by an irreversible cardiac functional impairment

(assessed by echocardiography) and increased fibrosis. This is in sharp contrast to the regenerative phenotype typically observed in untreated age-matched controls.

Conclusion: Neonatal hearts demonstrate rapid clearance of the ischemia-induced leukocyte infiltration, further reflecting the known fact of fast cardiac regeneration in newborn rodents. Of note, the adoptive transfer of adult T-cells into neonate recipients partially blocked cardiac regeneration and promoted an irreversible functional impairment. These data indicate that the cardiac repair process, and its related "regeneration vs. scarring" dichotomy, is critically impacted by the T-cell development status.

FPS 1/1-2

Ranolazine induced modulation of intracellular Na+ concentration reduces inflammation

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Introduction: Ranolazine, an established anitanginal drug, is known to reduce intracellular Na+ as well as Ca^{2+} levels. Changes in Ca^{2+} influx during proinflammatory stimulation modulates inflammation. We aimed to identify an anti-inflammatory and anti-atherosclerotic effect of ranolazine.

Methods: We used an atherosclerotic mouse model and human endothelial cells to analyze the effect of ranolazine. 3808 MERLIN-TIMI-36 patients were evaluated for changes in CRP two weeks after initiation of ranolazine treatment. In vitro analysis was performed in human umbilical vein endothelial cells.

Results: In vivo, LDL–/– mice on a high fat diet were treated with ranolazine resulting in a reduced atherosclerotic plaque burden. Overall plaque burden was evaluated using en face staining of the total aorta. Ranolazine treatment lead to a significant reduction of overall plaque burden. When analyzing the composition of the atherosclerotic plaque we found a reduction in cholesterol clefts in ranolazine treated animals, increased thickness of the fibrous cap area, and reduced macrophage count. CRP was determined in a total of 3808 patients, at baseline and after two weeks of treatment with ranolazine or placebo. Patients were included within 48-hours after the acute coronary syndrom, a condition that is well know to activate the acute phase reaction. As expected, a decrease of CRP was observed in both groups two weeks after the acute coronary syndrome. However, this decrease was much more pronounced in patients treated with ranazoline (–18.74 %, IQR –63.49 % to +95.68 %) compared to placebo (–7.22 %, IQR –57.53 % to +98.06 %; $p = 0.013$). To discover a potential mechanism for the antiinflammatory property of ranolazine, we used an in vitro cell culture approach. Ranolazine treatment of endothelial cells lead to a reduction of proinflammatory cytokines and adhesion proteins after stimulation with IL-1beta. Our data indicate that ranolazine treatment reduced the Na+ amount in endothelial cells thereby preventing IL-1beta induced influx of Ca^{2+} and reducing NF-kB response capacity.

Conclusion: The presented findings suggest anti-inflammatory effects of ranolazine in a mouse model of atherosclerosis, in human patients and in in vitro experiments. As a mode of action we propose a modulation of intracellular Na+ levels resulting in a decreased capacity to modulate Ca^{2+} levels required to mount a full response to inflammatory triggers.

FPS 1/1-3

Cell-based or cell-free therapy for hypoxic cardiomyocytes?

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Introduction: Recently the focus of cardiac regenerative therapy shifted to paracrine factors secreted by stem cells, with reparative potential similar to that of stem cells. We aimed to investigate the effect of either direct cell-based or indirect paracrine stimulation of hypoxic cardiomyocytes (CMC) for proliferation or regeneration under in vitro conditions.

Methods: We compared the gene expression change of hypoxic CMC (group Hyp-CMC) with stimulated hypoxic CMC, either by human bone-marrow derived MSC (group Hyp-CMC-MSC) or by secretome of MSC (group Hyp-CMC-SMSC), over different reoxygenation periods (4 h, 8 h, 24 h, 48 h and 72 h). Time-dependent expression of hypoxic marker HIF-1α, proliferation marker Ki-67 and cytokinesis marker RhoA of the three experimental groups was measured and compared with the normoxic CMC and MSC. The hypoxic CMC proliferation was additionally tested with an EZ4U proliferation assay. Protein expression changes of IL-18 was determined in all experimental groups. Further, we investigated the secretome content of hypoxic CMC, normoxic CMC and MSC with a cytokine panel.

Results: Time-dependent expression of hypoxia and proliferation markers were measured in all experimental groups. Gene expression of HIF-1α was upregulated in Hyp-CMC-SMSC in all tested reoxygenation periods except 72 h as compared to Hyp-CMC. When comparing Hyp-CMC-MSC to Hyp-CMC-SMSC, a weaker HIF-1α gene expression could be observed in most reoxygenation periods, except after 8 h. Ki-67 showed an increased expression after 4 h, 8 h and 48 h of reoxygenation in Hyp-CMC-SMSC, while in Hyp-CMC the expression was elevated after 24 h and 48 h reoxygenation before returning to baseline level. Cytokinesis marker RhoA showed an upregulation in Hyp-CMC-SMSC after 24 h, 48 h and 72 h while showing no change in Hyp-CMC and downregulation in Hyp-CMC-MSC. The cytokine panel revealed an expression of CXCL-12/SDF-1 and CCL5/RANTES in Hyp-CMC secretome and CD40 l, IL-17, IL17E and CXCL-12/SDF-1 in MSC secretome.

Conclusion: Our study highlights the changes in expression of different hypoxic and proliferation associated genes in different culture conditions, showing a stronger expression of HIF-1α, RhoA and Ki-67 in secretome stimulated hypoxic CMC (Hyp-CMC-SMSC) after longer reoxygenation periods. Further, a comparable and better treatment potential of MSC secretome stimulation to MSC stimulation of CMC after hypoxia could be observed in our in vitro model, suggesting the shift of cell-based to cell-free cardiac regeneration therapies.

FPS 1/1-4

Tenascin-C promotes adverse cardiac remodeling by modulating angiotensin-converting enzyme 1

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Introduction: Myocardial fibrosis is a key contributor to myocardial stiffness and reduced cardiac function in left ventricular hypertrophy (LV). Upregulation of extracellular matrix protein, Tenascin-C (TN-C) in cardiac tissue and serum are associated with cardiac fibrosis and dysfunction [1]. Nevertheless, the underling signaling mechanism of TN-C induced myocardial fibrosis and cardiac dysfunction are largely unknown. Furthermore, both diabetes type 1 and 2 are also known mediators of cardiac fibrosis. The interplay between hyperglycemia induced fibrosis and fibrotic mediators is but mostly unknown yet. The primary aim of the study was to investigate the impact of several fibrosis driving mediators onto expression of fibrosis related proteins, as well as the impact of TN-C on the transition of fibroblast to myofibroblast. Secondary, the hinge between diabetes and cardiac fibrosis, with special focus on TN C, was evaluated.

Methods: Human ventricular cardiac fibroblasts (HCF) were cultured in fibroblast basal medium. Cells were serum starved for 24 h and afterwards treated with different compounds for 24 h: 1) TGF-β (20 ng/ml), 2) ANG-II (200 nM/l), 3) TN-C (1 µg/ml). TN-C application was combined with a TLR-4 inhibitor (TAK 242, 4 µM/l) in some samples. Additionally, HCF were treated with the same mediators in hyperglycemic medium (25 mM) for 24 h post starvation. Confocal microscopy was used to detect α-smooth muscle actin (α-SMA) in treated cells. RT–qPCR was used to asses mRNA expression of α-SMA, TN-C, Col-1, Col-3 and ACE1. Treatment impact on cell viability was assessed by XTT assay.

Results: Cells that were exposed to TGF-β treatment presented significantly increased α-SMA levels (9.9-fold, $p < 0.001$), Col1, Col3 ($p < 0.01$) and TN-C expression ($p < 0.05$). In hyperglycemic conditions, TGF-β treatment lead to an even greater increase in α-SMA expression (48.2-fold, $p < 0.001$). ANG-II
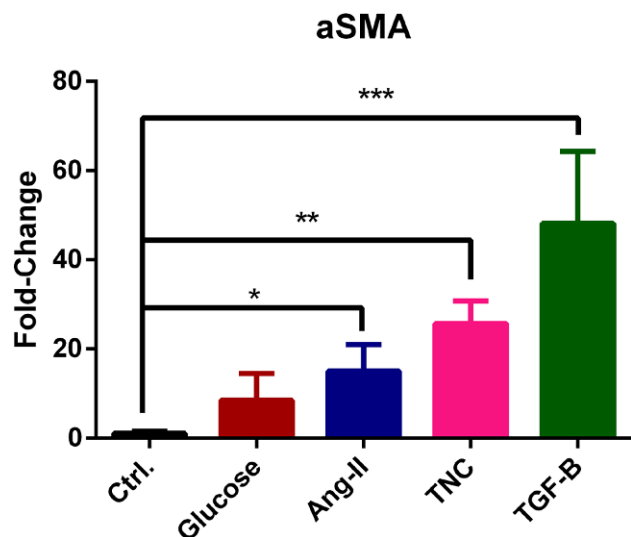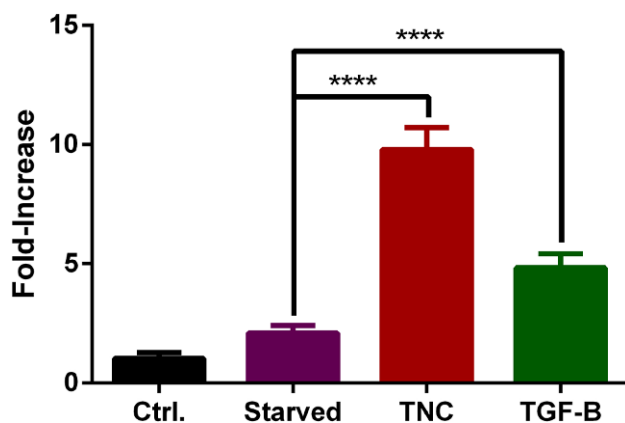

Fig. 1 | FPS 1/1-4

Abb. 2 | FPS 1/1-4

treatment in normal conditions lead to an increase of α-SMA (7.4-fold, $p < 0.05$) while a combination of both ANG-II and TGF-β resulted in a lower increase (6 fold, $p < 0.05$). In hyperglycemic conditions, α-SMA expression was elevated significantly higher (15.1-fold, $p < 0.001$). Moreover, TN-C application for 24 h also initiated the incensement of α-SMA expression (1.73-fold, $p < 0.05$). When applied in hyperglycemic medium, TN-C also lead to an elevated increase of α-SMA expression (25.7-fold, $p < 0.001$). Interestingly, ACE1 expression in HCF was markedly upregulated by TN-C (9.6-fold, $p < 0.01$), being even significantly higher compared to TGF-β treatment (4.8-fold, $p < 0.05$ vs TN-C treatment). This increase of ACE1 was unaffected by whether the mediators were applied in hyperglycemic medium or not. Application of hyperglycemic medium without any further mediator lead to a significant increase of α-SMA expression (2.2-fold, $p < 0.05$), as well as in TN-C level (6.8-fold, $p < 0.01$). Metabolic activity measurements showed no significant differences between various mediators in their effect on cell viability.

Conclusion: In summary, we established a reproducible cellular model of myocardial fibrosis. In addition, we for the first time demonstrated that TN-C promotes the transition of fibroblast to myofibroblast and ACE1 expression in cardiac fibroblast. This suggests that TN-C plays a causative role in myocardial fibrosis. Furthermore, we outlined the magnification effect of diabetic environments on fibrotic developments and on the expression of related protein markers.

FPS 1/1-5

Neuregulin-1 treatment improves diastolic function and left ventricular morphology in a rat model of chronic kidney disease

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Introduction: Cardiovascular diseases are the leading cause of morbidity and mortality in patients with chronic kidney disease (CKD). Left ventricular hypertrophy (LVH) and diastolic dysfunction (DD) are common cardiovascular complications of CKD. However, the pathogenesis and the underlying molecular signaling mechanism of uremic cardiomyopathy are poorly understood. More recently, the impairment of Neuregulin-1 (NRG-1)/ErbBs signaling was identified as major player in diastolic dysfunction and fibrosis. Therefore, the aim of the present study was to investigate 1) the role of (NRG-1)/ErbBs and 2) potential cardiorenal protective effects of NRG-1 therapy in a rat model of chronic kidney disease.

Methods: Male Wistar rats were used and randomized into different groups: 1) SHAM-operated, 2) CKD induced by 5/6 nephrectomy and 3) nephrectomized rats were treated with 10 μg/kg/d of recombinant human NRG-1 (hrNRG-1) through tail vein injection for consecutive 10 days two weeks after the induction of CKD. After the treatments, serum and urea creatinine levels were measured to verify the development of CKD and transthoracic echocardiography was performed to highlight cardiac morphology and function. Furthermore total RNA was isolated and RT-qPCR was performed to evaluate the gene expression levels of inflammatory cytokines. In addition, NRG-1 protein levels were measured in kidney and heart tissue by ELISA.

Results: In the 5/6 nephrectomized group, serum urea and creatinine levels were significantly higher ($p < 0.05$ vs Sham operated group). There was no difference in LV ejection fraction between the groups, however nephrectomized rats showed impaired diastolic function (e' was significantly decreased and E/e' was significantly increased; $p < 0.05$, respectively) and concentric hypertrophy. This was accompanied by a decrease of NRG-1 protein levels in cardiac and kidney tissue ($p < 0.05$ vs Sham, respectively). Moreover, the expression of pro-inflammatory cytokines increased in LV tissue samples in CKD ($p < 0.05$ vs Sham, respectively). Of note, rats treated with hNRG1 markedly improved cardiac diastolic dysfunction and LV hypertrophy ($p < 0.05$). In addition, surrogate markers of kidney dysfunction were also improved, suggesting the renoprotective effects of NRG-1.

Conclusion: CKD resulted in LV diastolic dysfunction and concentric hypertrophy in a CKD rat model. NRG-1 treatment prevented cardiorenal dysfunctions in nephrectomized rats. Thus, NRG-1 treatments may represent a novel therapeutic approach for improving both cardiac and renal dysfunction in patients with CKD.

FPS 1/1-6

Metabolic effects of HDAC inhibition in a feline model of HFpEF

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Introduction: Heart Failure (HF) with preserved Ejection Fraction (HFpEF) is a growing global health issue without proven effective therapies. Impaired cardiac energy metabolism, particularly derangements in fatty acid oxidation, has been reported in patients with HFpEF. Histone deacetylase (HDAC) inhibitors are a European Medicines Agency approved therapy for certain cancers and have previously been shown to have a beneficial effect in rodent models of heart failure. The prupose of this study was to investigate the impact of pan-HDAC inhibition on the plasma metabolome in a large animal model of slow-progressive pressure overload recapitulating clinical features of HFpEF.

Methods: Male domestic short hair cats ($n = 29$, age 2 mo) underwent either aortic constriction ($n = 16$) using a customized pre-shaped band or sham ($n = 13$) operation. At 2-months post-banding, banded (b) animal and sham (s) animals were assigned to the following groups for treatment with either SAHA (pan-HDAC inhibitor) or vehicle: b + SAHA ($n = 8$), b + veh ($n = 8$), s + SAHA ($n = 8$), and sham ($n = 5$). Animals received daily subcutaneous injections for 2-months. At 4-months post-banding, EDTA treated plasma samples were collected and processed using cold methanol extraction. Plasma metabolomic profiling was performed using targeted liquid chromatography-high resolution mass spectrometry (HILIC-HRMS).

Results: Comparison of SAHA-treated banded vs. SAHA-treated sham samples revealed that aortic banding significantly attenuated the metabolic reprogramming induced by SAHA. The strongest overall metabolic changes were observed between s+SAHA vs. sham. Lipids, more specifically phosphatidyl- and lysolipid levels, were significantly reduced by SAHA treatment. There was also a reduction in fatty acid levels, but this did not reach statistical significance after adjustment for multiple testing. Furthermore, SAHA treatment reduced circulating levels of carnitine and creatine, suggestive of enhanced utilization of these metabolites. In addition, an increase in oxygen consumption in-vivo and a reduction in body weight was observed in SAHA-treated animals. SAHA also improved mitochondrial function in adult feline ventricular cardiomyocytes. Thus, our results suggest that SAHA induces relevant metabolic reprogramming in a feline model towards increased mitochondrial fatty acid oxidation.

Conclusion: SAHA treatment induced metabolic reprogramming in both sham and banded animals with a reduction in phosphatidyl lipid and lysolipid levels. These results are indicative of a decrease in plasma lipoproteins. These metabolic effects may be advantageous for patients with HFpEF, where they develop metabolic derangements associated with impaired energy supply. HDAC inhibition may be a promising therapeutic target for the growing population of patients with HFpEF.

FPS 1/1-7

Release of extracellular matrix protein biglycan triggers aortic valve calcification

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Introduction: Calcific aortic valve disease (CAVD) is caused by an osteoblastic phenotype switch of valvular interstitial cells (VICs), the predominant cell type in heart valves. Mechanical strain is a crucial trigger for calcification. However, it remains unknown how mechanical strain is translated into osteoblastic activity. Danger associated molecular patterns (DAMPs) are released from stressed cells and are known to activate Toll-like receptors. We hypothesized that a structural protein of the extracellular matrix (ECM), namely biglycan (BGN), is released upon mechanical injury activating pattern recognition receptor Toll-like receptor 3 (TLR3)

Methods: Aortic valves were obtained from patients undergoing aortic valve replacement or from explanted hearts. To simulate valvular strain conditions, VICs were exposed to 15 % cyclic stretch. Released BGN was characterized via size-exclusion chromatography. Recombinant huBGN and huTLR3 ectodomains were analyzed for interaction via isothermal titration calorimetry. To illustrate the most plausible binding mode between huTLR3 and huBGN, we modelled a complex maintaining the collinearity of the dimer axes as a restraint in subsequent protein-protein docking and local refinement. Finally, we examined two clinical cohorts (GERA, $n = 55{,}192$ with 3469 aortic stenosis cases; UK Biobank, $n = 257{,}231$ with 2213 aortic stenosis cases) whether genetic variation at 12 genes implicated in the BGN/TLR3 signaling pathway was associated with aortic stenosis in humans.

Results: Mechanical challenge of VICs resulted in activation of the TLR3 pathway. TLR3 inhibition abrogated strain-dependent activation of VICs. Supernatant from mechanically challenged cells activated TLR3 reporter cells. BGN induced both TLR3 expression and specific receptor activation in a dose-dependent fashion in a comparable fashion to the TLR3 agonist Poly (I:C). Size exclusion chromatography showed co-elution of huTLR3-ECD in the BGN fraction, indicating physical interaction between BGN and TLR3. 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: In conclusion, we propose a mechanism underlying CAVD, in which BGN constitutes a selective and potent endogenous TLR3 ligand, perpetuating valvular calcification. Our data unravel the TLR3-RUNX2 axis to be an evolutionary conserved pathway of morphogenesis and osteogenesis, offering novel therapeutic strategies to counteract CAVD in humans.

FPS 1/1-8

Neutrophil extracellular traps induce MCP-1 release from endothelial cells at the plaque rupture site in acute myocardial infarction

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Introduction: Leukocyte-mediated inflammation is crucial in acute myocardial infarction (AMI). We recently observed that neutrophil extracellular traps (NETs) are increased at the culprit site, promoting activation and differentiation of fibrocytes, cells with mesenchymal and leukocytic properties. Fibrocyte migration is mediated by monocyte chemoattractant protein (MCP)-1 and C-C chemokine receptor type 2 (CCR2). We investigated the interplay between NETs, fibrocyte function, and MCP-1 in AMI.

Methods: Culprit site and femoral blood of AMI patients was drawn during percutaneous coronary intervention. We characterized CCR2 expression of fibrocytes by flow cytometry. MCP-1 and the NET marker citrullinated histone H3 (citH3) were measured by ELISA. Fibrocytes were treated in vitro with MCP-1. Human coronary arterial endothelial cells (hCAECs) were stimulated with isolated NETs, and MCP-1 was measured by ELISA and qPCR. The influence of MCP-1 on NET formation in vitro was assessed using isolated neutrophils.

Results: We have included 50 consecutive AMI patients into the study. NETs and concentrations of MCP-1 were increased at the CLS. NET stimulation of hCAECs induced MCP-1 on RNA and protein level. Increasing MCP-1 gradient was associated with fibrocyte accumulation at the site of occlusion. In the presence of higher MCP-1 these fibrocytes expressed proportionally less CCR2 than peripheral fibrocytes. In vitro, MCP-1 dose-dependently decreased fibrocyte CCR2 and reduced ex vivo NET release of healthy donor neutrophils.

Conclusion: NETs induce endothelial MCP-1 release, presumably promoting a chemotactic gradient for leukocyte and fibrocyte migration. MCP-1 mediated inhibition of NET formation could point to a negative feedback loop. These data will shed light on vascular healing.

FEATURED POSTER SESSION 2

FPS 2/2-1

Which is the best ECG-algorithm to predict the culprit lesion (RCA/Cx) in inferior wall STEMI

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Introduction: The electrocardiogram (ECG) is not only a powerful tool to detect an acute myocardial infarction (AMI), but also to identify the site of ischemia. There is evidence that the ECG can also help to predict the culprit artery in inferior ST-elevation myocardial infarction (STEMI). It was the aim of this study to compare several common ECG-algorithms predicting the culprit lesion.

Methods: In this retrospective cohort study, we included 300 patients with inferior STEMI who underwent primary PCI at our Department of Cardiology between 2012–2015. ST-segment elevations and depressions were measured, and the exact location of the culprit lesion was determined based on the PCI-protocol. We analysed Sensitivity, specificity, positive and negative predictive value and their diagnostic accuracy of several common ECG-algorithms. In a further step we chose the 3 best algorithms (Tierala: complex algorithm; Zimetbaum: ST-elevation in III>II AND STD >1 mm in I OR aVL; Herz A) and investigated which circumstances lead to a failure of the algorithm. We also tested several "single step criteria". Then we performed a logistic regression (backwards: Wald) to identify factors which might have caused the failure of the algorithms.

Autor	Sens.	Spez.	PPV	NPV	Accuracy	ROC-AUC (Konf.interv.)
Tierala (96)	90,20%	58,50%	88,70%	62,30%	83,30%	0,74 (0,63-0,79)
Zimetbaum (104)	91,10%	52,30%	87,30%	61,80%	82,70%	0,72 (0,43-0,59)
Herz A (95)	91,10%	52,30%	87,30%	61,80%	82,70%	0,72 (0,61-0,76)
Fiol (100)	90,60%	50,80%	86,90%	60,00%	82,00%	0,71 (0,66-0,81)
Bairey (102)	77,40%	69,20%	90,10%	45,90%	75,70%	0,73 (0,64-0,80)
Chia (101)	74,00%	73,80%	91,10%	44,00%	74,00%	0,74 (0,67-0,82)
Herz A+B (95)	72,30%	64,60%	88,10%	39,30%	70,70%	0,69 (0,67-0,81)
Herz B (95)	80,00%	21,50%	78,70%	23,00%	67,30%	0,51 (0,64-0,80)
Vales (103)	71,90%	43,10%	82,00%	29,80%	65,70%	0,58 (0,49-0,66)

Accuracy = right positive + right negative results; ROC-AUC = ROC Area Under the Curve

Fig. 1 | FPS 2/2-1 Statistical data of the chosen algorithms

Results: In 235 of 300 patients the RCA was the occluded artery and in 65 the CX, respectively. There were highly significant differences in the ST-Deviations in lead I, III, aVR, aVL, aVF and V1 between RCA and CX-occlusions. Most of the previously published ECG-criteria and -algorithms could not reach their primary published results in our cohort. The best performance was shown by a complex algorithm, published by Tierala et al. (83 % accuracy) followed by the algorithms of Zimetbaum and Herz A (82.7 % accuracy). The failure of Tierala's algorithm was associated with the dominance of the vessel system. Age, sex and most cardiovascular risk factors did not crucially influence the algorithm. The best "single step criterion" was an ST-elevation in III ≥ II or III > II, predicting the infarcted vessel in more than 80 %. However, in this case, the high accuracy is reasoned by the ability of this criterion to predict the RCA as infarcted vessel in 91–99 %. The ability of this criterion to predict the Cx as infarcted vessel was very low (23–52 %).

Conclusion: It is possible to predict the infarct-related artery in inferior STEMI with a high accuracy using the algorithms of Tierala, Herz A and Ziemetbaum. The use of the single step criterion "ST-elevation in III ≥ II or III > II" presents a quick and fairly exact way to identify the infarcted vessel.
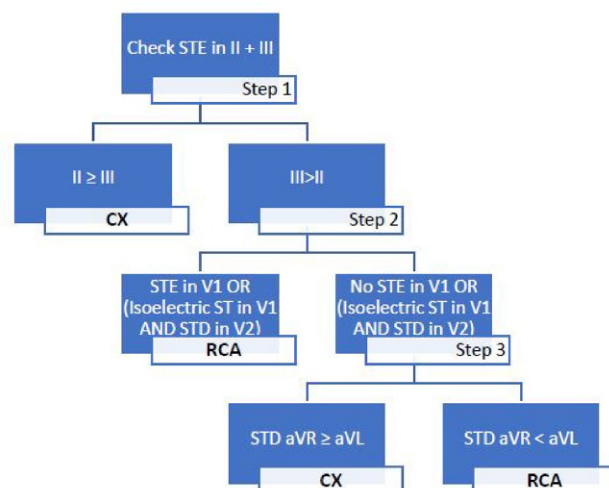

Abb. 2 | FPS 2/2-1 Algorithm of Tierala et al.

FPS 2/2-2

Pacemaker lead-induced progression of primary vs. secondary tricuspid regurgitation

M. Riesenhuber[1], A. Spannbauer[1], T. Pezawas[1], C. Schukro[1], M. Gwechenberger[1], G. Stix[1], A. Anvari[1], T. Wrba[2], C. Khazen[3], M. Andreas[3], G. Laufer[3], C. Hengstenberg[1], M. Gyöngyösi[1]

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Introduction: Lead-induced tricuspid regurgitation (TR) is an important factor for morbidity and mortality after pacemaker (PM) implantation. Currently no data are available comparing worsening of primary or secondary TR due to implantation of a PM. Guidelines define secondary TR as functional TR due to right ventricular dilatation (RVD). Primary TR is defined by primary TR leaflet dysfunction without RVD. The aim of the present retrospective analysis was to assess the TR after PM implantation with at least one permanent, transtricuspid lead comparing patients with vs. without RVD.

Methods: Patients with PM implantation ($n = 990$) were enrolled if they had routine echocardiography with assessment of TR before first PM implantation and immediately after.

Table 1 Patient characteristics

	No pre-existing RV dilatation n=747	Pre-existing RV dilatation n=243	P Value
Age (years)	70.4 ± 12.5	70.4 ± 10.9	0.95
Female sex	285 (38.2 %)	92 (37.9 %)	0.94
Pre Pacemaker:			
no or mild TR	579 (77.5 %)	148 (60.9 %)	<0.001
moderate TR	131 (17.5 %)	60 (24.7 %)	
severe TR	37 (5.0 %)	35 (14.4 %)	
Post Pacemaker:			
no or mild TR	476 (63.7 %)	75 (30.9 %)	<0.001
moderate TR	216 (28.9 %)	85 (35.0 %)	
severe TR	55 (7.4 %)	83 (34.2 %)	
Pre Pacemaker:			
Left ventricular function			
normal	431 (57.8 %)	97 (39.9 %)	<0.001
mild reduction	121 (16.2 %)	44 (18.1 %)	
moderate reduction	100 (13.4 %)	35 (14.4 %)	
severe reduction	94 (12.6 %)	67 (27.6 %)	
Mitral regurgitation:			
no or mild	343 (53.3 %)	61 (32.3 %)	<0.001
moderate	254 (39.4 %)	83 (43.9 %)	
severe	47 (7.3 %)	45 (23.8 %)	
sPAP (mmHg)	44.7 ± 14.2	58.2 ± 17.6	<0.001

RV: right ventricle; sPAP: systolic pulmonary artery pressure; TR: tricuspid regurgitation.
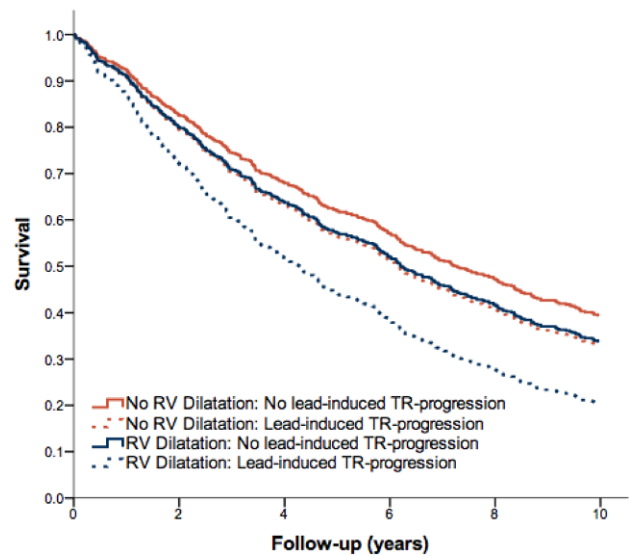
Fig. 1 | FPS 2/2-2 Survival of patients with primary vs. secondary tricuspid regurgitation (TR) with vs. without PM-lead induced TR-progression

RVD and severity of TR were characterized visually. Based on RVD in baseline echocardiography, patients were divided into 2 groups: without pre-existing RVD (primary TR) or with pre-existing RVD (secondary TR). Mortality data were obtained from "Statistics Austria".

Results: In total, 747 patients without pre-existing RVD and 243 patients with pre-existing RVD were identified (Table 1). TR worsening was observed in 38.7 % of patients without pre-existing RVD and in 54.3 % of patients with pre-existing RVD ($P < 0.001$). Progression from mild/moderate to severe TR was observed in 6.7 % of patients with primary TR, compared to 25.6 % of patients with secondary TR ($P = 0.001$). Using an ordinal regression model, the probability to suffer from severe TR after PM implantation in patients without pre-existing RVD (primary TR) was 14.8 % (95 % CI 11.0–19.7 %), compared to 41.6 % (95 % CI 40.3–42.8 %) in patients with pre-existing RVD (secondary TR, $P < 0.001$). As indicated in Fig. 1, worsening of TR was associated with higher mortality in the group of pre-existing RVD (HR 1.45, 95 % CI 1.01–2.08, $P = 0.046$), but not in the group without RVD (HR 1.19, 95 % CI 0.93–1.53, $P = 0.16$).

Conclusion: Pre-existing secondary TR was associated with higher rates of lead-induced progression to severe TR and with higher mortality compared to primary TR. Leadless pacing or tricuspid valve clipping post-PM implantation [1] is feasible and could be an alternative for patients with pre-existing functional TR with an indication for PM.

FPS 2/2-3

Adherence to dual anti-platelet therapy after acute coronary syndrome and its impact on patient outcome–a nationwide perspective

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Introduction: Secondary prevention after acute coronary syndrome (ACS) mirrors a key position in the reduction of morbidity and mortality in this highly vulnerable patient population. Especially dual anti-platelet therapy (DAPT)–including aspirin plus a P2Y12 inhibitor–proved to be one of the most beneficial therapeutic approaches for the reduction of re-events and stent thrombosis. However, profound epidemiological measures on adherence to DAPT intake after ACS remain scare, but seem of major importance in terms of preventing fatal cardiac adverse events. Therefore, we aimed to investigate adherence to DAPT after ACS and its impact on patient outcome from an Austrian nationwide perspective.

Methods: Within this population-based national observation all patients presenting with ACS between 04/2011 and 8/2015 in Austria were enrolled. Patient characteristics and co-morbidities were assessed via the Austrian national health insurance system and elucidated according to ICD10 definitions. Adherence to DAPT was investigated according to handing in prescriptions for aspirin and P2Y12 inhibitors at local pharmacies. Patients were followed prospectively until the primary study endpoint (= mortality) was reached. Cox Regression hazard analysis was used to investigate the impact of non-adherence to DAPT on patient outcome and was adjusted for a comprehensive subset of confounders within the multivariate model.

Results: During the observation period a total of 22.331 patients (median age: 65 years [55–75]; male: 69.7 % [$n = 15{,}176$]) met the inclusion criteria. Patients presenting with the indication for oral anticoagulation ($n = 2165$; 9.7 %), individuals that died during the index event ($n = 151$; 0.7 %), patients that presented with a re-ACS ($n = 396$; 1.7 %) or those who were lost during follow-up ($n = 96$; 0.4 %) were not included within the final analysis. Of alarming importance 70.7 % ($n = 15{,}792$) of all patients presenting with ACS did not take DAPT as recommended by current guidelines. The highest rate of drug interruption/end of therapy was observed within the first month after the index event with almost 50 % of all cases. During patient follow-up until 14 months after the index event 513 individuals died. Non-adherence to DAPT proved a strong an independent association with mortality with an adjusted hazard ratio of 1.25 (95 %CI: 1.09–1.41; $p < 0.001$) (see Fig. 1).

Conclusion: The present nationwide investigation highlighted an overall low adherence to DAPT after ACS, with the highest interruption/end of therapy rate within the first month after the index event. Since the intake of DAPT after ACS was associated with a 20 % risk reduction for fatal cardiovascular events during the observation period, awareness in terms of drug-adherence and intensified patient follow-up should be promoted, in order to prevent fatal atherothrombotic events.

Abb. 1 | FPS 2/2-3

FPS 2/2-4

Machine learning enables prediction of cardiac amyloidosis by routine laboratory parameters: a proof-of-concept study

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Introduction: Cardiac amyloidosis (CA) is a rare and complex condition with poor prognosis. While novel therapies improve outcomes, many affected individuals remain undiagnosed due to a lack in awareness among clinicians. This study was undertaken to develop an expert-independent machine learning (ML) prediction model for CA relying on routinely determined laboratory parameters. The present study was driven by the conception that irrespective of geographical and health care system idiosyncrasies, a palette of routine laboratory parameters is usually obtained from patients presenting with heart failure (HF) symptoms, as a baseline evaluation. We hypothesized here that patients with CA-associated HF display a distinct laboratory parameter pattern as compared to patients presenting with other HF entities and that machine-learning algorithms may help to identify these patterns.

Methods: This was a single-center study that was performed within the frames of a prospective HF registry and all participants gave their written informed consent. Transthyretin CA was diagnosed by endomyocardial biopsy before 2016. All patients with transthyretin CA underwent genetic testing to differentiate between wild-type and hereditary disease. Light chain CA was diagnosed by endomyocardial biopsy using Congo-red staining and immunohistochemistry to categorize the amyloidogenic precursor protein. Alternatively, light chain CA was diagnosed when extra-myocardial biopsy was positive for light chains and transthoracic echocardiography (TTE) or cardiac magnetic resonance (CMR) imaging showed signs of left ventricular hypertrophy. The spectrum of HF controls included patients with HF and preserved or reduced EF (HFpEF or HFrEF), valvular heart disease, cardiac sarcoidosis, and other HF conditions. In a first step, we used linear models as our baseline prediction model. In a second step, we built an ML prediction model based on gradient-boosted ensemble of decision trees, whose performance was compared to the baseline. All prediction models were developed on a training cohort, consisting of patients with proven CA (positive cases, $n = 121$) and amyloidosis-unrelated heart failure (HF) patients (negative cases, $n = 415$). Performances of all prediction models were
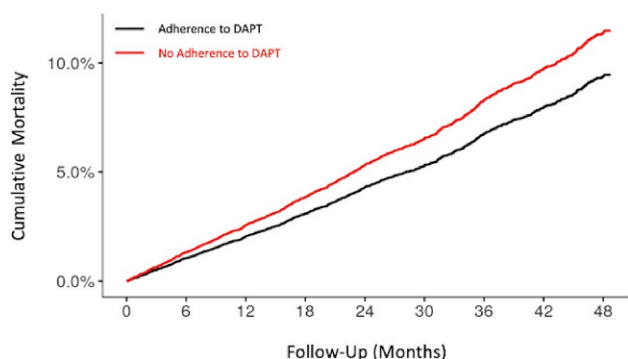
evaluated on a separate prognostic validation cohort with 37 CA positive and 124 negative patients.

Results: In a first step, we developed a diagnostic algorithm based on logistic regression with iterative imputation of missing values. Before training the diagnostic algorithm, we excluded 16 parameters with a high missing values ratio (cut-off at 60 % missing ratio; 15 parameters), and highly collinear parameters (cut-off at 0.98 Pearson correlation; 4 parameters); 13 parameters had both–a high missing values ratio and a high collinearity index. When evaluated on the independent test set, the area under the receiver-operative characteristic curve (ROC AUC) score of this model was 0.58, with sensitivity, specificity of 67.6 %, 53.2 %, respectively. In a second step we used an ML prediction algorithm based on gradient-boosted ensembles of decision trees. When we evaluated the ML prediction model on the validation cohort, it achieved a ROC AUC score of 0.86, with sensitivity and specificity of 89.2 % and 78.2 %, respectively.

Conclusion: The sensitivity of the ML model is high, which means that this model could be used for positive CA patients screening. However, it is slightly worse at excluding non-amyloid patients than at detecting positives, with a specificity of 78.2 %. Therefore, such an automated prediction model should only be used for screening CA patients followed by other confirmatory tests. We tested our algorithm on pre-selected cohorts manifesting symptoms of HF with an amyloidosis prevalence of 23 %, which is relatively high for a rare disease with a prevalence of 1 in 10.000. Our work demonstrates that ML makes it possible to utilize basic laboratory parameters to generate a distinct CA-related HF profile compared with CA-unrelated HF patients. This proof-of-concept study opens a potential new avenue in the diagnostic workup of CA and may assist physicians in clinical reasoning.

FPS 2/2-5

First 1-year follow-up data snapshot analysis of more than 5200 patients treated with edoxaban in routine clinical practice in Germany, Austria and Switzerland–(DACH): Data from the ETNA-AF registry

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Introduction: Edoxaban has been approved for stroke prevention in patients with atrial fibrillation based on its comparable efficacy and superior safety compared to warfarin in the pivotal ENGAGE AF-TIMI 48 trial. ETNA-AF Europe (NCT02944019) was initiated in agreement with the EMA to evaluate benefits and risks of edoxaban treatment in unselected patients in routine clinical practice. Background: In the ENGAGE AF-TIMI 48

Outcome table

ANNUALIZED EVENT RATES FOR, n (%/year)	ETNA-AF DACH		
	Total	Edoxaban 60 mg OD	Edoxaban 30 mg OD
Patients, N (%)	5223 (100%)	4040 (77.4%)	1183 (22.6%)
TOTAL MORTALITY	174 (3.49)	82 (2.11)	92 (8.34)
CARDIOVASCULAR MORTALITY	82 (1.64)	40 (1.03)	42 (3.81)
MAJOR BLEEDING	36 (0.72)	25 (0.64)	11 (1.00)
CRNM BLEEDING	31 (0.62)	25 (0.65)	6 (0.55)
MAJOR OR CRNM BLEEDING	67 (1.35)	50 (1.29)	17 (1.55)
MAJOR GI BLEEDING	12 (0.24)	6 (0.15)	6 (0.55)
ICH	10 (0.20)	9 (0.23)	1 (0.09)
ISCHEMIC STROKE	27 (0.54)	18 (0.46)	9 (0.82)
HAEMORRHAGIC STROKE	3 (0.06)	3 (0.08)	0 (0.00)
MYOCARDIAL INFARCTION	27 (0.54)	17 (0.44)	10 (0.91)

Abb. 1 | FPS 2/2-5

trial (edoxaban phase 3 study including total of 21,000 patients), 294 patients were randomized and treated with the approved dose 60 mg edoxaban (30 mg when meeting the dose reduction criteria) in Germany, Austria and Switzerland (DACH) region. The non-interventional study Edoxaban Treatment in routiNe clinical prActice for patients with non-valvular Atrial Fibrillation, provides the full 1-Year-Follow-up (3rd) outcome data snapshot for 5223 patients in the DACH region.

Methods: 5993 patients from across 365 hospital and office-based physicians from Germany, Austria and Switzerland were enrolled, and will be followed-up for 4 years. This snapshot analysis includes baseline and first outcome data of 5223 patients (87.15 % of all enrolled patients) that have completed their first 1-year follow-up visit (mean follow-up: 344.6 days).

Results: The average age of patients was 73.3 ±9.5 years, the mean weight was 83.4 ±17.7. Mean CHA2DS2-VASc-Score was (3.2 ±1.37), common comorbidities include hypertension (83.6 %), diabetes (26.5 %), congestive heart failure (4.4 %) and history of myocardial infarction (3.2 %). The current AF types includes paroxysmal (55.0 %), persistent (24.4 %) and long standing persistent and permanent (20.5 %). Patients receiving the 30 mg dose (22.6 %) were older (78.6 ±7.9), had a lower creatinine clearance (53.4 ±21.8) and had a higher risk for both stroke and bleeding as compared to those on the 60 mg dose (77.4 %). Overall, the incidence of clinical events was low (table 1): all-cause mortality: 3.49 %/y, major bleeding 0.72 %/y, intracranial haemorrhage 0.20 %/y, and ischemic stroke 0.54 %/y.

Conclusion: We found low bleeding and stroke rates in 5223 unselected, mainly elderly AF patients treated with edoxaban in routine clinical practice. These findings were consistent across edoxaban doses and reinforce the effectiveness and safety of NOACs such as edoxaban in routine clinical care in DACH.

FPS 2/2-6

Adherence to high-intensity statin therapy after acute coronary syndrome and its impact on patient outcome–a nationwide perspective

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Introduction: Secondary prevention after acute coronary syndrome (ACS) mirrors a key position in the reduction of morbidity and mortality in this highly vulnerable patient population. Especially lipid lowering therapy—via high-intensity statins (atorvastatin and rosuvastatin)—proved to be one of the most beneficial therapeutic approaches for the reduction of re-events and stent thrombosis. However, profound epidemiological measures on adherence to statin intake after ACS remain scare, but seem of major importance in terms of preventing fatal cardiac adverse events. Therefore, we aimed to investigate adherence to high-intensity statin therapy after ACS and its impact on patient outcome from an Austrian nationwide perspective.

Methods: Within this population-based national observation all patients presenting with ACS between 04/2011 and 8/2015 in Austria were enrolled. Patient characteristics and co-morbidities were assessed via the Austrian national health insurance system and elucidated according to ICD10 definitions. Adherence to high-intensity statins was investigated according to handing in prescriptions for rosuvastatin and atorvastatin at local pharmacies. Patients were followed prospectively until the primary study endpoint (= mortality) was reached. Cox Regression hazard analysis was used to investigate the impact of non-adherence to high-intensity statin therapy on patient outcome and was adjusted for a comprehensive subset of confounders within the multivariate model.

Results: During the observation period a total of 23,240 patients (median age: 65 years [55–75]; male: 67.7 % [$n=15{,}728$]) met the inclusion criteria. Individuals that died during the index event ($n=366$; 1.6 %), presented with a re-ACS ($n=569$; 2.4 %) or were lost during follow-up ($n=158$; 0.6 %) were not included within the final analysis. Of alarming importance 66.4 % ($n=15.422$) of all patients presenting with ACS did not take high-intensity statins as recommended by current guidelines. The highest rate of drug interruption/end of therapy was observed within the first month after the index event with more than 50 % of all cases. During patient follow-up until 01/2018 a total of 3522 (15.2 %) individuals died. Non-adherence to high-intensity statins had a strong an independent association with mortality with an adjusted hazard ratio of 1.16 (95 %CI: 1.06–1.25; $p<0.001$) (see Fig. 1).

Conclusion: The present nationwide investigation highlighted an overall low adherence to high-intensity statins after ACS, with the highest interruption/end of therapy rate within the first month after the index event. Since the intake of high-intensity statins after ACS was associated with a 14 % risk reduction for fatal cardiovascular events during the observation period, awareness in terms of drug-adherence and intensified

patient follow-up should be promoted, in order to prevent fatal atherothrombotic events.

Prognostic value of cardiac magnetic resonance imaging in ST-elevation myocardial infarction patients with preserved ejection fraction

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Introduction: Left ventricular ejection fraction (LVEF) is the principle parameter for patient-specific prognostication and forms the basis for several treatment decisions after ST-elevation myocardial infarction (STEMI). However, approximately half of patients nowadays have preserved ejection fraction after the acute event. The number of recurrent major adverse cardiovascular events (MACE) in this patient group is substantial, yet, it is largely unknown how to risk stratify these patients. The aim of this study was to evaluate the prognostic value of myocardial tissue characterization by cardiac magnetic resonance (CMR) imaging in a STEMI population with preserved LVEF following primary percutaneous coronary intervention (PCI).

Methods: In total, 451 STEMI patients undergoing primary PCI were prospectively enrolled from 2011 to 2017. CMR examinations were conducted 3 (interquartile range[IQR]:2–4) days after PCI for STEMI. LVEF, infarct size, microvascular obstruction (MVO) and myocardial strain values were measured. Primary endpoint was defined as MACE composite including death, re-infarction and congestive heart failure.

Results: A preserved LVEF (≥50 %) was detected in 286 patients (= 63 %). In the overall cohort, 46 patients experienced a MACE event, 20 MACE events occurred in the group with preserved LVEF. From all CMR parameters assessed, presence of MVO (hazard ratio[HR]: 2.61 [95 %CI:1.03–6.63]; $p=0.04$) and global longitudinal strain (GLS; HR: 1.27 [95 %CI:1.05–1.52]; $p=0.01$) significantly predicted MACE in the LVEF-preserved population. The associations of MVO ($p=0.02$) and GLS ($p=0.01$) with MACE remained significant after adjusting for clinical risk features (TIMI risk score). Moreover, the addition of MVO and GLS to TIMI risk score significantly ($p=0.02$) increased the prognostic validity (AUC: 0.80 [95 %CI:0.70–0.89]) as compared to TIMI risk score alone (AUC: 0.63[0.49–0.78]).

Conclusion: In contemporary treated STEMI patients showing preserved LVEF, a CMR-based risk prediction approach including MVO and GLS provided strong prognostic value that was incremental to traditional outcome markers.
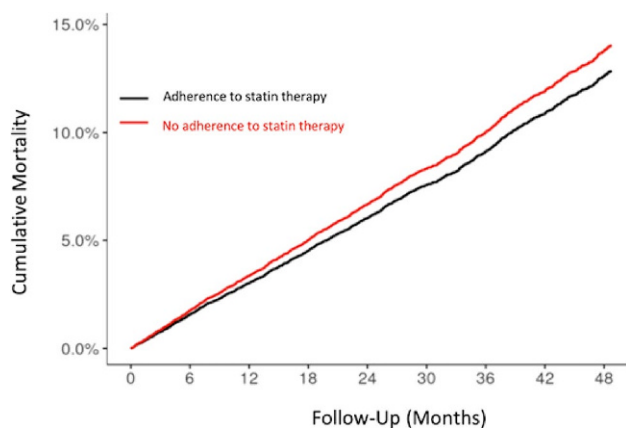

Abb. 1 | FPS 2/2-6

FPS 2/2-8

Myocardial tissue characterization and outcome in transcatheter aortic valve replacement

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Introduction: Cardiac decompensation in severe aortic stenosis (AS) involves accumulation of myocardial extracellular matrix and congestion, which can be objectively quantified by cardiac magnetic resonance (CMR) and bioelectrical impedance spectroscopy (BIS). This study sought to determine whether the extent of extracellular matrix on CMR pre-intervention correlates with congestion and is associated with outcome.

Methods: Consecutive patients scheduled for transcatheter aortic valve replacement (TAVR) underwent measurement of volume status using BIS, and CMR including extracellular volume (ECV) quantification, late gadolinium enhancement imaging, and T2 mapping, for the assessment of diffuse interstitial changes, myocardial scar, and edema, respectively. Subjects were prospectively followed; the combination of all-cause death and heart failure (HF) hospitalization was selected as primary study endpoint.

Results: 180 patients (80.6 ±7.2 y/o, 48.9 % female) with valid BIS and CMR data were included. Fluid levels significantly correlated with ECV ($r=0.403$, $p<0.001$) and T2 relaxation times ($n=100$, $r=0.378$, $p=0.003$), and were independently associated with ECV by multivariate linear regression analysis ($p<0.001$) after adjustment for important confounders, including left ventricular ejection fraction and hematocrit. Among patients receiving TAVR ($n=170$), 21.2 % ($n=36$) experienced an event (20 deaths, 12 HF, 4 both) 13.4 ± 7.5 months following treatment. ECV levels≥median of 27.0 % were significantly associated with death (log rank, $p=0.008$), HF hospitalization ($p<0.001$), and the combined endpoint by Kaplan Meier estimates ($p<0.001$, Fig. 1). Quantitatively, every 1 % increase in ECV increased the event hazard by 13 % [hazard ratio 1.129, 95 % confidence interval 1.047–1.217, $p=0.002$]. After multivariate adjustment for clinical, laboratory and CMR parameters, ECV≥median
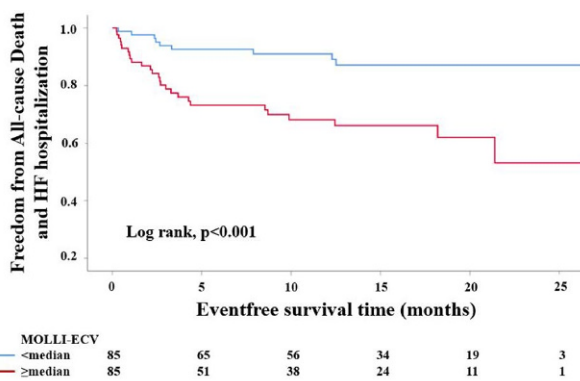
HF indicates heart failure; MOLLI-ECV, Extracellular volume fraction by Modified Look-Locker Inversion recovery sequence

Fig. 1 | FPS 2/2-8 Kaplan Meier estimates
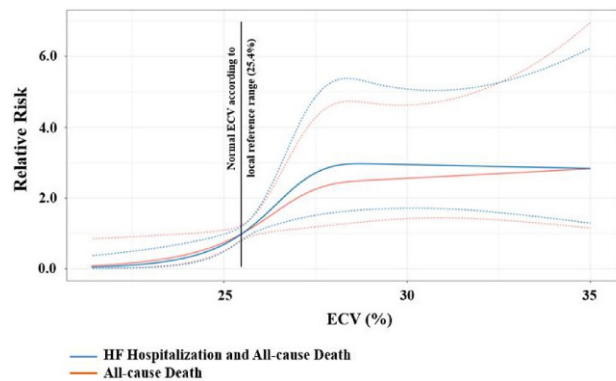
Dashed lines are 95% confidence intervals. ECV indicates extracellular volume fraction; HF, heart failure.

Fig. 2 | FPS 2/2-8 Spline curve analysis

remained independently associated with outcome ($p=0.044$), alongside with serum albumin ($p<0.001$), EuroSCORE II ($p=0.009$), and body fluid levels ($p=0.027$) by Cox regression analysis. Moreover, compared to AS patients with a normal ECV fraction according to the local reference range, every increase in ECV was associated with higher event hazards by spline curve analysis (Fig. 2).

Conclusion: Cardiac decompensation in AS comprehends myocardial ECV expansion and body fluid accumulation. Fluid accumulation appears to impact the amount of ECV on CMR, and both parameters are independently associated with adverse outcomes following TAVR. Whether ECV has potential to guide treatment strategies in severe asymptomatic AS merits further research.

FEATURED POSTER SESSION 3

FPS 3/3-1

Relationship between baseline cardiac biomarkers and cardiovascular death or hospitalization for heart failure in DECLARE-TIMI 58

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Introduction: Dapagliflozin reduced the risk of the composite of cardiovascular death or hospitalization for heart failure (CVD/HHF) in patients (pts) with type 2 diabetes (T2D) in DECLARE-TIMI 58. We hypothesized that cardiac biomarkers may help to identify pts who are at higher baseline risk and thus may benefit more from treatment with SGLT2i. The objective of this study was therefore to evaluate the association of baseline hsTnT and NT-proBNP levels with CVD/HHF and with the magnitude of benefit with dapagliflozin.
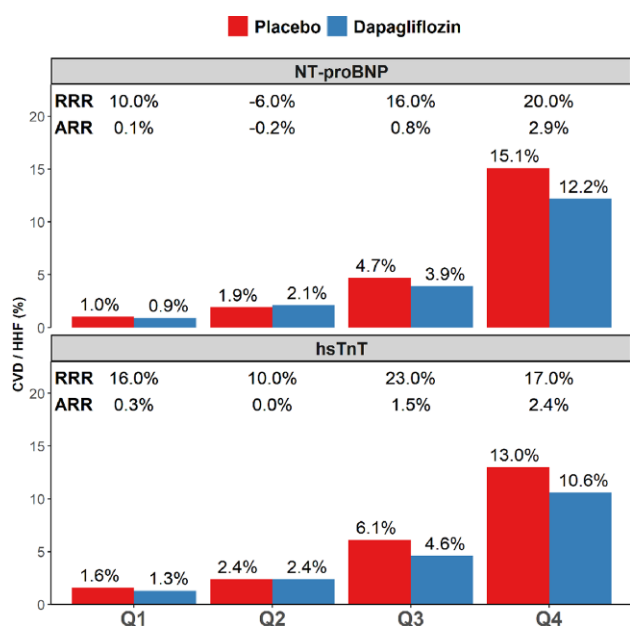
Fig. 1 | FPS 3/3-1

Methods: This was a prespecified biomarker study from DECLARE TIMI 58, a randomized, double-blind, placebo-controlled CV outcomes trial of dapagliflozin in pts with. Baseline NT-proBNP and hsTnT levels were measured in the TIMI Biomarker Core Laboratory in 14,565 pts (median follow-up 4.2 y). Patients were stratified by baseline biomarker levels. Relative risk ratios using hazard ratios and absolute risk reductions using Kaplan-Meier event rates at 4 years with dapagliflozin were calculated for CVD/HHF within biomarker quartiles.

Results: The median baseline NT-proBNP and hsTnT levels were 75 pg/mL (IQR 35–165) and 10.2 pg/mL (IQR 6.9–15.5), respectively. Patients with higher levels of NT-proBNP and hsTnT had higher KM event rates of CVD/HHF (Q4 vs Q1: NT-proBNP: 13.7 % vs 1.0 %; hsTnT: 11.8 % vs 1.4 %; P-trend <0.001). Dapagliflozin consistently reduced the relative risk of CVD/HHF regardless of baseline NT-proBNP (P INT 0.72) or hsTnT quartiles (P INT 0.93). However, given their higher baseline risk, pts with elevated levels of NT-proBNP and/or hsTnT tended to derive even greater absolute risk reduction with dapagliflozin [ARR in Q4 for NT-proBNP 2.9 % (NNT 35); hsTnT 2.4 % (NNT 42)] [Figure].

Conclusion: Patients with higher NT-proBNP or hsTnT levels are at increased risk of CV death and HHF. Dapagliflozin reduced the risk of CV death/HHF irrespective of NT-proBNP and hsTnT levels, with greater absolute risk reductions seen in pts with higher baseline biomarker levels.

Left ventricular size in aortic regurgitation: Sex-specific thresholds for the definition of severe AR on echocardiography and CMR

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Introduction: Left ventricular (LV) dilatation is a key feature in patients with chronic aortic regurgitation (AR). While echocardiography is the primary imaging tool for assessment of LV morphology, cardiovascular magnetic resonance imaging (CMR) is increasingly being used for accurate quantification of AR and LV size and function. However, specific cut-off values for LV volumes to define severe AR have never been established for either imaging modality.

Methods: We performed CMR in consecutive patients with at least mild AR on echocardiography. CMR analysis included phase-contrast velocity-encoded imaging for the measurement of regurgitant fraction (RegF) at the sinotubular junction. We assessed LV end-diastolic volumes (LVEDV) with echocardiography and CMR, and indexed LVEDV to body surface area (LVEDVi.BSA). Based on previous literature, we defined severe AR as RegF ≥30 % on CMR. We used logistic regression models, adjusted for age and sex, to test the association between various LV size parameters and severe AR, and used C-statistics to establish sex-specific LVEDV and LVEDVi.BSA cut-off values.

Results: Results. 261 consecutive patients (62 % male, 59.8 ±20.8 y/o) were included. 156 (59.8 %) had at least moderate AR by echocardiography. On CMR, mean RegF was 17.4 ±17.6 % and a total of 57 (21.8 %) had severe AR, defined by a RegF ≥30 %. There was good correlation between LV size parameters on echocardiography and CMR (r >0.81 and p <0.001 for all), however, CMR values were consistently higher compared to echocardiography (p <0.001 for all). Logistic regression models revealed a similar performance of echocardiographic and CMR parameters in identifying severe AR (Fig. 1A; adjusted odd ratios are displayed per 1-SD increase). By ROC analysis, the optimal cut-off values to detect severe AR differed substantially between sexes and imaging modalities. Specific LV size thresholds indicating severe AR are displayed in Fig. 1B.

Conclusion: Echocardiography and CMR perform similarly well in identifying LV dilatation as an indicator of severe AR. However, our study highlights the importance of using sex-specific thresholds for improved diagnostic accuracy. Furthermore, LV dilatation is less sensitive to detect severe AR in women compared to men.

Association with severe AR

		Echo Adj. OR (95% CI) per 1-SD increase	CMR Adj. OR (95% CI) per 1-SD increase
All	LVEDV	3.95 (2.52 - 6.17)	4.12 (2.56 - 6.63)
	LVEDV i.BSA	4.10 (2.62 - 6.43)	4.10 (2.60 - 6.47)
Men	LVEDV	4.26 (2.57 - 7.05)	3.74 (2.30 - 6.08)
	LVEDV i.BSA	4.83 (2.81 - 8.31)	4.17 (2.49 - 6.97)
Women	LVEDV	2.00 (1.05 - 3.84)	2.81 (1.42 - 5.54)
	LVEDV i.BSA	2.01 (1.04 - 3.92)	2.58 (1.33 - 5.00)

a
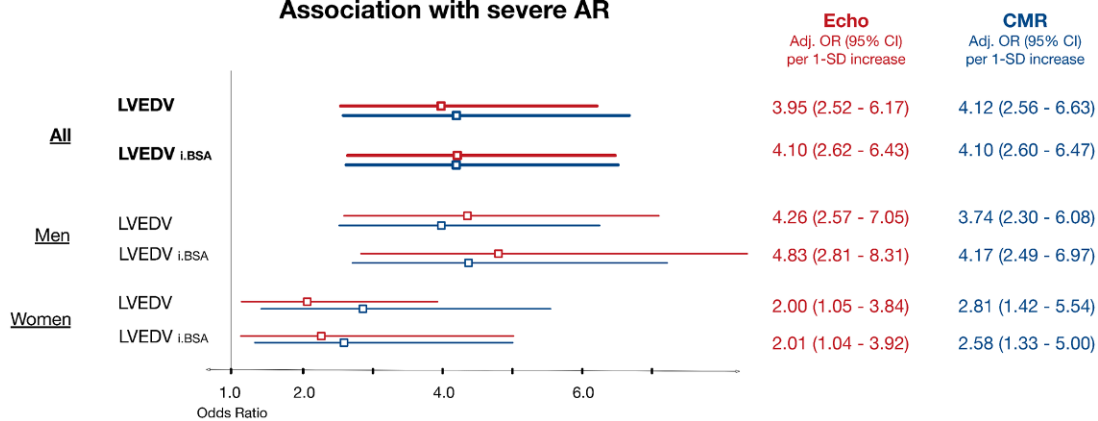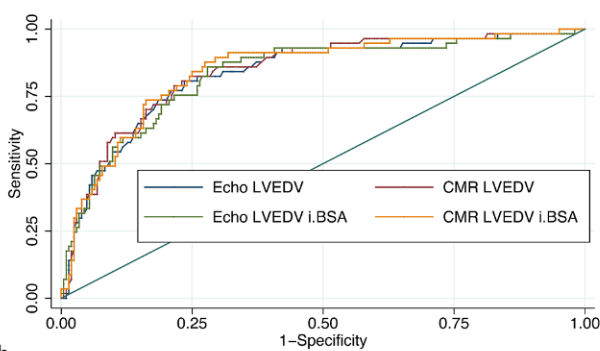
LV size parameter	AUC	Cut-off	Sensitivity / Specificity
Men			
Echo LVEDV	0.83	152 ml	0.85 / 0.69
CMR LVEDV	0.83	216 ml	0.81 / 0.73
Echo LVEDV$_{i.BSA}$	0.83	72 ml/m^2	0.90 / 0.64
CMR LVEDV$_{i.BSA}$	0.83	113 ml/m^2	0.81 / 0.74
Women			
Echo LVEDV	0.72	116 ml	0.78 / 0.79
CMR LVEDV	0.75	142 ml	0.78 / 0.76
Echo LVEDV$_{i.BSA}$	0.70	62 ml/m^2	0.78 / 0.74
CMR LVEDV$_{i.BSA}$	0.73	92 ml/m^2	0.67 / 0.87

b

Abb. 1 | FPS 3/3-2

FPS 3/3-3

Cardiac magnetic resonance derived global longitudinal strain outperfoms established functional parameters in prognostication after ST-elevation myocardial infarction

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Introduction: Although left ventricular ejection fraction (LVEF) is recommended for left ventricular (LV) systolic function assessment and risk stratification of patients with ST-elevation myocardial infarction (STEMI), its prognostic value is limited. Other measures of LV function such as global longitudinal strain (GLS) and mitral annular plane systolic excursion (MAPSE) might provide additional prognostic information post-STEMI. However, comprehensive investigations comparing these parameters in terms of prediction of hard clinical events following STEMI are lacking so far.

Objective: We aimed to investigate the comparative prognostic value of LVEF, MAPSE and GLS by cardiac magnetic resonance (CMR) imaging in acute STEMI patients.

Methods: This observational study included 407 consecutive acute STEMI patients treated with primary percutaneous coronary intervention (PCI). Comprehensive CMR investiga-
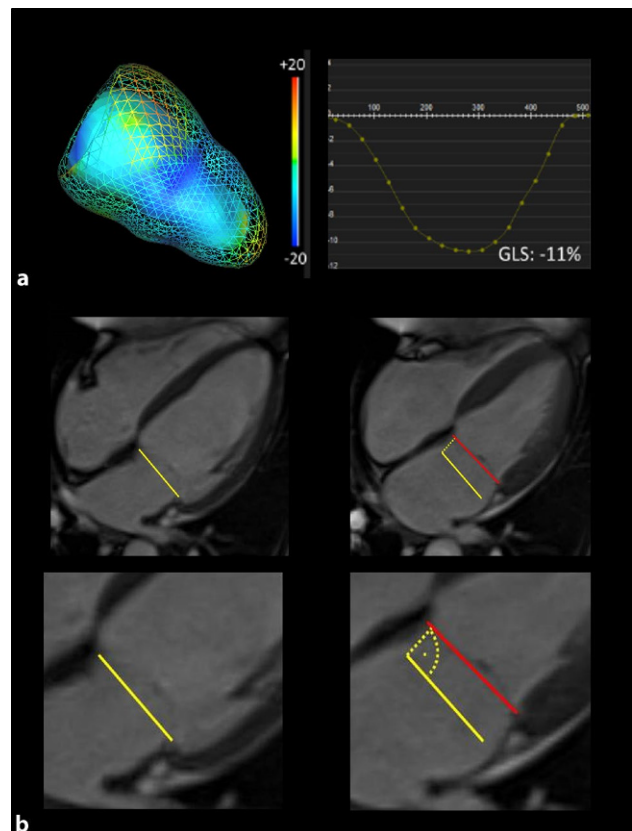
Abb. 1 | FPS 3/3-3 Assessment of 3D-GLS (A) and MAPSE (B) by CMR

Abb. 2 | FPS 3/3-3 Multivariable Cox regression analysis for the prediction of MACE

	Univariable		Multivariable	
	HR (95%CI)	p-value	HR (95%CI)	p-value
Model A				
LV EF, %	0.95(0.92-0.98)	**<0.001**	-	-
LV GLS, %	1.22(1.10-1.35)	**<0.001**	1.22(1.10-1.35)	**<0.001**
LV GRS, %	0.95(0.92-0.99)	**0.007**	-	-
LV GCS, %	1.12(1.05-1.19)	**<0.001**	-	-
Septal MAPSE, mm	0.81(0.72-0.91)	**<0.001**	-	-
Model B				
LV GLS, %	1.22(1.10-1.35)	**<0.001**	1.22(1.11-1.35)	**<0.001**
IS, % of LVMM	1.02(0.9-1.04)	0.212	-	-
MVO, % of LVMM	1.11(1.03-1.20)	**0.010**	-	-
Model C				
LV GLS, %	1.22(1.10-1.35)	**<0.001**	1.22(1.10-1.34)	**<0.001**
Hypertension	2.18 (1.09-4.38)	**0.028**	2.07(1.03-4.17)	**0.043**
Diabetes mellitus	2.30 (1.01-5.20)	**0.046**	-	-
Number of affected vessels	1.34(0.89-2.04)	0.162	-	-
TIMI flow post-PCI	0.65(0.47-0.91)	**0.011**	0.64(0.46-0.90)	**0.010**

tions were performed 3 [interquartile range (IQR): 2–4] days after PCI to determine LVEF, GLS and MAPSE as well as myocardial infarct characteristics. Primary endpoint was the occurrence of MACE defined as composite of death, re-infarction and congestive heart failure.

Results: During a follow-up of 21 [IQR: 12-50] months, 40 (10 %) patients experienced MACE. Patients with MACE showed significantly lower LVEF (49 % vs. 53 %, $p = 0.005$) and MAPSE (7.9 mm vs. 9.1 mm, $p = 0.001$), as well as higher GLS values (–10.2 % vs. -12.3 %, $p < 0.001$). GLS showed the highest prognostic value with an area under the curve (AUC) of 0.71 (95 % CI 0.63–0.79; $p < 0.001$) compared to MAPSE (AUC: 0.67, 95 % CI 0.58–0.75; $p = 0.001$) and LVEF (AUC: 0.64, 95 % CI 0.54–0.73; $p = 0.005$). After multivariable analysis, GLS emerged as independent predictor of MACE (HR: 1.22, 95 % CI 1.11–1.35; $p < 0.001$). Of note, GLS remained associated with MACE ($p < 0.001$) even after adjustment for infarct size and microvascular obstruction.

FPS 3/3-4

T1-mapping and echo-global longitudinal strain are early markers of cardiac involvement in patients with Fabry disease

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Introduction: Fabry cardiomyopathy (FD-CMP) is characterized by left ventricular hypertrophy and progressive myocardial fibrosis with consecutive functional impairment. T1 mapping by cardiac magnetic resonance imaging (CMR), indicating glycosphingolipid accumulation, was found to be a useful tool in patients with Fabry disease. Additionally, mechanical dysfunction can be detected by abnormal global longitudinal strain (GLS). Timely treatment with enzyme replacement therapy (ERT) or chaperones may prevent progression of FD-CMP. We sought to examine CMR and GLS as cardiac imaging modalities for early detection of cardiac involvement.

Methods: Cardiac magnetic resonance imaging including measurement of Late Gadolinium Enhancement (LGE), representing fibrosis, left ventricular ejection fraction (LVEF), left ventricular mass (LVM) and T1-mapping as well as echocardiography with measurement of global longitudinal strain (GLS) were performed as part of the regular check-ups for patients with Fabry disease.

Results: The study cohort of 30 FD patients (mean age 44.2 ± 14.2 years, 53 % female) in different stages of disease showed reduced GLS values already at the time of baseline echocardiography (mean –17.2 ± 4.2 %), correlating with LVEF (mean 67.7 ± 10.7 %, r = –0.445; $p = 0.043$) Baseline native T1 relaxation times (T1) were 910 ± 88 ms and median LVM was 163 g (IQR 114–181). Follow-up after 39 ± 18 months revealed significantly declined GLS values compared to baseline (–15.51 %; $p = 0.009$) correlating negatively with T1 times (r = –0.687; $p = 0.03$), while

LVEF and the extent of LGE was not changed significantly compared to baseline. Baseline T1 correlated with worsening of GLS (r = 0.67; p = 0.033), while LGE and LVEF did not correlate with the course of GLS. ERT appeared to have no influence on the extent of GLS impairment.

Conclusion: Low native CMR T1 and GLS may serve as early indicators for cardiac involvement in FD-CMP and correlate with disease progression while LGE and LVEF were inappropriate to assess slight deterioration of left ventricular function.

FPS 3/3-5

Validation of the ESC/EASD cardiovascular risk stratification model in diabetic patients

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Introduction: Recently, the European Society of Cardiology (ESC) and European Association for the Study of Diabetes (EASD) introduced a new cardiovascular (CV) risk stratification model to aid further treatment decisions in individuals with diabetes. Our study aimed to validate the ESC/EASD CV risk assessment in type 2 diabetes mellitus (T2 DM) patients for the first time and to compare the prognostic significance of NT-proBNP with the ESC/EASD CV risk stratification model.

Methods: We prospectively included 2186 T2 DM patients with a median follow-up of 60 months. Routine laboratory parameters including NT-proBNP were assessed. Patients were categorized as moderate, high and very high risk according to the respective ESC/EASD CV risk categories. The primary outcome measure was death from any cause. The two secondary outcomes were defined as unplanned hospitalization for cardiovascular and cardiac events.

Results: The ESC/EASD CV risk stratification model could only be applied to 1807 patients, 33 (1.5 %) were classified as moderate, 321 (14.9 %) as high and 1453 (67.6 %) as very high risk. Both, the ESC/EASD risk stratification model and NT-proBNP were associated with CV outcome (CV events: adj. HR: 1.47 [1.09–1.98], p = 0.011 and adj. HR per IQR: 2.61 [2.27–3.01], p < 0.0005; cardiac events: adj. HR: 1.57 [1.09–2.25], p = 0.016 and adj. HR per IQR: 2.94 [2.50–3.47], p < 0.0005; respectively) but only NT-proBNP proved to independently predict all-cause mortality (adj. HR per IQR: 2.32 [1.93–2.80], p < 0.0005). Fig. 1 displays the Kaplan–Meier curves for the respective endpoints. NT-proBNP outperformed the ESC/EASD CV risk stratification model as determined by C-statistic (CV events: 0.74 vs 0.55, p < 0.001; cardiac events: 0.76 vs 0.54, p < 0.001; all-cause mortality: 0.73 vs 0.52, p < 0.001) and net reclassification improvement of 69 % for CV events, 73 % for cardiac events and 67 % for all-cause death (p < 0.001 for all).

Conclusion: NT-proBNP measurement provides a valid tool to identify T2 DM patients at risk and shows superiority in terms of risk prediction and discriminatory power compared to the ESC/EASD CV assessment.

ESC/EASD CV RISK STRATIFICATION MODEL
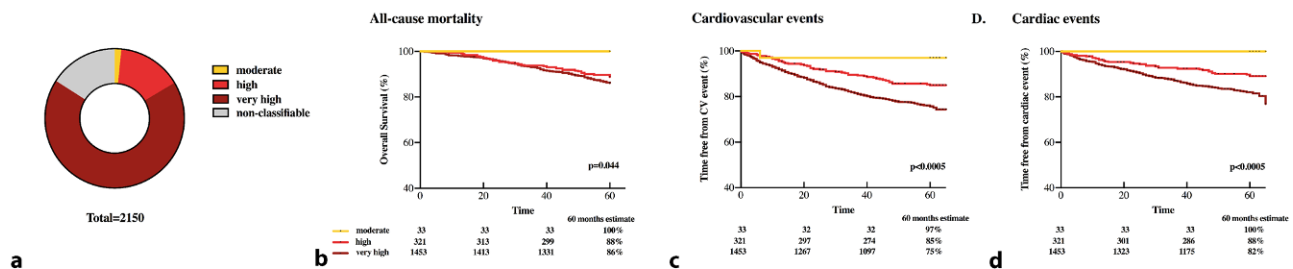
NT-proBNP

Abb. 1 | FPS 3/3-5

Abb. 1 | FPS 3/3-6

Subgroup	Dapagliflozin % (n) (N=8446)	Placebo % (n) (N=8427)	Hazard Ratio	P-INT for HR	Absolute Risk Difference	P-INT for ARD
CV death/HHF						
No CKD	3.4%	3.9%		0.29		0.002
eGFR<60 or UACR>30	7.5%	8.5%				
eGFR<60 and UACR>30	9.4%	17.7%				
MACE						
No CKD	7.2%	7.5%		0.62		0.15
eGFR<60 or UACR>30	11.6%	11.9%				
eGFR<60 and UACR>30	14.5%	19.8%				
HHF						
No CKD	1.5%	2.1%		0.76		0.001
eGFR<60 or UACR>30	4%	5%				
eGFR<60 and UACR>30	6.3%	11.9%				
CV death						
No CKD	2.2%	1.9%		0.13		0.14
eGFR<60 or UACR>30	4.3%	4.4%				
eGFR<60 and UACR>30	3.9%	7.5%				
ACM						
No CKD	4.9%	4.4%		0.018		0.017
eGFR<60 or UACR>30	8.3%	10.1%				
eGFR<60 and UACR>30	12.2%	16.4%				

Hazard Ratio scale: 0.25 0.50 0.75 1.00 1.50 — Favors Dapa ← → Favors Placebo

Absolute Risk Difference scale: -15 -10 -5 0 3 — Favors Dapa ← → Favors Placebo

FPS 3/3-6

Effect of dapagliflozin on cardiovascular outcomes in patients with type 2 diabetes according to baseline renal function and albuminuria status: Insights from DECLARE-TIMI 58

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Introduction: Renal dysfunction including both reduced estimated glomerular filtration rate (eGFR) and the presence of albuminuria have each been shown to predict cardiovascular (CV) outcomes. Sodium glucose cotransporter 2 inhibitors (SGLT2i), which promote glucose excretion in the kidneys, reduce CV events and hospitalizations for heart failure (HHF) in patients with type 2 diabetes mellitus (T2 DM). The objective of this study was therefore to analyze the CV efficacy of dapagliflozin according to baseline renal function and albuminuria status in DECLARE-TIMI 58.

Methods: The DECLARE-TIMI 58 trial compared dapagliflozin vs. placebo in 17,160 patients with T2 DM and a creatinine clearance >60 ml/min/1.73 m² at enrollment. The dual primary endpoints were CV death/HHF and MACE (MI, stroke, CV death). We categorized patients according baseline eGFR [<60 vs. ≥60 ml/min/1.73 m² according to the CKD-EPI formula] and urinary albumin:creatinine ratio (UACR) [<30 vs. ≥30 mg/g]. Cox regression models with interaction testing were applied.

The Gail-Simon test was used to test for interaction of the absolute risk differences.

Results: In total, 5198 (30.3 %) patients had albuminuria (UACR 30–300: $n = 4029$; UACR >300: $n = 1169$) and 1265 (7.4 %) had an eGFR <60 ml/min/1.73 m². Accordingly, 10.958 (63.9 %) patients had no manifestation of CKD, 5367 (31.3 %) had either an eGFR <60 ml/min/1.73 m² or albuminuria, and 548 (3.2 %) patients had both manifestations. Patients with more abnormal markers had higher event rates for CV death/HHF (KM event rates at 4 years of 3.9 %, 8.3 %, 17.4 %) and MACE (7.5 %, 11.7 %, and 18.9 %) for no, 1, or 2 markers of CKD, respectively. The relative risk reductions for CV death/HHF and MACE were generally consistent across the subgroups (both P-interaction >0.29), though numerically greatest (42 %) in patients with reduced eGFR and albuminuria. However, the absolute risk difference increased substantially in patients with greater kidney damage (absolute risk difference of CV death/HHF: –0.5 %, –1.0 %, and –8.3 %, respectively; P-INT for ARD 0.002; Figure). See figure for MACE and component outcomes.

Conclusion: Patients with baseline renal disease had higher rates of adverse CV outcomes. Dapagliflozin reduced events with generally consistent relative risk, but reduced the absolute risk of CVD/HHF by the greatest amount in patients with kidney disease evidenced by both reduced eGFR and albuminuria.

FPS 3/3-7

Adaptive development of concomitant secondary mitral and tricuspid regurgitation after TAVR

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Introduction: Concomitant secondary atrioventricular regurgitation is frequent in patients with severe aortic stenosis scheduled for transcatheter aortic valve replacement (TAVR). The future implications of leaving associated valve lesions untreated after TAVR remain unknown. Aim of the present study was to characterize the evolution of concomitant secondary
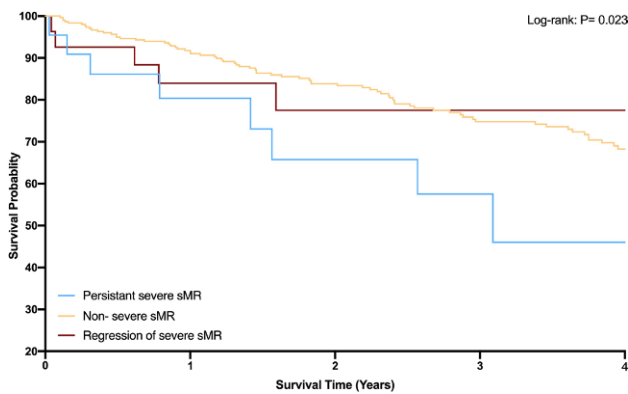
Fig. 1 | FPS 3/3-7
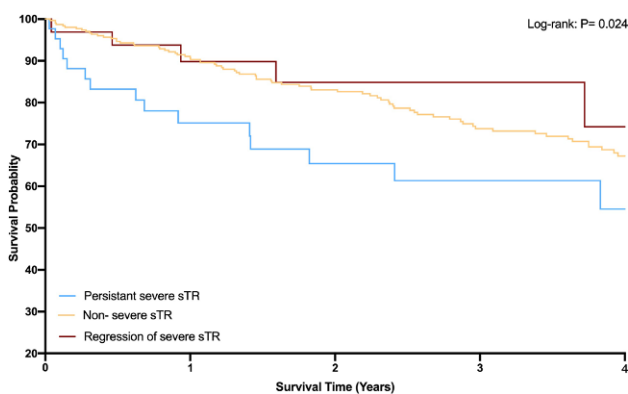
Abb. 2 | FPS 3/3-7

atrioventricular regurgitations and to evaluate their impact on long-term prognosis.

Methods: We prospectively enrolled 429 consecutive TAVR patients. All patients underwent comprehensive clinical, laboratory, and echocardiographic assessments prior to TAVR, at discharge, and yearly thereafter. All-cause mortality was chosen as primary study endpoint.

Results: At baseline, severe concomitant secondary mitral regurgitation (sMR) was present in 54 (13 %) and severe concomitant secondary tricuspid regurgitation (sTR) in 75 patients (17 %). After TAVR 59 % of patients with severe sMR at baseline experienced sMR regression, whereas analogously sTR regressed in 43 % of patients with severe sTR at baseline. Persistence of sTR and sMR were associated with excess mortality after adjustment for our bootstrap-selected confounder model with an adjusted HR of 2.44 (95 %CI 1.15–5.20, $P = 0.021$) for sMR and of 2.09 (95 % CI 1.20–3.66, $P = 0.01$) for sTR. Furthermore patients showing regression of atrioventricular regurgitation exhibited survival rates indistinguishable to those seen in patients without concomitant atrioventricular regurgitation (sMR: $P = 0.83$; sTR: $P = 0.74$)

Conclusion: Concomitant secondary atrioventricular regurgitation in patients with severe AS is a highly dynamic process with up to half of all patients showing regression of associated valvular regurgitation after TAVR and subsequent favorable post-interventional outcome. Persistent atrioventricular regurgitation is a major determinant of TAVR futility and proposes a window of early sequel intervention.

FPS 3/3-8

Periprocedural outcomes of patients treated with the MitraClip device for severe tricuspid regurgitation

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Introduction: Tricuspid regurgitation (TR) is common and has a serious prognostic impact for the affected patients. TR is widely undertreated due to the high perioperative risk of isolated tricuspid surgery and other treatment options are limited. Transcatheter treatment of TR with the MitraClip system (MC) has been shown to be safe and feasible to reduce TR severity in certain centers of excellence. Here we report the clinical and procedural characteristics as well as complications and periprocedural outcomes of the first 14 patients treated with the MC for TR at the LKH University Clinic of Graz.

Methods: Symptomatic patients with severe to torrential TR which were assessed eligible through our interdisciplinary heart team were treated with MC for TR. Procedural characteristics, complications and clinical outcomes were obtained by evaluation of the procedural protocol, the medical record and the stored echocardiographic loops. Procedural success was defined as reduction of TR severity >1 grade and was evaluated by periprocedural TOE. Acute device success was defined as persistent grasping and attachment of the clip on two leaflets. Procedural safety was assessed by the evaluation of periprocedural complications.

Results: From April 2019 to February 2020 14 patients with NYHA II (33 %) or III (67 %) underwent treatment for severe to torrential tricuspid regurgitation by transcatheter implantation of one or more MC on the tricuspid valve. From 14 patients 9 received combined intervention on the mitral and tricuspid valve, and 5 on the tricuspid valve alone. 1- (14 % of the cases), 2- (36 %), 3- (21 %) or 4- (29 %) clips were implanted per patient. Acute procedural success was reached in 93 % of the cases with a mean reduction of tricuspid regurgitation severity of 2.0 (+/- 0.82) grades as evaluated by periprocedural TOE. Acute device success was reached in 35 of 37 clips (94.6 %). Mean procedural duration was 192 min for the combined and 143 min for isolated TR procedure. Periprocedural death, survived resuscitation, myocardial infarction, non-fatal stroke, TIA or device embolization did not occur in any of the treated patients. Single leaflet attachment occurred in 1 of 14 patients (7 %). Severe bleeding with the need for transfusion of blood products occurred in 1 patient but in the context of concomitant surgery for peripheral artery disease during the same admission.

Conclusion: Transcatheter treatment of severe TR with MC has shown a high procedural safety and seems to be feasible to reduce periprocedural TR-severity in our cohort.

FEATURED POSTER SESSION 4: COVID-19 UND HERZ:

BA-1

ACE2 interaction networks in COVID-19: a physiological framework for prediction of outcome and potential therapeutics in patients with cardiovascular risk factors

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Introduction: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (coronavirus disease 2019; COVID-19) is associated with adverse outcome in patients with cardiovascular disease (CVD). We aimed to characterize the interaction between SARS-CoV-2 and Angiotensin Converting Enzyme 2 (ACE2) functional networks with focus on CVD.

Methods: Using bioinformatic tools and network medicine approaches we investigated ACE2 tissue expression and described ACE2 interaction network which could be affected by SARS-CoV-2 infection. We identified top ACE2 interactors, miRNAs which are shared regulators between the ACE2, virus-infection related proteins and heart interaction networks, using lung and nervous system networks as a reference. We also identified main SARS-CoV-2 risk groups and performed drug predictions for them.

Results: We found the same range of ACE2 expression confidence in respiratory and cardiovascular systems (averaging 4.48 and 4.64, respectively). Analysing the complete ACE2 interaction network, we identified 11 genes (ACE2, DPP4, ANPEP, CCL2, TFRC, MEP1A, ADAM17, FABP2, NPC1, CLEC4M, TMPRSS2) associated with virus-infection related processes. Previously described genes associated with cardiovascular risk factors DPP4, CCL2 and ANPEP were extensively connected with top regulators of the ACE2 network, including ACE, INS and KNG (Fig. 1). Enrichment analysis revealed several disease phenotypes associated with interaction networks of ACE2, heart tissue, and virus-infection related protein, with the strongest associations with the following diseases (in decreasing rank order): obesity, hypertensive disease, non-insulin dependent diabetes mellitus, congestive heart failure, and coronary artery disease. ACE2 interaction network connected directly with virus protein S through ACE2, CLECAM4 and TMPRSS2 (Fig. 1). We described for the first time microRNAs-miR (miR-302c-5p, miR-1305, miR-587, miR-26b-5p, and mir-27a-3p), which were common regulators of the three networks: ACE2, heart tissue and virus-infection related proteins (Fig. 2). We proposed a drug prediction model, which identified valproic acid, acetaminophen, estradiol, and cyclosporin A among others as potential compounds for COVID-19 treatment in patients with underlying heart failure or at risk for CVD.
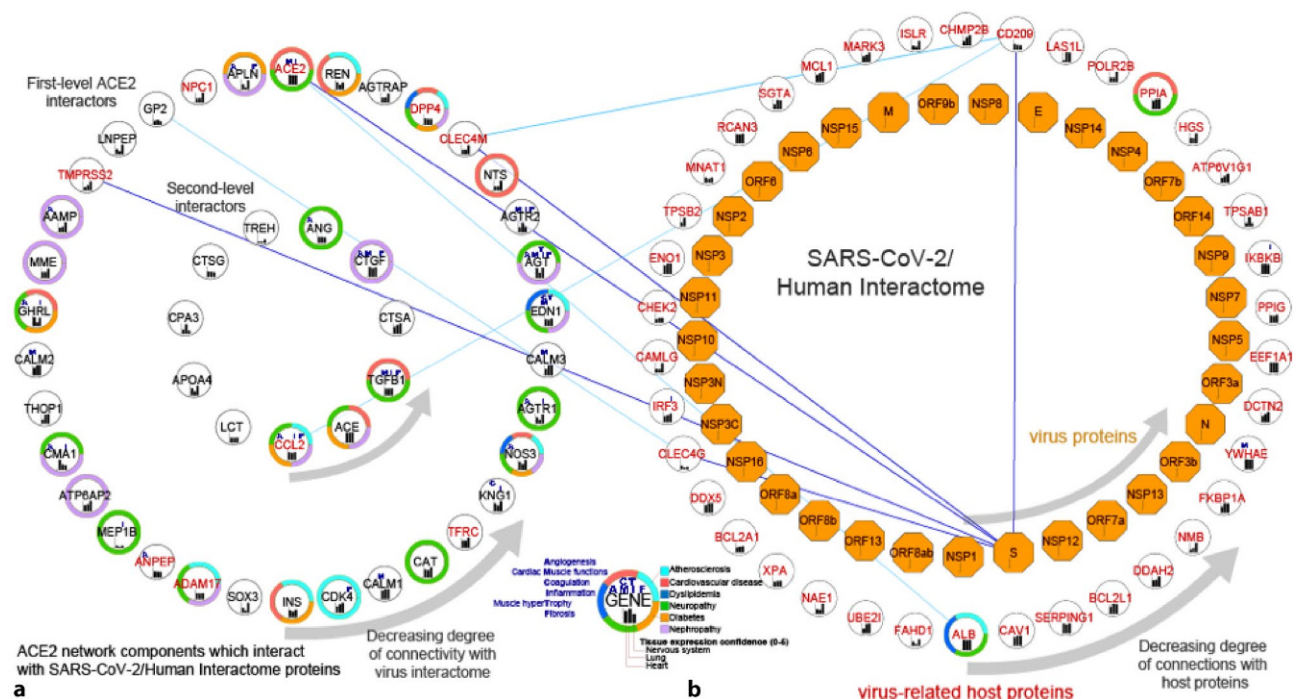
Fig. 1 | BA-1 Combined ACE2 network with SARS-CoV-2/Human interactome. (**a**) depicts ACE2 network components which interact with SARS-CoV-2/Human interactome proteins. (**b**) depicts the SARS-CoV-2/Human interactome. Nodes in the network A are sorted by number of connections with virus interactome. Nodes from the network B are sorted by the number of interactions with human proteins. Dark blue edges are showing connections between ACE2 network with virus protein S. Bright blue edges are showing second level interactors of virus glycoprotein S. Virus proteins are shown as orange octagons, while virus-infection related human proteins have red labels. Notice that ACE2 interaction network connects directly with virus protein S through ACE2, CLECAM4 and TMPRSS2. Also, SARS-CoV-2 interactome strongly connects with ACE2 network through INS, CDK4, CCL2 and ALB, all of them associated with atherosclerosis processes
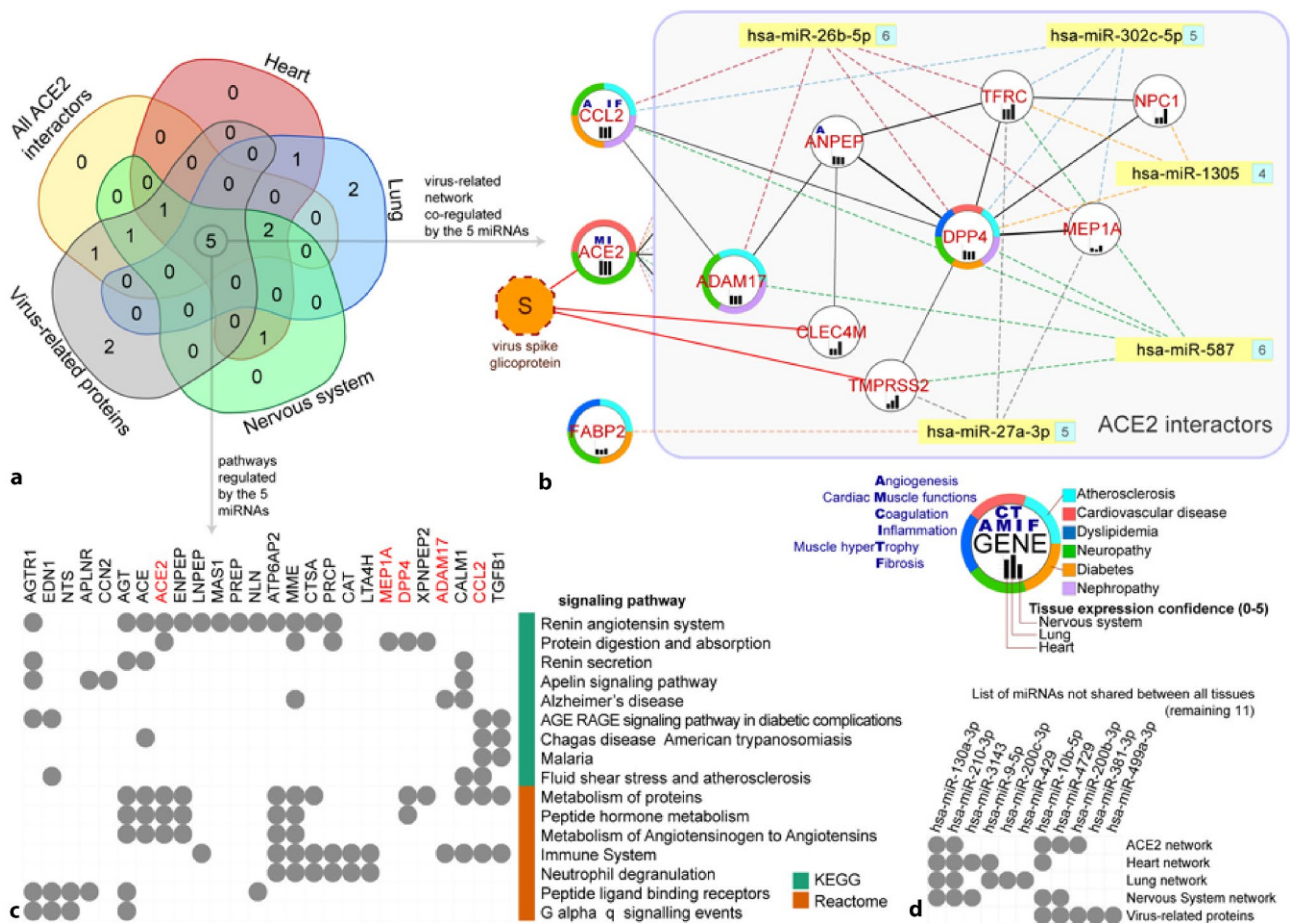
Fig. 2 | BA-1 Potential miRNA modulators of ACE2 network in COVID-19. (**a**) Venn diagram of the top overlapping 10 miRNAs of each network (i. e., complete ACE2 network, and its subnetworks expressed in heart, lung, nervous system, and virus-infection related proteins) regulating the highest number genes. Numbers in the diagram depict the number of shared miRNAs. (**b**) Interaction network between virus-infection related proteins (red labels) and 5 top miRNAs shared between analyzed networks. Numbers on the right side of the miRNAs depict the number of targeted genes within the network. CCL2 and FABP2 genes are not a direct interactors of the ACE2, so they are presented outside of the ACE2-interactors box. „S" refers to SARS-CoV-2 S spike glycoprotein. (**c**) Signaling pathways enriched among 36 targets regulated by the 5 miRNAs shared between all analyzed networks. Genes marked with red are associated with virus-infection related processes. (**d**) Presence of other top miRNAs in the analysed networks

Conclusion: Our study provides novel information regarding the complexity of signaling pathways affected by SARS-CoV-2 and proposes predictive tools as miR towards personalized diagnosis and therapy in COVID-19. Additionally, our study provides a list of available compounds that might have therapeutic potential for treatment or prevention of adverse outcome in patients with COVID-19 and CVD.

BA-2

Effect of the COVID-19 Pandemic on treatment delays in patients with ST-segment elevation myocardial infarction

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Introduction: Coronavirus disease 19 (COVID-19) and its associated restrictions could affect ischemic times in patients with ST-segment elevation myocardial infarction (STEMI). The objective of this study was to investigate the influence of the COVID-19 outbreak on ischemic times in consecutive all-comer STEMI patients.

Methods: We included consecutive STEMI patients ($n = 163$, median age: 61 years, 27 % women) who were referred to seven tertiary care hospitals across Austria for primary percutaneous coronary intervention between 24 February 2020 (calendar week 9) and 5 April 2020 (calendar week 14). The number of patients, total ischemic times and door-to-balloon times in temporal relation to COVID-19-related restrictions and infection rates were analyzed.

Fig. 1 | BA-2

Results: While rates of STEMI admissions decreased (calendar week 9/10 ($n=69$, 42 %); calendar week 11/12 ($n=51$, 31 %); calendar week 13/14 ($n=43$, 26 %)), total ischemic times increased from 164 (interquartile range (IQR): 107–281) min (calendar week 9/10) to 237 (IQR: 141–560) min (calendar week 11/12) and to 275 (IQR: 170–590) min (calendar week 13/14) ($p=0.006$). Door-to-balloon times were constant ($p=0.60$). There was a significant difference in post-interventional Thrombolysis in myocardial infarction (TIMI) flow grade 3 in patients treated during calendar week 9/10 (97 %), 11/12 (84 %) and 13/14 (81 %; $p=0.02$). Rates of in-hospital death and re-infarction were similar between groups ($p=0.48$). Results were comparable when dichotomizing data on 10 March and 16 March 2020, when official restrictions were executed.

Conclusion: In this cohort of all-comer STEMI patients, we observed a 1.7-fold increase in ischemic time during the outbreak of COVID-19 in Austria. Patient-related factors likely explain most of this increase. Counteractive steps are needed to prevent further cardiac collateral damage during the ongoing COVID-19 pandemic.

BA-3

Telemonitoring of patients with atrial fibrillation during the COVID-19 pandemic–The TeleCheck-AF study

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Introduction: During the COVID-19 pandemic, traditional face-to-face outpatient consultations for arrhythmia patients had to be cancelled at our clinic, while it became increasingly difficult for these patients to visit doctors in private practice. To overcome this limitation, we implemented a remote on-demand mobile health (mHealth) infrastructure, which was based on a mobile phone app using photoplethysmography (PPG) technology allowing rate and rhythm monitoring through teleconsultations.

Methods: Patients who contacted our outpatient clinic with arrhythmia symptoms or whose appointments had to be postponed were given the opportunity to monitor their rhythm

Fig. 1 | BA-3 Schematic overview of the telemonitoring process. QR code is scanned, patient measures for one minute, dashboard view for clinician shows regular rhythm (green), atrial fibrillation (red) and unclear tracings (blue)

using "FibriCheck" within the "TeleCheck-AF" initiative for seven days. They received a QR code for installation of the software on their smartphone and were connected to the clinician's telemedicine portal. Patients were told to measure their heart rate three times per day and in case of symptoms. Clinicians assessed the tracings and contacted the patients if therapeutic steps were indicated. We retrospectively analysed all patients monitored with PPG between April 1st and June 30th 2020 at our institution.

Results: Thirty patients were included in this retrospective analysis. Median age was 56 years (range 37–74 years), 27 % were female, median CHADS-Vasc-Score was 1 (0–4). The majority (90 %) of patients had previously diagnosed AF, 63 % were included due to palpitations after pulmonary vein isolation, 20 % after cardioversion, 3 % for rate control therapy, 3 % before consultation and 10 % after non-AF ablations. Within one week, patients recorded 22 ±9 PPGs, three patients performed a second week of measurements after a clinical intervention. 660 tracings were analysed, 15 % were classified as AF, 1.7 % as atrial flutter, 5.3 % as sinus rhythm with premature beats, 1.3 % as symptomatic bradycardia and only 3.9 % showed unclear results. One or more clinical interventions during or after telemonitoring were performed in 47 % of all included patients, 27 % were scheduled for ablation, medication was changed in 23 %, cardioversions were scheduled in 17 %.

Conclusion: Rhythm monitoring with a PPG-based mHealth application was feasible and often resulted in clinical interventions. Due to its great availability, PPG-based follow-up for patients with known AF may close a diagnostic gap not only in times of a pandemic.

BA-4

Comparison and prognostic impact of electrocardiographic findings in COVID-19 pneumonia and non-COVID-19 pneumonia

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Introduction: Coronavirus disease (COVID-19) was first described at the end of 2019 in China and has since spread across the globe. Cardiovascular morbidity is high among patients with COVID-19 and electrocardiographic changes in COVID-19 pneumonia have not been fully studied.

Methods: This is a retrospective, cohort study of consecutive patients with COVID-19 pneumonia who had an electrocardiogram (ECG) within 4 h of admission. We tested the prognostic impact of an abnormal admission ECG on short-term mortal-
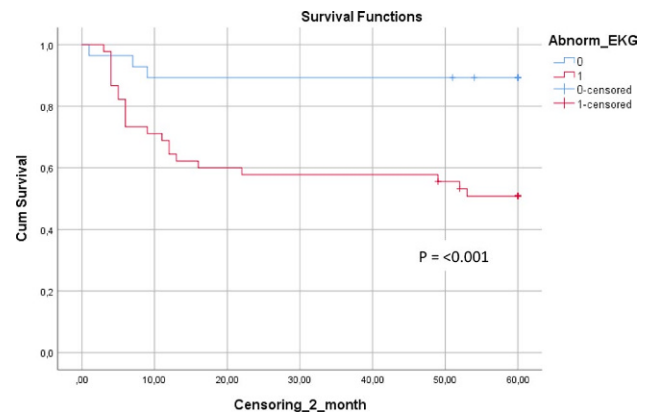
Abb. 1 | BA-4 Comparison and prognostic impact of electrocardiographic findings in COVID-19 pneumonia and non-COVID-19 pneumonia

ity and compared ECG findings among patients with COVID-19 pneumonia and an age- and gender matched subgroup of patients with non-COVID-19 pneumonia.

Results: A total of 73 patients with COVID-19 pneumonia were included in the final analyses and 60-day mortality was 34.2 %. The mean age of patients was 74 ±15.0 years and 60.2 % were men. The most common electrocardiographic findings were atrial fibrillation (31.5 %), left axis deviation (21.9 %) and bundle branch block (17.8 %). An abnormal admission ECG was found in 61.6 % of patients with COVID-19 pneumonia and was associated with increased 60-day mortality (crude hazard ratio [HR] 5.587, 95 % confidence interval [CI] 1.669–18.702, $P=0.005$), independent of clinical confounders (adjusted HR 3.691, 95 % CI 1.077–12.652; $P=0.038$) and comorbidities (adjusted HR 4.028, 95 % CI 1.133–15.625; $P=0.032$). A subgroup analysis of an age- and gender matched group of COVID-19 pneumonia ($n=47$) and non-COVID-19 pneumonia ($n=47$) revealed no significant electrocardiographic changes, except for a significantly higher heart rate in patients with non-COVID-19 pneumonia (median heart rate 97 bpm, range 80–118 bpm vs. median heart rate of 87 bpm, range 73–100 bpm; $P=0.001$).

Conclusion: Abnormal electrocardiographic findings are frequent in patients with COVID-19 pneumonia and associated with worse survival. Except for a higher heart rate in patients with non-COVID-19 pneumonia, we found no significant electrocardiographic differences among the two groups

Adjusted models	Abnormal ECG		
	HR	95% CI	P-value
Crude model	5.587	1.669-18.702	0.005
Clinical confounder clustera	3.691	1.077 – 12.652	0.038
Comorbidities blusterb	4.208	1.133 – 15.625	0.032

A adjusted for age and gender
B adjusted for history of heart failure, coronary artery disease, atrial fibrillation, arterial hypertension, diabetes mellitus, COPD and history of malignancy

Baseline characteristics	COVID-19 pneumonia (n=73)	Age- and gender matched subgroup		
		COVID-19 pneumonia (n=47)	Non-COVID-19 pneumonia (n=47)	P-value
Demographics				
Age, years (IQR)	74.0 +- 15.0	74.8 +- 11.1	74.6 +- 11.2	0.93
Sex (male), n (%)	44 (60.3)	29 (61.7)	29 (61.7)	1.0
Nursing home resident, n (%)	18 (24.7)	12 (25.5)	11 (23.4)	0.81
Symptoms and signs				
Fever, n (%)	56 (76.7)	35 (74.5)	27 (57.4)	0.08
Coughing, n (%)	41 (56.2)	27 (57.4)	12 (25.5)	0.002
Dyspnea, n (%)	46 (63.0)	29 (61.7)	32 (68.1)	0.52
GI-symptoms, n (%)	14 (19.2)	9 (19.1)	9 (19.1)	1.0
Temperature, °C (IQR)	38.3 (37.1-39.0)	38.1 (37.1-38.9)	37.5 (36.9-38.4)	0.17
Systolic blood pressure, mmHg (IQR)	130 (110-141)	130 (110-146)	143 (122-160)	0.022
Diastolic blood pressure, mmHg (IQR)	72 (65-90)	76 (65-90)	80 (70-90)	0.35
Mean arterial pressure, mmHg (IQR)	110 (97-125)	112 (96-126)	122 (107-140)	0.029
Comorbidities, n (%)				
Hypertension, n (%)	50 (68.5)	33 (70.2)	35 (74.5)	0.65
Diabetes mellitus, n (%)	21 (28.8)	14 (29.8)	13 (27.7)	0.82
Structural heart disease, n (%)	24 (32.9)	14 (29.8)	18 (38.3)	0.38
Coronary artery disease, n (%)	10 (13.7)	6 (12.8)	10 (21.3)	0.27
Heart failure, n (%)	17 (23.3)	11 (23.4)	9 (19.1)	0.61
Atrial fibrillation, n (%)	21 (28.8)	14 (29.8)	15 (31.9)	0.77
COPD, n (%)	8 (11.0)	6 (12.8)	8 (17.0)	0.56
History of malignancy, n (%)	15 (20.5)	9 (19.1)	14 (29.8)	0.23
Co-medication, n (%)				
Beta blocker, n (%)	29 (39.7)	17 (36.2)	16 (34.0)	0.89
RAS inhibitors, n (%)	36 (49.3)	22 (46.8)	22 (46.8)	1.0
MRA, n (%)	10 (13.7)	5 (10.6)	11 (23.4)	0.09
Calcium antagonist, n (%)	14 (19.2)	11 (23.4)	11 (23.4)	0.95
Diuretics, n (%)	17 (23.3)	10 (21.3)	18 (38.3)	0.06
Antiarrhythmics, n (%)	6 (8.2)	2 (4.3)	4 (8.5)	0.44
Oral anticoagulation, n (%)	15 (20.5)	11 (23.4)	8 (17.0)	0.47
60-day mortality, n (%)	25 (34,2)	16 (34.0)	8 (17.0)	0.06
Hospitalization length (IQR)	16 (9-29)	16 (9-27)	12 (10-18)	0.09

ECG findings	COVID-19 pneumonia (n=73)	Age- and gender matched subgroup		
		COVID-19 pneumonia (n=47)	Non-COVID-19 pneumonia (n=47)	P-value
Rhythm disturbances				
Heart rate (IQR)	87 (72-101)	87 (73-100)	97 (80-118)	0.01
Sinus tachycardia, n (%)	10 (19.2)	4 (8.5)	18 (38.3)	0.001
Atrial fibrillation, n (%)	17 (31.5)	11 (23.4)	9 (19.1)	0.61
Time intervals				
PR interval (IQR)	157 (138-174)	157 (139-174)	163 (145-195)	0.07
QRS interval (IQR)	96 (95-110)	92 (82-105)	92 (84-105)	0.78
QTc time (IQR)	440 (420-465)	437 (418-464)	441 (424-466)	0.75
QRS abnormalities				
Right axis deviation, n (%)	4 (5.5)	2 (4.3	1 (2.1)	1.0
Left axis deviation, n (%)	16 (21.9)	11 (23.4)	9 (19.1)	0.61
BBB, n (%)	13 (17.8)	6 (12.8)	9 (19.1)	0.40
RBBB, n (%)	6 (8.2)	2 (4.3)	3 (6.4)	1.0
LBB, n (%)	6 (8.2)	4 (8.5)	4 (8.5)	1.0
Sokolow index (IQR)	14 (10-19)	15 (10-20)	18 (12-22)	0.11
LVH, n (%)	2 (2.7)	1 (2.1)	4 (8.5)	0.36
Pathological Q wave, n (%)	5 (6.8)	2 (4.3)	3 (6.4)	1.0
PRWP, n (%)	14 (19.2)	9 (19.1)	11 (23.4)	0.61
Clockwise rotation, n (%)	20 (27.4)	14 (29.8)	9 (19.1)	0.20
Counterclockwise rotation, n (%)	17 (23.3)	11 (23.4)	14 (29.8)	0.52
Low voltage, n (%)	9 (12.3)	5 (10.6)	8 (17.0)	0.37
ST-segment and T-wave abnormalities				
ST-elevation, n (%)	2 (2.7)	2 (4.3)	0	0.24
ST-depression, n (%)	4 (5.5)	3 (6.4)	3 (6.4)	1.0
T-wave inversion, n (%)	9 (12.3)	4 (8.5)	7 (14.9)	0.52
Abnormal ECG	45 (61.6)	26 (55.3)	28 (59.6)	0.68

BA-5

Cardiovascular characteristics and intra-hospital outcome of COVID-19 inpatients at a tertiary referral centre

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Introduction: As of 27 July 2020, Sars-CoV-2 has infected more than 16 million people and caused nearly 650,000 deaths worldwide, while still rapidly spreading worldwide [1]. Retrospective studies have suggested that patients diagnosed with COVID-19 have a high prevalence of pre-existing cardiovascular diseases and cardiovascular risk factors and these are main determinants of outcome. Patients hospitalized with COVID-19 at our Tertiary Referral Centre were characterized in terms of cardiovascular disease, risk factors, and outcome.

Methods: Retrospective registry analysis identified 96 confirmed (PCR) COVID-19 positive patients who had been hospitalized at our Tertiary Referral Centre. Patients were classified based on their history of pre-existing cardiovascular disease and/or risk factors (arterial hypertension, diabetes mellitus, dyslipidaemia, smoking).

Results: 46.9 % of hospitalized COVID-19 positive patients were male and median age was 72.8 years (table 1). 74 patients (77.1 %) had had cardiovascular disease (CVD: 46.9 %) and/or CVD risk factors (69.8 %). The most common CVDs were atrial fibrillation (19.8 %), ischaemic heart disease (18.8 %), peripheral artery disease (15.6 %), stroke/TIA (14.6 %), heart failure (12.5 %), and valvular heart disease (10.4 %). The most frequent cardiovascular risk factors were hypertension (62.5 %), dyslipidaemia (37.5 %), diabetes (24.0 %), and history of smoking (18.8 %). Common chronic diseases were renal failure (CKD III, 31.3 %), malignant neoplasms (22.9 %), and COPD/asthma

Tab. 1 | BA-5 Patient Characteristics

characteristic	cardiovascular patients	non-cardiovascular patients
number	74 (77.1%)	22 (22.9%)
sex distribution	male: 32 (43.2%) female: 42 (56.8%)	male: 13 (59.1%) female: 9 (40.9%)
median age <50 years [%] 50-60 years [%] 60-70 years [%] 70-80 years [%] >80 years [%]	74.4 years 2 (2.7%) 11 (14.9%) 10 (13.5%) 24 (32.4%) 27 (36.5%)	67.3 years 5 (22.7%) 3 (13.6%) 3 (13.6%) 7 (31.8%) 4 (18.2%)
BMI [kg/m²]	28.2	24.8
length of hospitalisation	12.5 days	10.5 days
invasive ventilation	17.6%	4.5%
non-invasive ventilation	24.3%	36.4%
ICU	27.0%	22.7%
length of stay on ICU	15.4 days	14.2 days
respiratory failure during hosptalisation	36.5%	31.8%
death median age of death	29.7% 79.6 years	18.2% 83.8 years

(16.7%). 25 patients (26.0%) were admitted to the ICU, 14 patients (14.6%) required invasive ventilation, and 26 patients (27.1%) died. The occurrence of each of these events was significantly more common in men than in women: ICU (odds ratio: 2.57; p-value: 0.0495), invasive ventilation (OR: 5.18; p: 0.0170), and death (OR: 2.26; p: 0.0828). The median age of all deceased patients was 80.3 years (non-cv patients: 83.8 years; cv patients: 79.6 years) and 22 fatalities (84.6%) occurred in patients with CVD and/or risk factors. In patients with pre-existing CVDs, 16 out of 45 died, with increased mortality rates in comparison to non-cardiovascular patients using t-test especially in the following subgroups: heart failure (75%; p: 0.0006), ischaemic heart disease (61%; p: 0.0044) and history of myocardial infarction (75%; p: 0.0024).

Conclusion: The majority of patients hospitalized with COVID-19 at our centre had pre-existing cardiovascular disease and/or risk factors. The majority of COVID-19 positive patients and patients who died were older than 70 years of age. The presence of pre-existing cardiovascular diseases or risk factors increased the occurrence of adverse outcomes.

BA-6

Die Bedeutung der Telemedizin in Zeiten von COVID-19

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Einleitung: Im März 2020 wurde der Ausbruch der Coronavirusinfektion (corona virus disease 2019) – COVID-19 von der WHO als Pandemie ausgerufen. Seither wurden weltweit mehr als 15 Mio. Infektionen in mehr als 200 Ländern dokumentiert. Die Eindämmungsstrategie beinhaltete eine frühe Diagnose, Patientenisolierung und Quarantänemaßnahmen. Routinekontrollen im öffentlichen Gesundheitsbereich mussten drastisch reduziert und verschoben werden. Kardiologische und rhythmologische Gesellschaften haben zum Ausbau telemedizinischer Betreuung aufgerufen. Deshalb wird auch im Landesklinikum Wiener Neustadt seither vermehrt auf telemedizinische Versorgung von PatientInnen gesetzt, speziell in der Betreuung von PatientInnen mit implantierbaren Devices (Herzschrittmacher, CRT und ICD). Die Strategie zum Ausbau der Telemedizin wird folgend beschrieben.

Methoden: Im Landesklinikum Wiener Neustadt werden sämtliche Herzschrittmacher, CRT- und ICD-, sowie auch S-ICD-Devices implantiert und nachgesorgt. Eine telemedizinische Versorgung wird zwar schon seit Jahren angeboten, dennoch werden immer noch viele PatientInnen zu Kontrollen in die Ambulanz einbestellt. Mit den Quarantänemaßnahmen und Einschränkungen im öffentlichen Gesundheitssystem mussten viele dieser Kontrollen abgesagt bzw. verschoben werden. Eine optimale Versorgung der PatientInnen konnte somit nicht immer gewährleistet werden. Mit der COVID-19 Krise wurde daher eine Aufstockung der telemedizinischen Betreuung beschlossen und so werden laufend weitere PatientInnen eingeschlossen. Vor der Covid-19 Krise wurden im LK Wiener Neustadt 802 PatientInnen mit implantierbaren Devices digital telemedizinisch überwacht. In diesen Fällen konnte die Versorgung nach dem „Shut-down" weiterhin gewährleistet werden. PatientInnen, deren ambulante Kontrollen abgesagt werden mussten, wurden zumindest auf Batterieleistung im Rahmen der letzten Kontrollen überprüft. Dies bedeutete einen enormen zeitlichen Aufwand in der Durchsicht verschiedener Computerprogramme und es konnte auch keine sichere Prognose getroffen werden. Um lange Wartezeiten in der Ambulanz zu
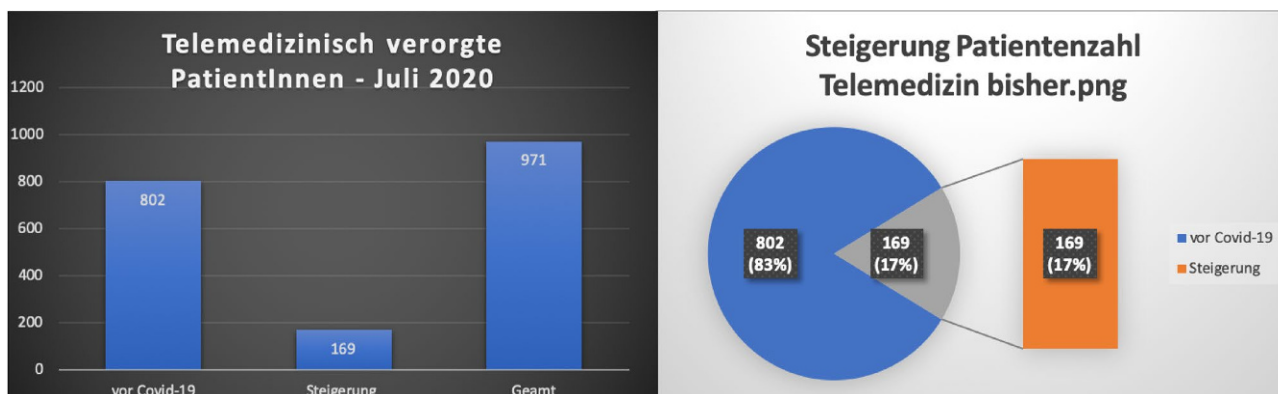
Abb. 1 | BA-6 Vergleich telemedizinisch versorgter PatientInnen vor und nach der COVID-19 Krise sowie Darstellung der Steigerung
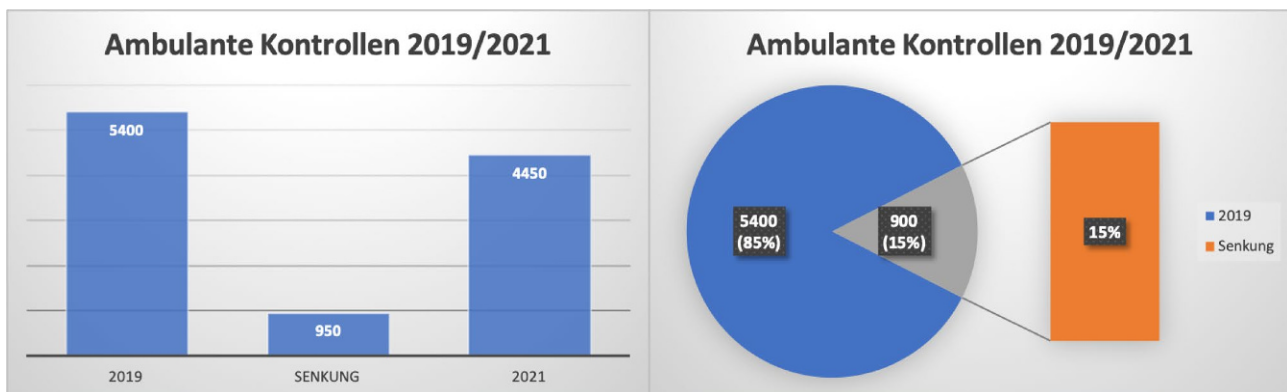
Abb. 2 | BA-6 Darstellung der erwarteten Senkung an ambulanten Kontrollen durch Aufstockung der telemedizinischen Versorgung im Jahr 2022 verglichen zu 2019

vermeiden wurden von den Firmen Mitarbeiter zu Verfügung gestellt, um die behandelnden ÄrztInnen zu entlasten und den Einschluss in die Telemedizin durchzuführen.

Resultate: Seit der Öffnung der Ambulanzen im April sind innerhalb von 3 Monaten zusätzlich 169 PatientInnen in die telemedizinische Versorgung eingeschlossen worden. Mittlerweile sind insgesamt 971 PatientInnen so überwacht, dies bedeutet eine Steigerung von 17 % und es werden laufend weitere PatientInnen eingeschlossen. Ende 2020 sollen so hochgerechnet ungefähr 1300 PatientInnen telemedizinisch betreut werden. Dies bedeutet eine Steigerung um 39 % im Vergleich zu 2019! Eine Senkung an ambulanten Kontrollen um mindestens 15 % wird dadurch erwartet. Im Jahr 2019 wurden durchschnittlich 20 PatientInnen täglich in der Device-Ambulanz des LK Wiener Neustadt begutachtet. Dies ergibt eine jährliche Anzahl an ungefähr 5400 ambulanten Kontrollen. Durch die Aufstockung der Telemedizin werden für 2021 nur noch ungefähr 4450 Kontrollen erwartet, also eine Senkung um mindestens 950 PatientInnen (15 %). Telemedizinisch können derzeit von einer Ärztin bzw. einem Arzt bis zu 50 Kontrollen an einem Arbeitstag durchgeführt werden. Behandlungsbedürftige PatientInnen können so rasch einbestellt und gezielt versorgt werden. Dadurch kommt es zu einer Entlastung an Rettungstransporten. Auch administrative Prozesse sowohl für Schreibkräfte als auch für Pflege und Ärzte können so reduziert werden. Außerdem werden Patientenkontakte und das Infektionsrisiko minimiert.

Schlussfolgerungen: Die telemedizinische Versorgung von PatientInnen mit implantierbaren Devices ermöglicht auch in Krisensituationen bzw. Pandemien wie COVID-19 eine optimale medizinische Versorgung. Die Aufstockung der telemedizinischen Versorgung führt zu erhöhter Patientensicherheit, sowohl durch Reduktion des Infektionsrisikos aufgrund kontaktloser Kommunikation, als auch durch uneingeschränkte Überwachung der Device-Daten. Außerdem können Transportkosten sowie auch administrative Prozesse für das Krankenhaus und das Personal eingespart werden.

BA-7

Haemophagocytic lymphohistiocytosis and cardiac injury in COVID-19: A stepwise immunomodualtory treatment-approach

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Introduction: Patients treated for coronavirus disease 2019 (COVID-19) are often in need of critical or intensive care. Apart from acute respiratory distress syndrome (ARDS), multi-organ failure including acute kidney injury (AKI), cardiovascular injury and involvement of the central nervous system have been repeatedly reported in terms of a sepsis-like syndrome. Moreover, COVID-19 leads to a typical pattern of laboratory results, creating a yet not fully understood syndrome. In theory, hyperinflammation, cytokine storm, and possibly secondary haemophagocytic lymphohistiocytosis (sHLH) have been discussed as aggravating factors in critically ill patients. HLH shows similar mortality rates in non-COVID-19 patients (around 40 %) as those registered of COVID-19 ICU-patients (up to 65 %), and viral infections have long been known to act as sHLH triggers. As there is urgent need for effective treatment options, immunosuppression has been suggested, and first reports are promising. However, more data on both its clinical use and real-world reports of sHLH triggered by COVID-19 are needed.

Methods: We reviewed patients with laboratory-confirmed COVID-19 infection who were admitted to an ICU in the metropolitan area of Vienna, Austria between April and May, 2020, and who were diagnosed with both sHLH and concomitant cardiac injury. Patients' clinical-, imaging-, and laboratory data during their ICU stay were assessed. SHLH was diagnosed using the HScore. Nine variables are assessed, and points are summated: core temperature, hepato- and/or splenomegaly, number of cytopenias, levels of TG, fibrinogen, ferritin and ASAT, history of immunosuppression and (if feasible) presence of bone marrow haemophagocytosis. A positive result yields a 93 % sensitivity and 86 % specificity for HLH. The score can easily be calculated online. Cardiac injury was defined as heart failure, cardiogenic shock, and/or dysrhythmia. The mmunosuppressive treatment regimen was conducted in a stepwise approach: 1) 1 g of methylprednisolone intravenously once daily for three days, 2) 1 g/kg of Pentaglobin (50 mg/ml human plasma protein containing ≥95 % of immunoglobulin [6 mg IgM, 6 mg IgA, 38 mg IgG], Biotest Corp., Dreieich, Germany) via continuous infusion over 48 h, 3) 200 mg of anakinra subcutaneously twice daily until clinical improvement in patients' condition. Anakinra was used as an off-label salvage treatment.

Results: Three patients tested positive for COVID-19, sHLH and concomitant cardiac injury were included. A 51-year old man, a 75-year old man, and a 74-year old woman were hospitalized and intubated due to fulminant ARDS due to COVID-19. Acute kidney injury necessitated continuous renal replacement therapy, upgraded with an immunoadsorption filter to treat cytokine storm. Cardiac injury developed slowly, and despite mild catecholamine support with noradrenaline in the beginning, the hemodynamic profile deteriorated. Dobutamine was

added due to heart failure with reduced ejection fraction and impaired left ventricular function. While levosimendan and argipressin led to a transient clinical improvement, the patients' cardiac injury and hemodynamics further worsened. Non-compensatory tachycardia and episodes of atrial fibrillation were successfully treated with continuous landiolol. After sHLH was diagnosed and immunosuppressive therapy was started (see Methods), catecholamine support could be reduced, probably due to a dampened cytokine storm and lesser concomitant cardiac injury. Unfortunately, two patients deceased due to multi-organ failure because sHLH diagnosis and the treatment regimen came too late to sustainably change the clinical course. However, the female patient–after having received the full immunomodulatory treatment–recovered from respiratory failure and cardiac injury, and showed a restitutio ad integrum.

Conclusion: On the basis of our experience with three patients showing both HLH secondary to COVID-19 and substantial cardiac injury, we suggest a routine screening of COVID-19 patients for sHLH by using the HScore. Especially patients clinically deteriorating and showing heart failure, cardiogenic shock and dysrhythmia with no sufficient response to standard treatment might be at particular high risk. A stepwise therapeutic approach comprising corticosteroids, immunoglobulins and anakinra, accompanied by immunoadsorption, may dampen cytokine storm effects, and potentially reduce mortality.

POSTERSITZUNG 1 – BASIC SCIENCE 1

PS 1/1-1

Interleukin-4 signalling enhances survival of circulating monocytes in vivo

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Introduction: Monocytes are innate immune cells which differentiate into individual subsets through sequential transition. Most monocytes leave the circulation and only a small fraction of classical CD14++CD16-/Ly6Chigh monocytes remain in the circulation and develop into other subsets (CD14++CD16+, CD14+CD16+/Ly6Clow) [1]. The life-time of monocytes differs depending on the individual subset between 1 day and a week. The mechanisms leading to the transition of one monocyte subset into another and their regulation of lifespan are still unknown to most extend [2]. Without external stimulation by anti-apoptotic signals (i.e. colony stimulating factors), monocytes undergo apoptosis. Interleukin-4 (IL-4) signalling, which is commonly known to be involved in Th2 driven immune responses, has been shown to increase the survival of basophil granulocytes in vitro but nothing is known about its role in monocyte homeostasis [3].

Methods: We studied the relevance of IL-4 receptor signalling on murine monocyte survival in C57BL/6 mice lacking functional IL-4 receptors either in all cells (full body knockout) or only in myeloid cells (conditional knockout). Blood, spleen, liver as well as bone marrow of adult animals were gathered for flow cytometric determination of leukocyte counts. Protein levels of 40 cytokines were quantified by an array and confirmed by qPCR and ELISA. Further, to evaluate if IL-4 signalling does also affect human monocyte survival, peripheral mononuclear cells from human volunteers were isolated and monocytes were obtained via negative selection. They were then used for apoptosis assessment by flow cytometry.

Results: We observed a ~25 % reduction of circulating leukocytes in IL4 receptor knockout strains. Furthermore, the amount of circulating monocytes was reduced by approximately 50 %, compared to the respective control mice. The amount of monocytes and their precursors did not differ in the bone marrow of knockout mice. To identify differences in cell-to-cell signalling, we performed a plasma cytokine analysis. Plasma MCP-1 levels were significantly reduced in knockout mice which was also confirmed by ELISA but further in vitro studies on murine and human monocytes showed no induction of MCP-1 expression by IL-4. We found increased amounts of monocytes in the spleens of both knockout strains, which hinted us to investigate the effect of IL-4 on apoptosis of monocytes in vitro. Stimulation of human monocytes with increasing amounts of IL-4 increased the proportion of viable monocytes after 24 h when compared to unstimulated monocytes.

Conclusion: Our findings identify a previously unknown role of interleukin-4 in the regulation of circulating monocytes under homeostatic conditions. Knockout of functional IL-4 receptors on either all cells or only myeloid cells reduced circulating monocyte counts by 50 %. This was followed by increased amounts of splenic Ly6Clow monocytes without quantitative alterations in the bone marrow, hinting us to speculate that the survival of monocytes in IL-4 receptor deficient mice could be reduced. This assumption is supported by our findings on human monocytes. Stimulation with increasing amounts of IL-4 led to prolonged survival of the monocytes, comparable to the effect of colony-stimulating factors. We speculate that the reduction observed in MCP-1 is due to an overall reduction of monocytes as primary MCP-1 producing cells. Taken together, we propose a novel role of the Th2 cytokine interleukin-4 in the regulation of circulating monocyte amounts.

PS 1/1-2

New insights into ischemic mitral regurgitation: valvular and myocardial pathophysiology

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Introduction: Objective Ischemic mitral regurgitation (MR) is a frequent complication of myocardial infarction (MI) characterized by adverse remodeling both at the valvular and myocardial levels. Persistent activation of endothelial cells leads to leaflet fibrosis through endothelial-to-mesenchymal transition (EMT) in association with changes in cardiomyocyte function. Here we illustrate the molecular mechanisms of valvular remodeling and changes in cardiomyocyte force production in large animal models of short- and long-term ischemic MR.

Methods: Moderate ischemic MR was induced by creating posterior papillary muscle infarct (7 pigs and 7 sheep) and its characteristics were compared to those of control animals (7 pigs and 4 sheep). Pigs and sheep were sacrificed after 6 weeks

and 6 months, respectively. In-vitro experiments were conducted on isolated porcine mitral valve endothelial cells and their potential to go through EMT under Tenascin C stimulation was explored. Besides the isolated myocytes from different regions of the heart were characterized

Results: . TNC was present at 6 weeks and 6 months and correlated well with leaflet thickness (R = 0.68; $p < 0.001$ at 6 weeks, R = 0.84; $p < 0.001$ at 6 months). To confirm the translational potential of our findings, we obtained mitral valves from patients with ischemic cardiomyopathy presenting MR ($n = 5$). Indeed, TNC was also expressed in the mitral leaflets of these. Furthermore, TNC induced EMT in isolated porcine mitral valve endothelial cells (MVEC). Interestingly, toll-like receptor 4 (TLR4)-inhibition prevented TNC-mediated EMT in MVEC.

Conclusion: We identified here for the first time a new contributor to valvular remodeling in ischemic MR, namely TNC, which induced EMT through TLR4. Our findings might set the path for novel therapeutic targets for preventing or limiting ischemic MR.

PS 1/1-3

CD8+CD28null T lymphocytes are associated with the development of atrial fibrillation after elective cardiac surgery

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Introduction: Post-operative atrial fibrillation (POAF) is assumed as a complex and multifactorial interaction of different pathogenic factors. Data suggests an inflammatory process as a main trigger of this specific type of atrial fibrillation. CD8+ T lymphocytes that lack the surface protein CD28 were found to be crucially involved in chronic inflammatory processes within the cardiovascular system. Of utmost interest, these so called CD8+CD28null T cells are known to present with auto-aggressive behavior and deleterious cytotoxic effects on human tissue. Therefore, the impact of cellular immunity on the development of POAF was sought to assess.

Methods: To elucidate the impact of cellular immunity on the development of POAF, we prospectively enrolled 129 patients undergoing elective cardiac valve and/or coronary-artery-bypass-graft surgery. Fluorescein-activated cell sorting (FACS) was performed to investigate lymphocyte subsets. Patients were stratified in two subgroups according to patients developing POAF ($n = 60$) and individuals free of POAF ($n = 69$). Binary logistic regression analysis was performed to assess the impact of cellular immunity on the development of POAF.

Results: Comparing patients developing POAF to individuals free of POAF the fraction of CD8+ lymphocytes was significantly higher in individuals developing POAF (30.5 % [POAF] vs. 25.7 % [non-POAF]; $p = 0.021$) Interestingly, the fraction of CD8+CD28null T lymphocytes was significantly higher in the POAF sub-group (66.7 % [POAF] vs. 61.6 % [non-POAF];

$p = 0.043$). Binary logistic regression further proved that the fraction of CD8+CD28null T cells was a strong prognosticator for the development of POAF with a crude odds ratio per one standard deviation of 3.45 (95 %CI 1.11–10.70; $p = 0.032$). The prognostic potential remained stable after adjustment for potential confounders (age, male gender and type of surgery) within the multivariate model with an adjusted odds ratio per one standard deviation of 3.21 (95 %CI 1.01–10.18; $p = 0.048$).

Conclusion: We found that cytotoxic CD8+CD28null T lymphocytes proved to be a strong and independent predictor for the development of POAF after elective cardiac surgery. Our results potentially indicate an auto-immune impact of this pre-existing, highly cytotoxic T cell subset in the pathogenesis of POAF.

PS 1/1-4

Pharmacological inhibition of fatty acid oxidation reduces inflammation in macrophages in vitro and in vivo

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Introduction: Disease progression is often accompanied by changes in cellular metabolism. Macrophages have been described to switch towards a glucose dependent metabolism during proinflammatory polarization. In recent years, better understanding of macrophage metabolism and changes upon polarization have supported the notion of metabolic reprogramming of macrophages that leads also to functional changes. The possibility of shifting metabolism has sparked numerous novel pharmaceutical concepts. Trimetazidine, targeting long-chain 3-ketoacyl coenzyme A thiolase of fatty acid oxidation (FAO) is used regularly as an antianginal drug. The rationale behind therapeutically using trimetazidine is, that during and after ischemia, stimulation of glucose oxidation would be expected to decrease proton production, which is supported by the observation that trimetazidine reduces intracellular acidosis during ischemia. With atherosclerosis being the main reason for angina, most of the patients using trimetazidine suffer from this disease. However, no adverse side effects linked to inflammation were reported for patients taking trimetazidine. Given the great interest in the field for modulating macrophage metabolism and possible effects thereof, we analyzed the impact of trimetazidine mediated FAO inhibition in an atherosclerotic mouse model in vivo and the effects of FAO in macrophages and endothelial cells in vitro.

Methods: In vivo: For this study 26 LDL-R–/– mice on a C57/BL6 background were used. All animals were male and at the age of 12 weeks ±3 days at the start of the study. At the age of 12 weeks they were put on a high fat diet. At the age of 18 weeks they were randomly allocated to the trimetazidine or control group consisting of 13 animals respectively. The treatment group received trimetazidine 15 mg/kg/day via tap water, the control group received tap water only. The animals were sacrificed at the age of 26 weeks in order to analyze the extent of the aortic plaque. In vitro cell culture: HUVECs To analyze

potential effects of trimetazidine on the endothelium, HUVEC were pretreated with or without 500 μM trimetazidine for 30 min. Subsequently, the cells were incubated with recombinant human (rh)-IL-1ß 200 unites/mL in serum reduced medium in order to measure the expression of adhesion molecules as well as as inflammation associated interleukins. In vitro cell culture: Macrophages PBMC were isolated from whole blood and seeded into culture dishes in RPMI medium containing serum and supplements. Full medium supplemented with M-CSF was changed three times over a period of 7 days with the first change of medium after 4 h to allow monocyte adherence to the plastic dish. Cells were polarized in full medium without M-CSF. Furthermore, metabolic changes following inflammatory stimulation after pretreatment with trimetazidine μM 500 were analyzed.

Results: In vivo: 26 LDL–/– mice on a high fat diet were sacrificed at the age of 26 weeks. No significant difference in body weight and blood cholesterol levels between treatment and control group was observed. Our results indicated a beneficial response of trimetazidine treatment in regard to atherosclerotic plaque burden. We observed a significant reduction of 34.1 % comparing plaque-covered areas of the aorta. Trimetazidine treatment led to a reduction of cholesterol clefts in the aortic root and an increase in fibrous cap size. Structural integrity of the atherosclerotic plaque was further enhanced by trimetazidine treatment indicated by a reduction of elastin fragmentation, a reduction of IL-1ß, a reduction of NET-formation and a decrease in cleaved caspase 1 within the plaque. In vitro cell culture: HUVECs Overall, there was no observable effect of trimetazidine treatment on IL-1β induced adhesion molecule expression. When analyzing inflammatory mediators in endothelial cells after IL-1β stimulation we found no regulation of IL-8 protein levels and slight albeit significant reduction of MCP-1 expression levels. In vitro cell culture: Macrophages Treatment with trimetazidine during macrophage activation and NLRP3 induction resulted in a reduced IL-1β secretion. This reduction was accompanied by a decrease of cleaved caspase 1 activity in the cells. This reduction was not dependent on direct activation of the NLRP3 cascade, as same results were found in long-term polarization.

Conclusion: We provide evidence that pharmacological inhibition of FAO in a proinflammatory setting overall reduces inflammation and modulates cellular responses to inflammatory triggers. In an atherosclerotic mouse model inhibiting FAO using the pharmacological compound trimetazidine resulted in reduced plaque formation, reduced plaque inflammation, and increased plaque stability. Furthermore, we suggest that endothelial cells are most likely not affected by a limited FAO. Our results are in line with previously published data that mainly quiescent endothelial cells depend on FAO, whereas activated endothelial cells rely on glucose metabolism to fulfill their energy needs. we propose that the additional pharmacologic inhibition of FAO does not change the initial metabolic signature of the cell and therefore does not influence the already activated pathways. We demonstrated that reduction of FOA in proinflammatory macrophages reduces the activation of the NLRP3 system and protects mitochondria from damage. Our data is in line with a previous report suggesting a cross talk between NOX-4 and FAO in NLRP3 activation. In conclusion, we suggest that inhibiting FAO and hence forcing cells towards a glucose dependent metabolism does not promote inflammation. Moreover, our in vitro and in vivo data demonstrate, that pharmacological inhibition of FAO has beneficial effects in reducing inflammation via attenuated activation of the NLRP3 system.

PS 1/1-5

Differentiated HL-60 cells are a suitable model to study neutrophil extracellular trap formation

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Introduction: Neutrophil granulocytes have a very short lifespan and cannot be cultured for an extended period of time. Furthermore, cell isolation from whole blood is effortful while recovery is limited, and requires time and procedures that could potentially pre-activate cells such as repeated centrifugation and red blood cell lysis. The immortalized HL-60 cell line was derived from a female suffering from acute promyelocytic leukemia and can be differentiated into neutrophil-like cells in culture. We aimed to establish and validate differentiation of the HL-60 cell line to study neutrophil extracellular trap formation (NETs) in our laboratory.

Methods: HL-60 cells were cultured in RPMI-1640 supplemented with 10 % of fetal bovine serum, 100 U/ml penicillin, 100 U/ml streptomycin, 2.5 ng/ml amphotericin B, and 2 mM L-glutamine in a cell culture incubator at 37 °C and 5 % CO_2. Dimethylsulfoxide (DMSO, 1.3 %) and all-trans retinoic acid (ATRA, 5 μM) were used for differentiation for 1 to 5 days. Differentiation experiments were repeated four times. Cells were monitored each day during differentiation for viability, and CD11b, CD66b, CD14, CD16, and Ki67 expression by flow cytometry. Similarly, cells were harvested on days 1–5 and lysed in Trizol reagent for RNA isolation, subsequent cDNA synthesis, and quantitative polymerase chain reaction (qPCR) of peptidyl-larginine deiminase 4 (PAD4), CD11b, low-density lipoprotein receptor (LDLR), deoxyribonuclease 1 (DNase 1), and sensors of inflammation. NET formation was assessed by measuring release of double-stranded DNA (dsDNA) with a fluorescent DNA-binding dye in response to stimulation with the calcium ionophore ionomycin. Neutrophils isolated from 10 healthy volunteers served as controls.

Results: Differentiation of HL-60 cells with DMSO led to a stable, daily increase of the adhesion marker CD11b and a significant decrease of the proliferation marker Ki67 in flow cytometry, which could not be steadily observed using ATRA. Quantitative PCR confirmed differentiation into neutrophil-like cells by daily upregulation of PAD4 mRNA, an enzyme required for NET formation, and CD11b mRNA expression. Messenger RNA expression levels of healthy volunteers were in the same range as those from HL-60 cells differentiated for 3–5 days. Cell viability was at least 90 % until day 3 and decreased thereafter. Stimulated NET formation increased until day 3.

Conclusion: Despite known limitations of immortalized cell lines, HL-60 cells differentiated into neutrophil-like cells using DMSO have similar expression of important marker molecules as compared to isolated neutrophils and are suitable to study NET formation.

PS 1/1-6

Higher level of cell-free-circulating-DNA is a risk marker for atherosclerosis in systemic lupus eryhrematosus

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Introduction: Autoimmune disease including systemic lupus erythematosus (SLE) have a higher risk of atherosclerosis and thus cardiovascular diseases (CVD), but the underlying mechanism is still poorly understood. Higher level of cell free DNA is suggested to be a risk marker for atherosclerosis progression. Here, we aim to study the cell free circulating DNA in SLE in connection with atherosclerosis.

Methods: DNA was extracted from age and sex matched control ($n = 91$) and SLE case ($n = 109$) groups, and the level of DNA was measured and compared with such groups. B mode Ultrasound common was used determine the occurrence of plaques. Peripheral blood mononuclear cells and endothelial cells were stimulated with the extracted DNA to determine mechanistic pathway.

Results: The prevalence of the circulating DNA level above 66th percentile was significantly higher in the SLE than the control group. The SLE patients with such level had higher risk of atherosclerotic plaques. To investigate the possible mechanism, we identified that DNA induced atherosclerosis initiator factor ICAM in endothelial cells. Furthermore, the DNA induced IL-1bet, IL-6 and NFkB considered to be risk factor in atherosclerosis.

Conclusion: Higher level of cell free circulating DNA is a risk marker for atherosclerosis and thus CVD. The DNA might induce atherosclerosis as well as pro-inflammatory effect.

PS 1/1-7

Elevated baseline levels of asymmetric dimethylarginine and the risk for arrhythmic death comparing patients with ischemic or dilated cardiomyopathy–a prospective, controlled long-term study

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Introduction: Elevated baseline levels of asymmetric dimethylarginine (ADMA), an endothelial nitric oxide inhibitor, are associated with adverse outcome. There is no data available, whether ADMA levels are associated with arrhythmic death (AD) in patients with ischemic cardiomyopathy (ICM) or non-ischemic, dilated cardiomyopathy (DCM).

Methods: Baseline ADMA was measured in 110 ICM, 52 DCM and 30 control patients. Primary outcome parameter of
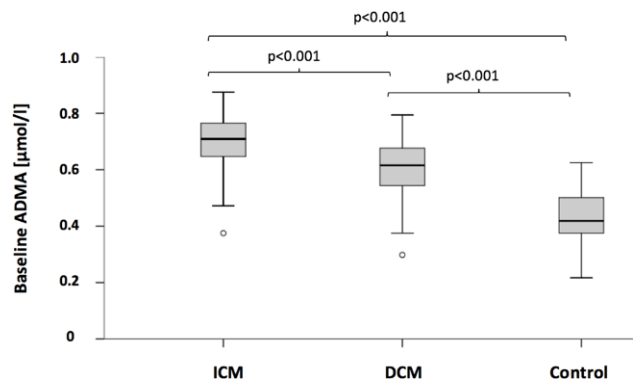
Fig. 1 | PS 1/1-7 Boxplots showing baseline ADMA levels in patients with ICM, DCM and control patients
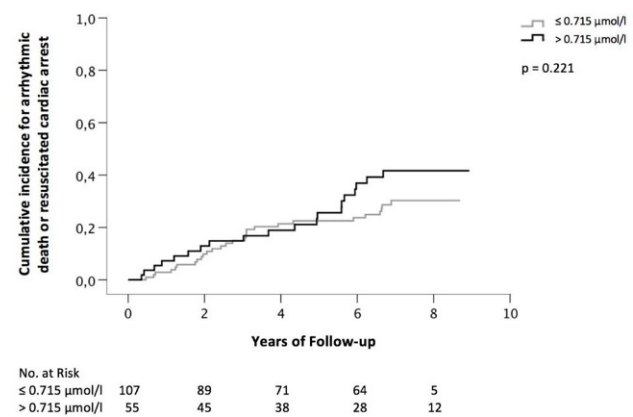
Fig. 2 | PS 1/1-7 Kaplan Meier curves demonstrating the association between AD or RCA and baseline levels of ADMA stratified to the upper tertile (>0.715 µmol/l) and the combined two lower tertiles (≤0.715 µmol/l)

this prospective study was arrhythmic death (AD) or resuscitated cardiac arrest (RCA).

Results: Baseline levels of ADMA were significantly higher in ICM ($p < 0.001$) and in DCM ($p < 0.001$) patients compared to controls (Fig. 1). During a median follow-up of 7.0 years, 62 (32.3 %) patients died. AD occurred in 26 patients and RCA was observed in 22 patients. Baseline levels of ADMA were not associated with a significantly increased risk of AD or RCA in ICM (hazard ratio (HR) = 1.37, $p = 0.109$) or in DCM (HR = 1.06, $p = 0.848$) patients. No significant association was found with overall mortality in ICM (HR = 1.39, $p = 0.079$) or DCM (HR = 1.10, $p = 0.666$) patients. Stratified Kaplan-Meier curves for ADMA levels in the upper tertile (>0.715 µmol/l) or the two lower tertiles (≤0.715 µmol/l) did not show a higher risk for AD or RCA ($p = 0.221$, Fig. 2) or overall mortality ($p = 0.548$). In patients with left ventricular ejection fraction ≤35 %, ADMA was not associated with AD or RCA (HR = 1.35, $p = 0.084$) or with overall mortality (HR = 1.24, $p = 0.162$).

Conclusion: Baseline levels of ADMA were elevated in patients with ICM or DCM. Baseline ADMA was not associated with a higher risk for arrhythmic death on the long term.

PS 1/1-8

Remote ischemic perconditioning improves vascular stiffness and endothelial dysfunction following myocardial ischemia and reperfusion in rats

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Introduction: Vascular stiffness and -endothelial dysfunction are independent factors for future cardiovascular events. Both are accelerated following acute myocardial infarction and subsequently may increase the risk for additional atherothrombotic events. Aim: to explore the vasculoprotective efficacy of remote ischemic perconditioning (RIPerc) on coronary- and aorta endothelial function as well as on vascular stiffnesss.

Methods: Male OFA-1 rats were subjected to 30 min of occlusion of the left anterior descending artery (LAD) followed by either 3 days or 4 weeks reperfusion and separated into three groups: (1) sham operated (Sham, without LAD occlusion; $n=6$); (2) myocardial ischemic reperfusion (MIR) ($n=8$ for 4 weeks reperfusion and $n=6$ for 3 days of reperfusion) and (3) MIR+RIPerc ($n=6$ for 4 weeks reperfusion and $n=5$ for 3 days reperfusion) group with three cycles of 5 min of I/R on hindlimb performed during myocardial ischemia. Assessment of vascular reactivity in isolated septal coronary artery and aortic rings as well as the vascular stiffness were performed by Wire Myograph System. In addition, the assessment of plasma Malondialdehyde (MDA) levels, as a marker for oxidative stress was performed by HPLC.

Results: MIR induced a significant impaired endothelial-dependent relaxation in both septal coronary artery and aorta in comparison to Sham group in the long-term and short-term experiment ($P < 0.05$). In addition, RIPerc also showed a tendency to preserve endothelial function in both vascular beds, without affecting MDA levels. Moreover, MIR induced vascular stiffness in aorta, which was reversed by RIPerc.

Conclusion: Myocardial infarction induces endothelial dysfunction in non-infarcted vessel and vascular stiffness in peripheral vasculature. These changes were partially and significantly improved by RIPerc, suggesting the vasculoprotective effects of RIPerc and subsequently reduction in future atherothrombotic events.

POSTERSITZUNG 2 – RHYTHMOLOGIE 1

PS 2/2-1

Impact of contact force sensing catheters on fluoroscopy time in left-sided atrial procedures

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Introduction: Contact force sensing catheters provide electrophysiologists with direct feedback and therefore improve safety and help to generate more effective lesions. The use of contact force may also reduce fluoroscopy and procedure times. The aim of this study was to systematically evaluate the impact of using contact force sensing catheters (CFSCs) on fluoroscopy times and procedure times in left-sided atrial procedures.

Methods: In this international multicenter study, data from 622 left-sided procedures (142 without and 480 with CFSCs) of 25 participating European centers were prospectively collected with a structured questionnaire. Examinations comprised 393 pulmonary vein isolations (PVIs, group 1[G1]), 122 PVIs with linear lesions (group 2[G2]), 65 left-sided accessory pathway ablations (group 3 [G3]) and 42 ablations for left atrial tachycardia (group 4 [G4]). Fluoroscopy and procedure times were compared with respect to the use of CFSCs, procedure type, and level of operator experience as possible confounders.

Results: With the use of CFSCs, fluoroscopy time was significantly reduced when performing pulmonary vein isolations (G1 median [IQR]: 19 [11.0–33.7] vs. 7.2 [4.0–13.0] min., $p < 0.001$, G2: 45.3 [34.9–61.8] vs. 7.3 [5.0–14.0], $p < 0.001$, respectively). For groups G3 and G4, no difference could be detected. Procedure times were shortened for pulmonary vein isolations with additional lesions only (G4: 210 [180–240] vs. 153 [127–200] min., $p < 0.001$). When assessing the effect of operator experience, all left-sided atrial procedures were combined. Significantly shorter fluoroscopy times with contact force were found at all career levels (early career <5 years: median –6 min, $p = 0.024$, mid-career 5–15 years: –15 min, $p < 0.001$, mentor status with experience >15 years: –6.5 min, $p < 0.001$). The use

of contact force proved to be especially beneficial in reducing fluoroscopy time in operators performing one to 19 procedures per month (1–9: median –15.8 min, $p < 0.001$, 10–19: –15.9 min, $p < 0.001$), whereas it lost its statistical significance when more than 20 procedures per month were performed (20–39: –3.2 min, $p = 0.100$, >40: –1.5 min, $p = 0.346$).

Conclusion: Contact force sensing catheters can help to reduce fluoroscopy times, especially when performing pulmonary vein isolations. This effect could be demonstrated for all career levels. A shortening of procedure time was also found for PVIs with additional linear lesions.

PS 2/2-2

Impact of pacing mode and different echo parameters on cardiac output (PADIAC)

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Introduction: Leadless pacemaker systems have fundamentally changed the field of device therapy. Since this technology is currently limited to single-chamber pacing, efforts are being made in the development of dual-chamber leadless pacemaker systems to enable AV-synchronous pacing. The extent to which AV-synchronous pacing increases cardiac output may vary depending on different parameters such as systolic, diastolic, or atrial function. This study sought to evaluate the association of an array of different echo parameters with changes in stroke volume between AV-synchronous and AV-asynchronous stimulation.

Methods: We recruited patients with a conventional permanent dual-chamber pacemaker who were in sinus rhythm with a complete AV-block and >90 % ventricular pacing rate upon device interrogation. After the recording of baseline characteristics with a structured questionnaire, the pacemaker was programmed to DDD 40/min. to allow for intrinsic activation of the atria. Then we performed an echo-guided AV delay optimization at rest. Echo measurements of all included patients were collected by a single investigator. We assessed parameters of left ventricular diastolic (averaged E/E' ratio, TR velocity, left atrial volume index [LAVI]) and systolic function (ejection fraction [EF], global longitudinal peak systolic strain [GLPSS]), as well as parameters of left (left atrial strain in the contraction phase) and right atrial (right atrial contraction excursion) contractility and the tricuspid annular plane systolic excursion (TAPSE) with a commercially available ultrasound system (Epiq7®, Philips Health Systems, The Netherlands). Finally, the left ventricular stroke volume–as a proxy of cardiac output–was measured three times and averaged after at least one minute of hemodynamic adaptation, both during DDD and VVI stimulation at the intrinsic heart rate. The sequence of programming the pacemaker mode was thereby randomized (VVI/DDD vs. DDD/VVI). A linear regression model was calculated to assess the association between change in stroke volume and different echo parameters.

Results: We enrolled 40 patients with a median age of 76.0 years (IQR: 70.5–81.0), and a median left ventricular ejec-

tion fraction of 60.8 % (IQR: 50.0–63.7). Seventy-eight percent of them were male, 38 % were diagnosed with coronary heart disease, and 30 % with diabetes mellitus. Every single subject showed an increase in stroke volume with AV-synchronous stimulation ranging between 0.1 ml and 37.7 ml (median: 12.8 ml, IQR: 5.5–16.0 ml, $p < 0.001$). In the regression analysis, all the echo parameters under investigation were not associated with the extent of the stroke volume increase in our cohort (EF $p = 0.082$, GLPSS $p = 0.681$, the grade of diastolic dysfunction $p = 0.289$, LAVI $p = 0.576$, left atrial strain in the contraction phase $p = 0.569$, right atrial contraction excursion $p = 0.921$, TAPSE $p = 0.537$). This was also confirmed in a multivariate analysis, which–besides the echo parameters–included clinical factors such as age, gender, BMI, glomerular filtration rate, and diabetes status.

Conclusion: In our cohort of patients in sinus rhythm with complete AV-block, all subjects profited from AV-synchronous pacing as opposed to VVI pacing at the intrinsic heart rate. This effect could be shown independently of different degrees of left ventricular systolic and diastolic dysfunction, different left atrial sizes and different degrees of left and right atrial contractility.

PS 2/2-3

The Wearable Cardioverter/Defibrillator: retrospective analysis of efficacy, safety and adherence

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Introduction: The use of the Wearable Cardioverter Defibrillator (WCD) is recommended in national, European and American guidelines. However, there are almost exclusively data from the manufacturer's own data network. Independent data on the experience with the WCD are rare. The aim of the retrospective study from one cardiologic department was to record efficiency, safety and compliance of the WCD.

Methods: The study included all patients, to whom a WCD was described between 01.11.2010 and 01.05.2018 at one cardiologic department. Clinical data were obtained from the patients' records and the data about the WCD from the information network of the manufacturer.

Results: This study enrolled 66 patients, 51 males (77 %) and 15 females (23 %). The median age was 55 years (IQR: 45–63). They suffered from ischemic cardiomyopathy ($n = 33$; 49 %), dilated cardiomyopathy ($n = 12$; 18 %), myocarditis ($n = 7$; 11 %), explantation of an implantable cardioverter/defibrillator (ICD; $n = 5$; 8 %) and other indications ($n = 6$; 9 %). The median wearing time of the WCD was 73 days (interquartile range-IQR: 39–126), with median daily use of 22.91 h (IQR: 19.58–23.61). Among 38 patients with LVEF ≤35 %, LVEF improved to ≥35 % in 19 patients (50 %) during WCD therapy. Over 1600 times the WCD detected a VT falsely. Four patients (8 %) suffered from 212 non-sustained VT. One patient was successfully shocked because of ventricular fibrillation (appropriate shock rate: 1.5 %). There were no inappropriate shocks. All patients, who wore the WCD, survived and one patient died when he did not wear the WCD. At the end of therapy 32 patients (48 %) received an ICD. In terms of wearing time and events (shocks, arrhythmias, artifacts) there were no significant differences between patients receiving ICD and those who did not receive an ICD. Patients who received an ICD had a significantly lower LVEF after 3 months than patients who did not receive an ICD.

Conclusion: Our data confirm, that the WCD is safe and that the patients, who wear a WCD, have a high adherence. More than half of the patients with reduced LVEF improved their systolic function during WCD therapy, thus obviating the need for ICD implantation. Questions about the effectiveness of the detection algorithm remained open.

PS 2/2-4

Do patients after acute myocardial infarction benefit from a wearable cardioverter defibrillator? Results from the Austrian WCD registry

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Introduction: In the first three months after myocardial infarction (MI), patients are at elevated risk of sudden cardiac death (SCD) due to ventricular tachycardia (VT) or ventricular fibrillation (VF). According to the ESC Guidelines, the implantation of an ICD should be postponed until 40 days after acute MI. The wearable cardioverter defibrillator (WCD) is a well-established therapy for patients at temporary high risk of SCD. However, the VEST trial failed to show a significant reduction in arrhythmic mortality (1.6 % with WCD vs. 2.4 % with OMT, p = n. s.) with WCD therapy in acute MI patients, with only 1.9 % of patients receiving WCD therapy. The most important cause may have been the lower than expected WCD wearing compliance (median 18 h/day), whereas the general WCD compliance is high in the Austrian WCD registry (median 23.5 h/day). We aimed to investigate incidence of WCD treatments and outcomes of all patients with acute MI and LVEF ≤35 % prescribed with a WCD 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 2019. The VEST trial included patients with a LVEF ≤35 % assessed after ≥8 h after MI±PCI or ≥48 h after CABG for acute MI. Exclusion criteria were an pre-existing ICD, a significant valve disease, an unipolar pacing system, chronic haemodialysis, the chest being too small/large for WCD, pregnancy or a patient being discharged to a skilled nursing facility for >7 days.

Results: Of 896 patients registered in the Austrian WCD registry, the VEST in- and exclusion criteria applied to 105 patients (12 %). The average age was 64 ±11 years (12 % female; LVEF 28 ±6 %). All patients were revascularized: 104 patients underwent acute PCI, one patient received a CABG. The median WCD prescription duration was 69 (range: 1;277) days, the median wearing duration was 23.5 (0;24) hours/day. 80 % (83/105) had an average wearing duration ≥22 h, 15 patients (14 %) had a wearing compliance between 22 and 18 h/day, only 7 patients (6.7 %) had a wearing duration below 18 h/day. Within the course of WCD prescription 4/105 (3.8 %) patients received 9 appropriate shocks, the per patient shock rate was 2 (1;5). These shocks were caused by 7 ventricular tachycardia events and 2 times by ventricular fibrillation. No inappropriate shock was delivered. The LVEF at the end of follow up was 35 % [15;59] (p <0.01). During follow-up, 46/105 patients (44 %) received an ICD after the WCD period (2 [2 %] for primary prevention, 44 [42 %] for secondary prevention), whereas 38 patients (36 %) did not need ICD implantation due to improvement of LVEF. 4/105 (3.8 %) patients died during follow-up: Two patients due to a non-cardiac event and did not wear the WCD at the time of death, one patient died due to asystole while wearing the WCD and one patient died due to refractory VF at the ICU not wearing the WCD.

Conclusion: The WCD therapy was aborted in 25 events (haemodynamically stable VTs) in 5/105 (5 %) patients, seven WCDs (6.7 %) were stopped due to the patients' desire. Two WCD prescriptions (2 %) were discontinued due to a terminal non-cardiac disease, nine patients stopped WCD without documented reason or follow-up. The arrhythmic mortality of the Austrian cohort was 1 % (1/105) compared to 1.6 % (25/1524) in the VEST trial and the total mortality was 3.8 % (4/105) compared to 3.1 % (48/1524). 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 both total and arrhythmic mortality rates are not significantly improved.

PS 2/2-5

Role of wearable rhythm recordings in clinical decision making–The wEHRAbles project

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Introduction: Multiple wearable devices for rhythm analysis have been developed using either photoplethysmography (PPG) or handheld ECG. The aim of this survey was to assess impact of these technologies on physicians' clinical decision-making regarding initiation of diagnostic steps, drug therapy and invasive strategies.

Methods: The online survey included 10 questions on types of devices, advantages and disadvantages of wearable devices as well as case scenarios for patients with supraventricular arrhythmias and atrial fibrillation (AF).

Results: A total of 417 physicians (median age 37 [IQR 32–43] years) from 42 countries world-wide completed the survey. When presented a tracing of a regular tachycardia by a symptomatic patient, most participants would trigger further diagnostic steps (90 % for single-lead ECG vs. 83 % for PPG, $p < 0.001$), while a single-lead ECG would be sufficient to perform an invasive EP study in approximately half of participants (51 % vs. 22 % for PPG, $p < 0.001$). When presented with a single-lead ECG tracing suggesting AF, most participants (90 %) would trigger further diagnostic steps. A symptomatic AF patient would trigger anticoagulation treatment to a higher extent as an asymptomatic patient (59 % vs. 21 %, $p < 0.001$). PPG tracings would only rarely lead to therapeutic steps regardless of symptoms. Most participants would like scientific society recommendations on the use of wearable devices (62 %).

Conclusion: Tracings from wearable devices, in particular single-lead ECGs, suggestive of arrhythmias are likely to trigger further diagnostic steps, but in the case of PPG recordings rarely therapeutic interventions. However, the lack of clear recommendations is an important clinical limitation for utilisation of this technology.

PS 2/2-6

Das Elektrophysiologie-Labor der kardiologischen Abteilung am Universitätsklinikum Krems. Eine retrospektive Kohortenanalyse des Jahres 2019

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Diagnose	Ablation n/N/%	primär erfolgreich n/N/%	Rezidiv < 1 a n/n/%
AVNRT N=63	63 (100%)	63 (100%)	0 (0%)
Vorhofflimmern N=60	60 (100%)	60 (100%)	10 (16,7%)
Vorhofflattern N=58	58 (100%)	57 (98,3%)	5 (8,8%)
atriale Tc N=20	7 (35,0%)	6 (85,7%)	0 (0%)
WPW N=18	16 (88,9%)	14 (87,5%)	0 (0%)
RVOT N=4	4 (100%)	3 (75%)	2 (66,6%)
LVOT N=2	2 (100%)	2 (100%)	1 (50%)

Abb. 1 | PS 2/2-6 Jahresoutcome der Ablativen aus 2018

Einleitung: Das elektrophysiologische (EP) Labor ist Teil der kardiologischen Abteilung des 600 Betten Universitätsklinikum Krems. Es ist damit eines von 3 EP-Labs in Universitätsklinikum Krems. Seit 1993 werden an 2 Tagen pro Woche ca. 240 elektrophysiologische Untersuchungen und Ablationen pro Jahr durchgeführt. Das EP Team, unter der Leitung von Universitätsklinikum Krems, besteht aus 3 Senior- und einem Junior-Elektrophysiologen. Am Standort Universitätsklinikum Krems werden ausschließlich endokardiale Untersuchungen angeboten. Die vorliegende Kohortenanalyse präsentiert demographische Daten und Leistungszahlen der Elektrophysiologie am Universitätsklinikum Krems aus dem Jahr 2019 und 1-Jahres Outcomezahlen aus dem Jahr 2018.

Methoden: In dieser retrospektiven Kohortenbeobachtungsstudie werden einerseits demographische Daten der Pa-

Diagnose	n/N	(%)
AVNRT	63	(26%)
Vorhofflimmern	60	(25%)
Vorhofflattern	58	(24%)
atriale Tc	20	(8%)
WPWm	13	(5%)
diagnostische EPU	11	(5%)
WPWc	5	(2%)
RVOT	4	(2%)
Organische VT	3	(2%)
LVOT	2	(0.5%)
AV- Knoten.	2	(1%)

Abb. 2 | PS 2/2-6 Anzahl der behandelten Rhythmusstörungen im Vergleich
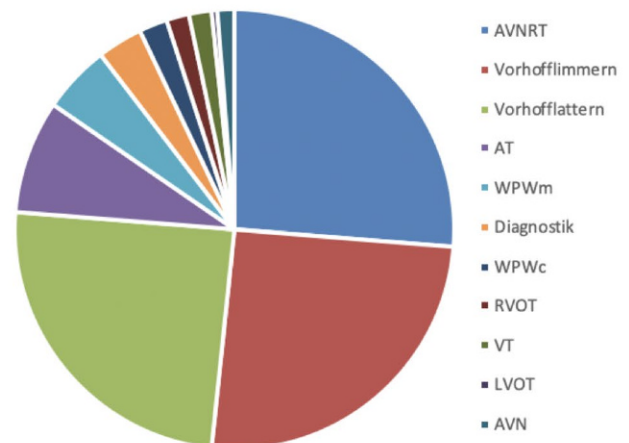
tienten, Anzahl und Prozentsatz der Untersuchungen, Erfolgs- und Rezidivraten aber auch Komplikationsraten beleuchtet. Darüber hinaus werden die 1-Jahres Outcome Daten des Jahres 2018 im Sinne von Rezidiven nach der stattgehabten Ablation präsentiert. Die Outcomedaten wurden über Nachkontrollen in der Rhythmusambulanz erhoben. Die präsentierten Daten werden in absoluten Zahlen, Mittelwert und Standardabweichung (SD) sowie median und interquartilen Range (IQR) nach Anwendbarkeit angeführt.

Resultate: Im Jahr 2019 wurden 241 EP-Untersuchungen an 218 Patienten durchgeführt. Das mittlere Alter der Patienten betrug 57 ± 14 Jahre. 89 (41 %) der Patienten waren Frauen. In 216 Fällen (90 %) wurde eine high-frequency-(HF)-Ablation der zugrundeliegenden Rhythmusstörung unternommen. Der überwiegende Anteil der EP-Untersuchgen betraf rechtsatriale Untersuchungen. Linksatriale Untersuchungen betrafen zumeist Pulmonalvenenisolationen. Nur ein kleiner Anteil der Untersuchungen betraf ventrikuläre Tachykardien. Abb. 1 zeigt eine Aufstellung der untersuchten/behandelten Rhythmusstörungen. 209 (96,7 %) der HF Ablationen wurden als primär erfolgreich eingestuft. Die Untersuchungszeit betrug im Median (IQR) 79 (54–173) min. Die Durchleuchtungszeit im Mittel 14 ± 12 min. Ein Jahresvergleich der Jahre 2015–2019 wird in Abb. 2 präsentiert. Die Anzahl der zugrungeliegenden Rhythmusstörungen und der elektrophysiologischen Untersuchungen bleibt über die Jahre konstant. Im Jahr 2019 traten in 12 Fällen (5,0 %) Komplikationen auf. Diese betrafen in erster Linie Komplikationen an der Einstichstelle. Das 1-Year Outcome des Jahres 2018 ist in Abb. 3 dargestellt.

Schlussfolgerungen: Im EP Labor des Universitätsklinikum Krems werden im Jahr ca. 240 links- und rechtsatriale Prozeduren angeboten. Weitere Beobachtungsstudien sind notwendig, um den Verlauf von Untersuchungszahlen und deren Outcome besser beurteilen zu können.

PS 2/2-7

Elektrophysiologische Interventionen unter oraler Antikoagulantientherapie

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Einleitung: Vor einem elektiven elektrophysiologischen Eingriff muss eine orale Antikoagulation (oAK) zeitgerecht abgesetzt werden, um peri- und postinterventionell Blutungen zu vermeiden. Ziel dieser Studie war es, zu überprüfen ob Patienten die NOAC-Therapie vor einer elektiven elektrophysiologischen Intervention gemäß den EHRA-Empfehlungen zum richtigen Zeitpunkt absetzen. Darüber hinaus wurde eine Patientengruppe unter NOAC Therapie mit einer Gruppe unter Vitamin-K-Antagonisten (VKA) in Hinblick auf postoperative Komplikationen verglichen.

Methoden: In den Jahren 2017 bis 2018 wurden Patientendaten von insgesamt 248 Patienten, die sich an der Universitätsklinik Innsbruck eines elektrophysiologischen Eingriffes unterzogen, und zur Zeit des Eingriffs eine Therapie mit einem NOAC oder VKA erhielten, gesammelt und ausgewertet.

Resultate: Bei Eingriffen mittleren Blutungsrisikos wurde das NOAC im Mittel 50 h, bei Eingriffen hohen Blutungsrisi-

kos im Mittel 71 h und bei Eingriffen mit sowohl hohem Blutungs- als auch hohem Thromboembolie Risiko im Mittel 49 h vor der Intervention abgesetzt. 33 (13,3 %) Patienten erlitten eine Komplikation; davon waren 23 (9,3 %) Blutungen und 1 (0,4 %) Thromboembolie. Bei Eingriffen mittleren Blutungsrisikos trat bei 3 (3,6 %), bei Eingriffen hohen Blutungsrisikos bei 4 (10,3 %) und bei Eingriffen mit sowohl hohem Blutungs-, als auch hohem Thromboembolie Risiko bei 16 (12,7 %) Patienten eine Blutung auf. Eine Thromboembolie trat bei einem Eingriff hohen Blutungs- aber moderatem thromboembolischen Risikos auf (HASBLED Score: 3 CHA_2DS_2-VASc-Score: 7). Die Komplikationsrate war in der NOAC-Gruppe (12,6 %) im Vergleich zur VKA-Gruppe (18,8 %) nicht signifikant niedriger. Präinterventionelle NOAC-Spiegel waren bei Patienten, die eine Blutungskomplikation erlitten (228,5 ng/ml) signifikant höher als bei Patienten ohne Blutungskomplikation (55,0 ng/ml).

Schlussfolgerungen: Patienten in allen drei Risikokategorien setzten die oAK früher ab, als in den EHRA Empfehlungen vorgegeben. Insgesamt kam es zu deutlich mehr Blutungen als zu thromboembolischen Ereignissen, wobei das Blutungsrisiko vom Risiko des Eingriffs selbst, vom NOAC-Spiegel bei Aufnahme und von der Art des Gefäßverschlusses abhing. Die höhere Komplikationsrate bei Patienten unter einer VKA-Therapie im Vergleich zur NOAC-Therapie deckt sich mit Analysen aus prospektiven randomisierten Studien.

PS 2/2-8

Near-zero fluoroscopy implantation of implantable electronic cardiovascular devices using electroanatomic mapping

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Introduction: Ionised radiation exposure is a significant drawback in the implantation of implantable electronic cardiovascular devices (IECD). Ultralow-dose radiation (ULD) or near-zero fluoroscopy (NZF) using electroanatomic mapping (EAM) modalities has been proposed [1] as an effective and safe alternative to conventional implantation protocols using fluoroscopy. We report the first 2 cases of NZF implantation of IECD using EAM in our center.

Methods: Two patients (P1 and P2, both male, aged 81 and 61 years old respectively) were referred to our hospital for IECD

Fig. 1 | PS 2/2-8 P1 lead position

Fig. 2 | PS 2/2-8 P2 leads position

implantation. P1 suffered from permanent atrial fibrillation and symptomatic bradycardia (due to slow ventricular response) and was referred for a VVI-pacemaker implantation. P2 was a post-myocardial infarction and post-myocarditis patient with a history of ventricular fibrillation and thus with an indication for ICD-implantation for secondary prevention. A decision to implant a DDD-ICD was made.

Results: Both patients underwent IECD placement using NZF by mapping the right atrium and ventricle using the Abbott NavX 3D-mapping system. A pocket was fashioned for the pulse generator in the subcutaneous space of the left pectoral area. The cephalic vein cutdown approach was preferred, according to our standard implantation strategy, and a sheath was placed. Then, a non-deflectable quadripolar 5F catheter was used to build geometry and the shell of the coronary Sinus (CS), right Atrium (RA), right atrial appendage, right ventricle (RV), and the His area. After building an acceptable geometry the pacing lead was then introduced. The pace sense part of the lead was connected to the NavX system junction box by using a small alligator clip, so the lead could be visualised by the mapping system. Subsequently, in P1, the RV lead was screwed into place; satisfactory pacing thresholds and R waves were obtained. In P2 the atrial lead was first screwed in place, then taken as a reference to build the geometry of RV, and following the RV lead was screwed into place; satisfactory pacing thresholds and sensing were also here obtained for both leads. Minimal fluoroscopy (P1: 17 sec., 9.54 µGym^2, P2: 38 sec., 9.00 µGym^2) was used to confirm that there was enough slack and that the helix of the leads was extended completely.

Conclusion: Near-zero fluoroscopy using electroanatomic mapping is an effective and safe alternative to conventional implantation of implantable electronic cardiovascular devices.

POSTERSITZUNG 3 – AKUTES KORONARSYNDROM 1

PS 3/3-1

Prasugrel compared to ticagrelor in clinical practice of primary PCI

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Introduction: Prasugrel and ticagrelor have similar recommendations in the setting of primary PCI by current guidelines. Data comparing both in daily clinical practice of primary PCI for ST-elevation myocardial infarction is limited. Purpose: To compare the effect of prasugrel and ticagrelor on in-hospital outcomes after primary PCI.

Methods: We prospectively enrolled 5365 patients treated with prasugrel ($n = 2785$, 51.9 %) or ticagrelor ($n = 2580$; 48.1 %) in the setting of primary PCI from January 2011 to December 2018 in a nationwide registry. In-hospital outcomes were compared and multiple logistic regression analysis was performed.

Results: Prasugrel treated patients were younger, less often in cardiogenic shock, with lower rates of previous stroke and had shorter ischemic time. Both groups showed similar rates of previous MI, diabetes and current resuscitation. In the univariate analysis mortality was lower in patients with Prasugrel (2.5 % vs. 4.4 % $p < 0.01$). Similarly, MACE (3.3 % vs. 5.3 %, $p < 0.01$) and NACE (4.0 % vs. 5.7 % $p < 0.01$) were lower in Prasugrel treated patients, whereas major bleeding events did not differ (0.4 % vs. 0.6 % $p = 0.24$). After adjustment in multivariable analysis mortality (0.99 95 % CI 0.57 to 1.72), MACE (OR 0.99 95 % CI 0.65 to 1.52) as well as NACE (0.86 95 % CI 0.61 to 1.22) did not differ in patients treated with Prasugrel compared to Ticagrelor.

Conclusion: Patients treated with prasugrel showed improved outcomes compared to ticagrelor in a large cohort of primary PCI. However, after adjustment for confounders the advantage of prasugrel in primary PCI did not persist.

PS 3/3-2

GLP-1 is an independent predictor of long-term mortality in patients with myocardial infarction complicated by cardiogenic shock–A substudy of the IABP-SHOCK II trial

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Introduction: The incretin hormone Glucagon-like-peptide 1 (GLP-1) is a major stimulus for glucose dependent insulin secretion and holds cardioprotective efficacy. This has made the GLP-1 system a preferred target for diabetes therapy. Secretion of GLP-1 happens in response to nutritional but also inflammatory stimuli. Consequently, marked elevation of circulating GLP-1 levels were found in critically ill patients featuring marked association to markers of inflammation. Our study sought to investigate GLP-1 levels in patients with cardiogenic shock (CS) complicating myocardial infarction and a possible prognostic correlation to short- and long-term outcome.

Methods: We serially assessed circulating GLP-1 levels in a prospectively planned biomarker substudy in the IABP-SHOCK II trial. Blood samples were drawn during index PCI and at day 2. The blood was centrifuged immediately, and serum was frozen at –87 °C. GLP-1 was measured with a standard ELISA-kit. All-cause mortality at short- (30 days), intermediate- (1 year) and long-term (6 years) follow-up was used for outcome assessment.

Results: In this study we found circulating GLP-1 to be markedly elevated in patients with myocardial infarction complicated by CS ($n=172$) at time of index PCI. Patients with fatal short-term outcome ($n=70$) exhibited higher GLP-1 levels (86 [45–130] pM) at ICU admission in comparison to patients with 30-day survival (48 [33–78] pM; $p<0.001$) ($n=102$). In repeated measures ANOVA the course of GLP-1 levels between baseline and day 2 showed a significant interaction between survivors and non-survivors ($p=0.04$). By univariate Cox-regression analysis GLP-1 levels >median were predictive of short- (hazard ratio [HR] 2.43; 95 % confidence interval [CI] 1.50–3.94; $p<0.001$), intermediate- (HR 2.46; 95 %CI 1.62–3.76; $p<0.001$) and long-term (HR 2.12; 95 %CI 1.44–3.11; $p<0.001$) outcome. This association remained after multivariable correction (HR 2.01; 95 %CI 1.37–3.07; $p<0.001$). In a landmark analysis we found a significant higher mortality in patients with GLP-1 levels >median from day 30 to 1 year (HR 2.56; 95 %CI 1.08–6.09; $p=0.03$). In contrast, beyond 1 year up to 6 years no difference has been observed anymore (HR 1.02; 95 %CI 0.41–2.58; $p=0.96$).

Conclusion: Elevated plasma levels of GLP-1 are an independent predictor for impaired prognosis in patients with myocardial infarction complicated by CS at short-, intermediate and long-term follow-up. In a landmark analysis this prognostic effect is sustained up to 1 year. The functional relevance of GLP-1 in this context is currently unknown and needs further investigations.

PS 3/3-3

Interleukin-6 is the best predictor of long-term cardiovascular mortality in patients with acute coronary syndrome undergoing percutaneous coronary intervention

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Introduction: Interleukin-6 (IL-6) has a key position in the process of atherosclerosis and inflammation. Elevated IL-6 is associated with cardiovascular diseases such as myocardial infarction. Nevertheless, it is unknown what in the predictive accuracy for mortality of IL-6 as compared to the traditional predictors. Aim of the study is to characterize independent predictors of long-term CV mortality in patients with acute coronary syndrome (ACS) undergoing percutaneous coronary intervention (PCI).

Methods: We included 322 patients into this prospective observational study. Several biomarkers (IL-6; C-reactive protein [CRP]; high-sensitivity CRP [hsCRP]; CD40) were assessed at the time point of intervention. Patients were followed-up for 6 years. Adjusted Cox-regression analysis was used for prediction of events.
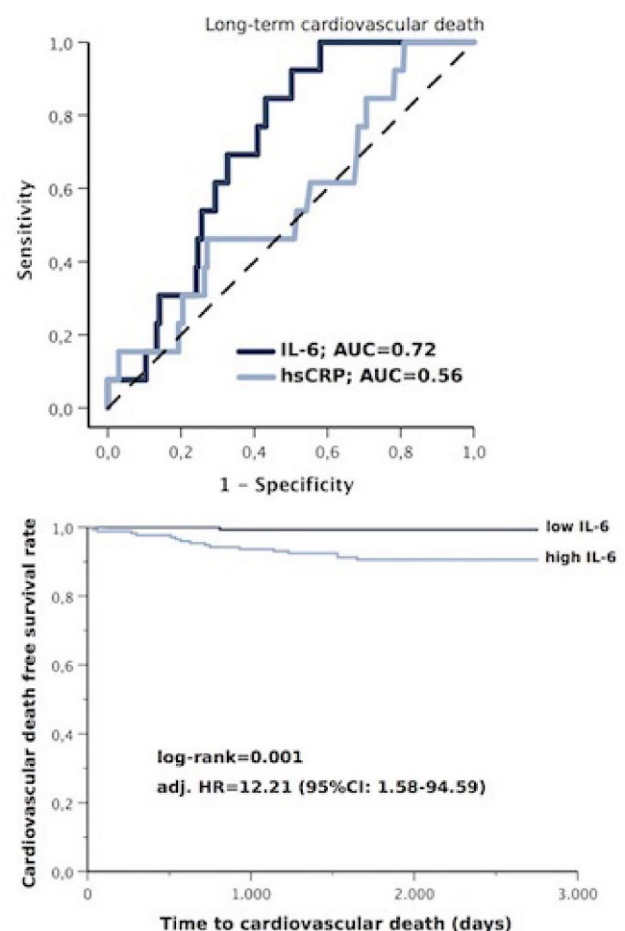
Abb. 1 | PS 3/3-3

Results: Elevated IL-6 values (≥ 3.3 pg/mL) emerged as an independent predictor for CV mortality. Only a moderate correlation between hsCRP levels and IL-6 subgroup (low/high) was found ($r = 0.394$; $p < 0.001$). The ROC curve analysis has shown that IL-6 was more accurate for CV mortality prediction than hsCRP (IL-6: AUC = 0.72; 95 %CI: 0.62–0.81; $p = 0.009$ vs hsCRP: AUC = 0.56; 95 %CI: 0.41–0.72; $p = 0.445$; Fig. 1). The primary endpoint of long-term cardiovascular death occurred more frequently in patients with high IL-6 vs low IL-6 (0.5 % vs 9 %; $p = 0.001$). The multivariate cox regression analysis has revealed that patients with increased IL-6 values were at 12-fold higher risk to die than those with low IL-6 (adj. HR = 12.21, 95 %CI: 1.58–94.59; Fig. 2). The positive predictive value of IL-6 to predict mortality was 9 %, the negative predictive value 99 %, sensitivity 94 % and specificity 48 %. Only other independent predictor of mortality was presentation with diabetes (adj. HR = 4.18, 95 %CI: 1.39–12.53).

Conclusion: In the setting of ACS, increased IL-6 values are associated with higher long-term CV mortality with an excellent negative predictive value of 99 % and sensitivity of 94 %. IL-6 performs as a superior predictor for CV death as compared to the traditional risk markers and hsCRP as well.

PS 3/3-4

Potent platelet inhibition results in improved long-term clinical outcome independent from clinical variables

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Introduction: Dual antiplatelet therapy with P2Y12-inhibitors and aspirin is a cornerstone treatment in patients with ACS, who underwent percutaneous coronary intervention. Differences between clopidogrel and novel P2Y12-inhibitors (e. g. ticagrelor and prasugrel) are well-known. Aim of this study was to investigate whether dual antiplatelet therapy with potent P2Y12-inhibitors as compared to clopidogrel is associated with long-term clinical benefit.

Methods: In this prospective observational study we enrolled 708 patients with acute coronary syndrome (ACS) treated with clopidogrel ($n = 137$), ticagrelor ($n = 260$) or prasugrel ($n = 311$). Major adverse cardiac events (MACE over 1 year) and long-term mortality (over 5 years) were assessed. Multiple
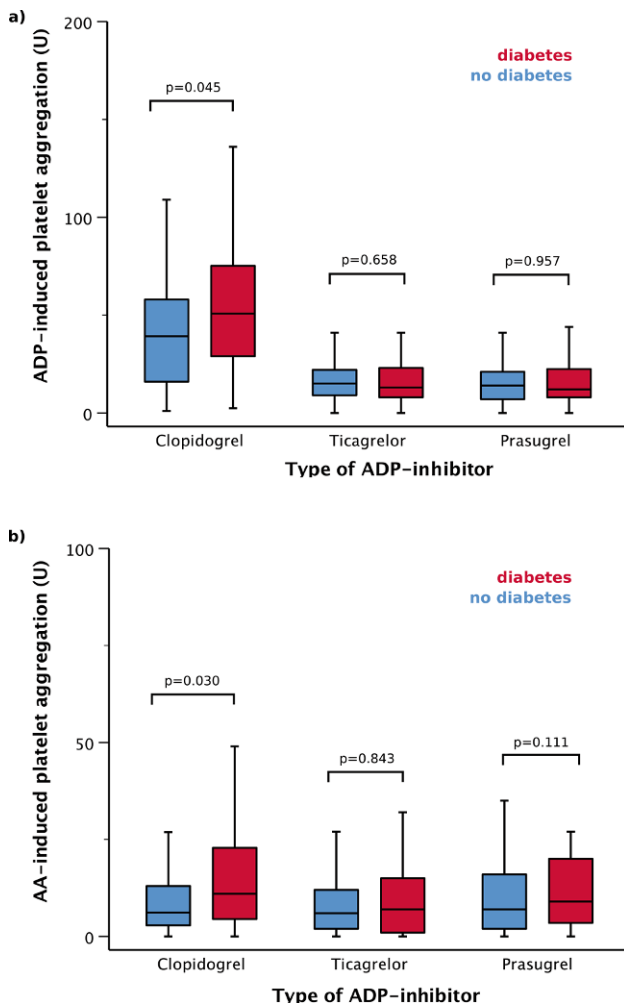
Fig. 1 | PS 3/3-4
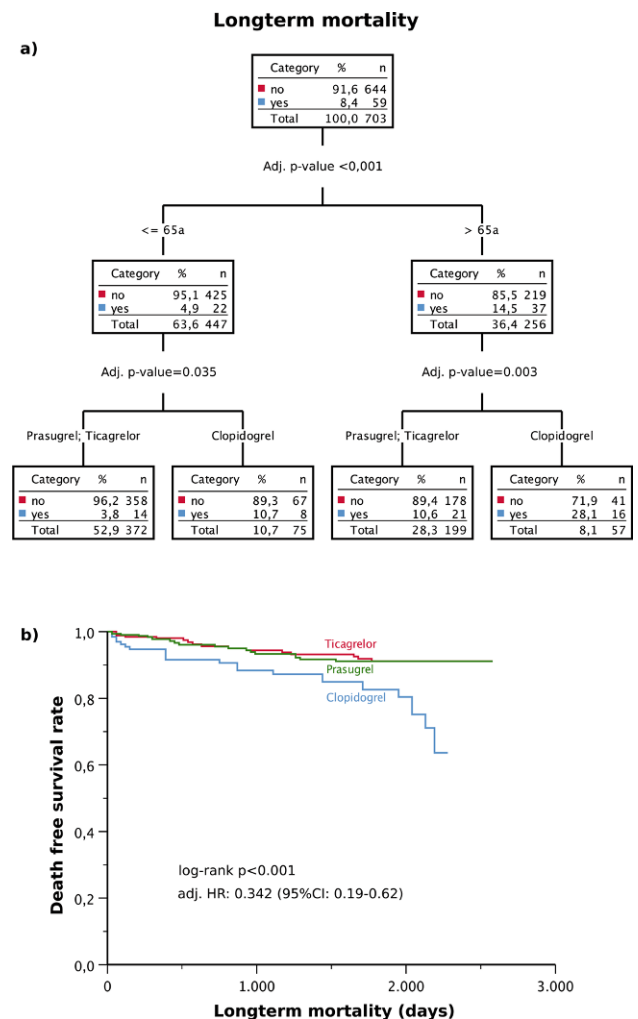
Abb. 2 | PS 3/3-4

Electrode Aggregometry (MEA) was used to define ADP- (adenosine diphosphate) and AA- (arachidonic acid) induced platelet aggregation.

Results: Type of ADP-inhibitor emerged as independent predictor of long-term mortality and MACE: patients treated with potent platelet inhibitors prasugrel or ticagrelor were at lower hazard to die within 5 years (adjusted HR = 0.350; 95 %CI: 0.20–0.61) or experience a MACE (adjusted HR = 0.345; 95 %CI: 0.20–0.59) as compared to clopidogrel. The effect of prasugrel and ticagrelor was homogenous independently from clinical risk factors. In contrast, clopidogrel effect decreased with increasing severity of ACS: patients with STEMI (ST-segment elevation myocardial infarction) had 37 % and with NSTEMI (Non-ST-elevation myocardial infarction) 25 % higher platelet aggregation as compared to patients with instable angina ($p = 0.039$). Patients diagnosed with diabetes achieved less potent ADP and AA- induced platelet inhibition when treated with clopidogrel, compared to those without diabetes ($p = 0.045$; $p = 0.030$, respectively).

Conclusion: In the setting of ACS, treatment with ticagrelor or prasugrel is associated with an improved long-term mortality and short-term MACE as compared to clopidogrel.

PS 3/3-5

NSTEMI as initial manifestation of pheochromocytoma induced multi organ failure

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Introduction: A 62 year-old female patient presented with epigastric pain radiating to the thorax, and vomiting. She had a past medical history of neurofibromatosis type 1 (NF1) and active smoking (60 packyears). No permanent medication was taken before admission. Her vital signs at presentation were blood pressure 191/118 mm Hg, heart rate 110 beats per minute (bpm), temperature 35.2 °C, and peripheral oxygen saturation of 96 %.

Methods: Diagnostic measures included electrocardiogram (ECG), chest X-ray, body CT scan, echocardiography, and laboratory analyses.

Results: The ECG showed left anterior hemi block, no R-progression in V1–V6, and a negative T-wave in aVL. The chest X-ray did not show any signs of pleural effusion, pulmonary edema or pneumonia. Echocardiography revealed normal ejection fraction but severe myocardial hypertrophy. Laboratory analyses showed elevated hs-Troponin T (166 pg/ml; 14 pg/ml upper reference limit), rising to 530 pg/ml after 1 h, consistent with the diagnosis of NSTEMI. Coronary angiography identified a significant stenosis of the right coronary artery which was treated with PCI. In the following hours, the patient's alertness decreased successively, and laboratory diagnostics revealed multi organ failure and lactic acidosis. The patient was intubated and suffered recurrent ventricular tachycardia with necessity for CPR. Hemoglobin levels dropped dramatically. A CT scan revealed a structure highly suspicious of pheochromocytoma accompanied by an active bleeding (Fig. 1). In addition, subarachnoid bleeding in the right hemisphere and a large
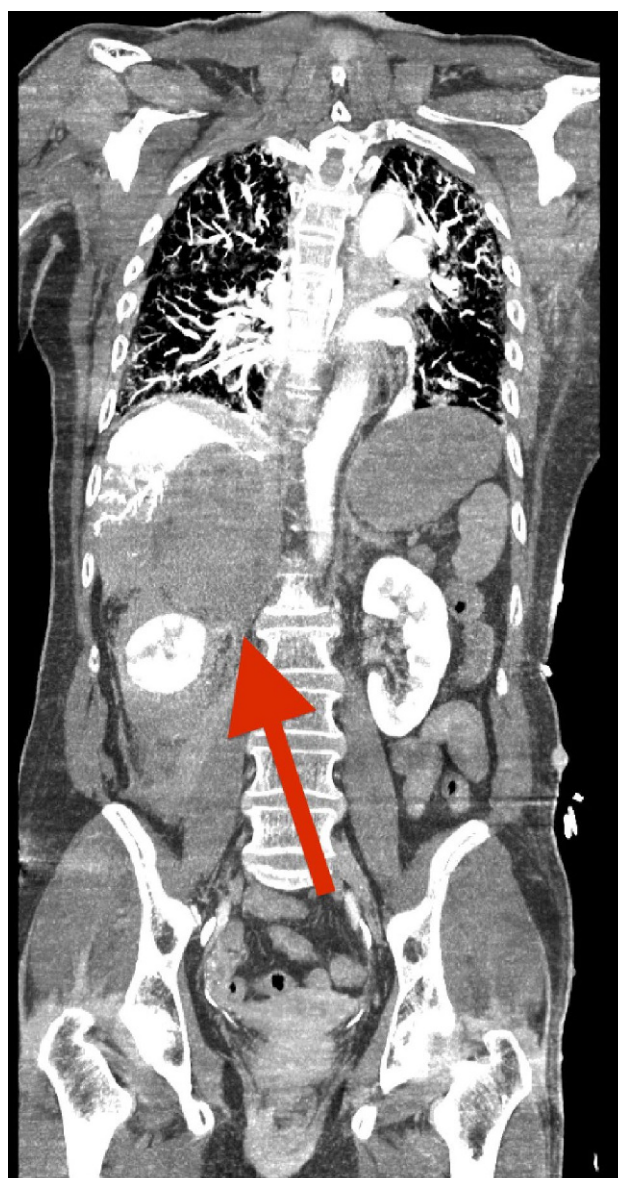
Abb. 1 | PS 3/3-5 Abdominal CT-Scan coronal view, the red arrow is pointing to the pheochromocytoma

retroperitoneal hematoma were identified. The patient died 16 h after initial admission due to multi organ failure. Autopsy confirmed the diagnosis of pheochromocytoma. Postmortem analysis revealed extremely high levels of norepinephrine (103.000 pg/ml; 200 pg/ml upper reference limit) and epinephrine (33.700 pg/ml; 88 pg/ml upper reference limit). Collectively, laboratory analyses and imaging confirm the diagnosis of acute pheochromocytoma crisis.

Conclusion: Pheochromocytoma crisis mimicked the classical presentation of NSTEMI. In patients with acute coronary syndrome and known neurofibromatosis, suspicion of potential pheochromocytoma crisis may accelerate diagnostics, appropriate therapy, and may potentially avoid lethal multi organ failure.

PS 3/3-6

Proteomic profiling of acute stent thrombi reveals critical involvement of the complement system

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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. All-cause mortality was assessed using Kaplan-Meier analysis.

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 $p = 0.032$). 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.

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.

PS 3/3-7

Prognostic impact of left ventricular thrombus resolution after myocardial infarction on cardiovascular events and mortality

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Introduction: Left ventricular thrombus (LVT) is a rare but dreaded complication during the acute phase of acute coronary syndrome (ACS). Incidence rates differ among the observational studies from 1.6 % up to 39.0 % indicating that many LVT cases might remain undetected. While the prognosis of patients presenting with LVT after ACS has been controversially discussed, it seems intuitive that individuals in whom the thrombus remains have an increased risk for cardiovascular events and mortality. However, profound data on long-term outcome of this highly vulnerable patient population are not available in current literature. Therefore, we aimed to investigate the impact of LVT resolution on patient's outcome from a long-term perspective.

Methods: We collected data of patients with acute coronary syndrome ($n = 2011$) who underwent treatment between 01/2016 and 09/2019. Patients with a confirmed LVT were included in this analysis. Repeated echocardiographic data, treatment management and clinical outcomes were collected during follow-up. All-cause mortality and major adverse cardiac events (MACE), defined as nonfatal stroke, nonfatal myocardial infarction, nonfatal systemic embolism and cardiovascular death were chosen as primary and secondary endpoints.

Results: Among 2011 patients with ACS, 52 patients (2.6 %) developed left ventricular thrombosis (median age: 63 ±13). 80.5 % of LVT patients presented with STEMI. Out of those 52 patients with LVT, 6 died before hospital discharge and 3 did not receive follow-up imaging. In total 13 patients (24 %) died. Mean time to thrombus resolution was 23 ± 31 weeks. Mean follow-up time was 98 ± 69.6 weeks. 43 patients received oral anticoagulation including 7 patients (16 %) receiving novel oral anticoagulants (NOACs) and 32 patients (84 %) Vitamin K antagonists
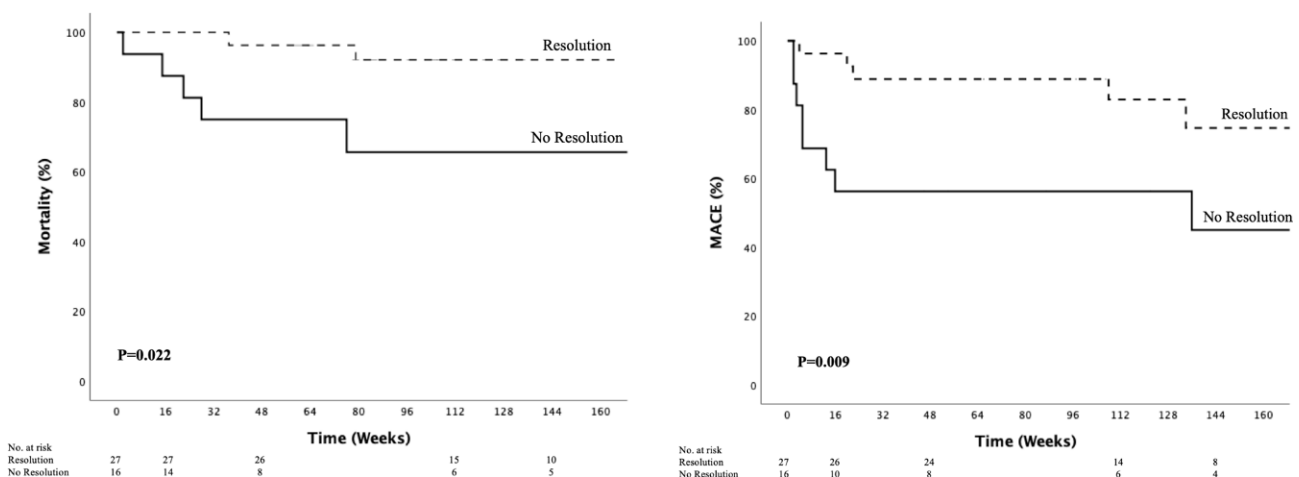
Abb. 1 | PS 3/3-7 Survival Curves

(VKA). All patients developed LVT after anterior wall infarction. From the time of hospital admission all patients were followed prospectively until the primary endpoint was reached. Thrombus resolution was observed in 27 patients (62.8). As expected, thrombus resolution was associated with a significant lower risk of MACE with a crude hazard ratio (HR) of 3.89 (95 % CI 1.30–11.65; $P = 0.015$) and mortality with a crude HR of 5.59 (95 % CI 1.07–29.07; $P = 0.041$). Notably, the prognostic impact remained stable after comprehensive adjustment for potential confounders with an adjusted HR of 5.38 for MACE and an adjusted HR of 6.10 for all-cause mortality.

Conclusion: Present data clearly highlighted the prognostic potential of thrombus resolution on both MACE and all-cause mortality in individuals presenting with LVT after ACS. Therefore, thrombus resolution might be considered for risk stratification and an intensified anti-thrombotic approach should be taken into account in this highly vulnerable patient population.

PS 3/3-8

Impact of Copeptin and Troponin-I levels on mortality in patients with suspected myocardial infarction

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Introduction: High Copeptin and Troponin I serum levels are associates with poor survival. This study aims to investigate the survival in patients with different serum levels of these biomarkers.

Methods: In a retrospective analysis, 1214 unselected consecutive patients with suspicion of acute MI with determined Troponin-I (TnI) and Copeptin (Cop) plasma levels were included. Patients were classified into 6 groups: Gr1–negative TnI (below detection limit; LOD) and Cop (Cutoff-level 10 pmol/l); Gr2–negative TnI and positive Cop; Gr3–TnI between LOD and 99th percentile upper reference limit (URL) and negative Cop; Gr4–TnI between LOD and URL and positive Cop; Gr 5–positive TnI (URL) and negative Cop; Gr 6–positive TnI and Cop. A Kaplan-Meier survival analysis was conducted and a pairwise comparison between groups was performed.

Results: The mean survival time within the Gr1–6 was as followed: 67.1 months (95 %CI 66–68.1), 61.2 months (95 %CI 58.6–63.8), 57.3 months (95 %CI 50.4–64.2), 52.3 months (95 %CI 43.4–61.3), 67.5 months (95 %CI 65.3–69.8) and 54 months (95 %CI 49.6–58.4), respectively. A log rank test was conducted and the survival distributions for the six groups were statistically signifi-

cantly different ($p < 0.005$). Pairwise log rank comparisons were conducted to determine which groups had different survival distributions. There was a statistically significant difference in survival distributions for Gr1 vs Gr2 ($p < 0.005$), Gr1 vs Gr3 ($p < 0.005$), Gr1 vs Gr4 ($p < 0.005$), Gr1 vs Gr6 ($p < 0.005$), Gr2 vs Gr4 ($p = 0.017$), Gr2 vs Gr5 ($p = 0.008$), Gr2 vs Gr6 ($p = 0.002$), Gr3 vs Gr5 ($p < 0.005$), Gr4 vs Gr5 ($p < 0.005$) and Gr5 vs Gr6 ($p < 0.005$).
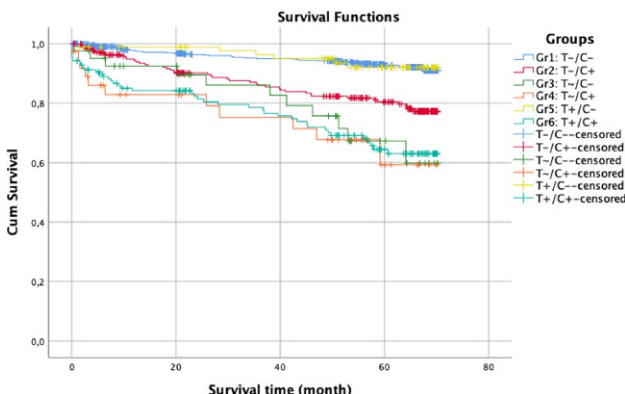
Conclusion: In the groups with patients with TnI below LOD as well as with TnI above URL copeptin serum levels have a significant impact on survival time. However, in the groups with TnI between LOD and URL (Gr3–4) Copeptin has no impact on survival time.

POSTERSITZUNG 4 – BILDGEBUNG

PS 4/4-1

Clinical risk score to predict early left ventricular thrombus after percutaneous coronary intervention for ST-elevation myocardial infarction

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Introduction: Compared with transthoracic echocardiography (TTE), cardiac magnetic resonance (CMR) imaging has a much higher sensitivity for left ventricular (LV) thrombus detection in patients after acute ST-elevation myocardial infarction (STEMI). However, routine CMR imaging is currently not recommended post-STEMI. This observational study sought to develop a practical risk score for the prediction of early LV thrombus formation after STEMI to identify patients in whom routine CMR might be appropriate.

Abb. 1 | PS 3/3-8
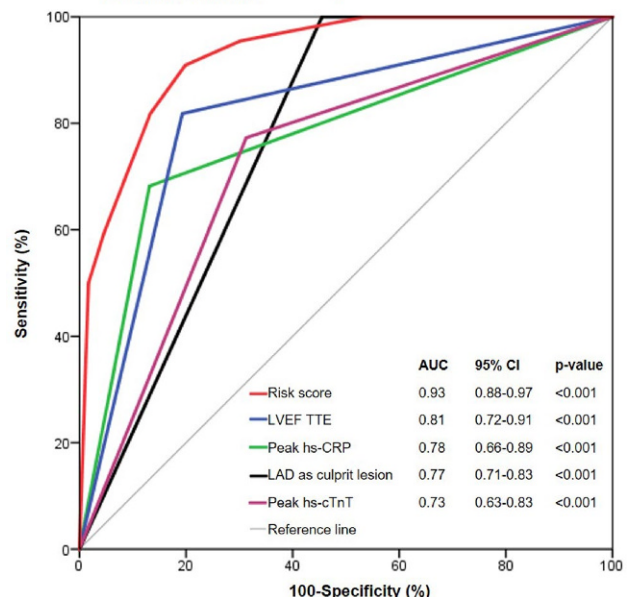
Abb. 1 | PS 4/4-1

Methods: Five hundred and fifty-six consecutive STEMI patients underwent TTE and CMR at 3 [IQR: 2–4] days after primary percutaneous coronary intervention (PCI) for acute STEMI.

Results: A LV thrombus was visualized in 12 patients (2.2 %) using TTE and in 22 patients (4 %) using CMR. A weighted risk score including multivariable associates of LV thrombus formation (LV ejection fraction by TTE, peak high-sensitivity cardiac troponin T and peak high-sensitivity C-reactive protein) and left anterior descending coronary artery as culprit vessel, with a range of 0 to 7 points (median risk score: 2 points) showed a strong and significantly higher area under the curve (0.93; 95 % CI 0.88–0.97; $p < 0.001$) for LV thrombus prediction than each individual risk factor alone ($p < 0.001$) (Fig. 1). The sensitivity and the specificity of the risk score was 91 % and 80 %, respectively. The incidence of LV thrombi was 0.3 % in patients with 0 to 2 points ($n = 374$, very low risk group) and 11.5 % in patients with 3 or more points ($n = 182$, high risk group).

Conclusion: The proposed risk score provides great value for the prediction of early LV thrombus and could be useful to identify STEMI patients in whom routine CMR for LV thrombus evaluation could be most meaningful. Additional investigation is warranted to validate the clinical application of the score.

PS 4/4-2

Changes in right ventricular function and cardiac output in patients after transcatheter tricuspid valve repair

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Introduction: Changes in right ventricular (RV) function and cardiac output in patients after transcatheter tricuspid valve repair (TTVR) remain under discussion. Recent restrospective registry data indicated a significant increase of tricuspid annular plane systolic excursion (TAPSE) and fractional area change (FAC) 6 months after TTVR. Cardiac output increased, but not significantly. Conversely, other authors showed a trend towards decreases of TAPSE and RV systolic tissue Doppler parameters (S-TDI) 30 days after TTVR.

Methods: The aim of this study was to investigate the evolution of RV function and cardiac output after TTVR. The following echocardiographic parameters were attained before and after edge-to-edge TTVR: Severity of TR (vena contracta, effective regurgitation orifice area, regurgitation volume), TAPSE, fractional area change (FAC), and NYHA functional class. Cardiac output (CO) and cardiac index (CI) were calculated. Changes after TTVR were determined applying a paired sample t-test.

Results: Pre- and post-procedural echocardiography were available in 20 (76.7 ± 5.5 years, 65 % female) patients who underwent TTVR between 01/19 und 02/20, 28 (±37) days before, and 72 (±88) days after the intervention. TR was reduced by at least 1 grade in 90 % of patients. No significant changes of the following parameters were recorded after TTVR: TAPSE (16.4 ±6 vs 16.2 ±4.5 cm, $p = 0.898$), FAC (0.41 ±0.11 vs. 0.40 ±0.1, $p = 0.875$), CI (1.9 ±0.7 vs. 1.7 ±0.5 $p = 0.171$), CO (3.5 ±1.4 vs. 3.1 ±0.9). NYHA functional class improved in 72 % of patients (grade ≥III, 68 % pre-procedure vs. 23 % post-procedure, $p = 0.063$).

Conclusion: Although the severity of TR and functional status improved, we could–at this early point–not show significant changes of functional RV parameters after TTVR.

PS 4/4-3

Echocardiographic right ventricular function assessment in patients with chronic heart failure–comparison between TAPSE and myocardial deformation analysis

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Introduction: Growing evidence points towards a pivotal role of right ventricular function (RV) for disease burden in patients with chronic heart failure. Echocardiographic determination of RV myocardial deformation parameters is a time consuming but promising method that might be superior to conventional parameters of RV function, such as TAPSE. In a prospective heart failure cohort, we measured different echocardiographic parameters of RV function and assessed correlations with NT-proBNP using multivariate statistical models.

Methods: In the present analysis of the "Role of Comorbidities in Chronic Heart Failure" (RoC-HF) study, we performed off-line speckle tracking analysis of all study participants with appropriate echocardiographic examination. Main inclusion criteria were a previous diagnosis of chronic heart failure and a left ventricular ejection less than 50 %. Speckle tracking was performed using the software Image Arena (TOMTEC Imaging Systems GmbH, Unterschleissheim, Germany).

Results: We were able to acquire speckle tracking data from 101 patients (49 % of the overall cohort) who had a mean age of 64 ± SD 10 years. NT-proBNP was 805 (IQR 244–2296 pg/ml) and mean LVEF was 37 ±10 %. Mean RV end-systolic endocardial global longitudinal strain (RV GLS) was –20 ±6, mean RV fractional area change (RV FAC) was 39 ±10 %, and TAPSE was 18 ±7 mm. All parameters of right ventricular function correlated significantly with NT-proBNP in univariate correlation analysis (RV GLS: Pearson r = 0.247, $P = 0.016$; RV FAC: r = –0.206, $P = 0.038$; TAPSE: r = –0.386, $P < 0.001$). However, in multivariate linear regression analysis with adjustment for age, sex, BMI, serum creatinine, LVEF, right atrial area, and diagnosis of atrial fibrillation, only TAPSE was significantly correlated with NT-proBNP (adjusted beta = –0.193, $P = 0.009$). Using the same model, neither RV GLS nor RV FAC were significantly related with NT-proBNP (RV FAC: beta = 0.021, $P = 0.807$; RV GLS: –0.009, $P = 0.912$).

Conclusion: In a carefully selected cohort of patients with chronic heart failure, TAPSE was a more valuable parameter to determine disease severity than RV GLS. Our study raises doubts about the clinical usefulness of RV myocardial deforma-

tion analysis, the more as it can not be performed in a relevant subset of patients and is time-consuming.

PS 4/4-4

Aortic forward flow at baseline after ST-elevation myocardial infarction–prognostic value of phase contrast magnetic resonance imaging

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Introduction: The value of non-invasively assessed hemodynamic parameters by velocity encoded phase-contrast imaging at aortic level in comparison to established parameters of outcome after ST-elevation myocardial infarction (STEMI) on outcome is not known.

Methods: In this observational and prospective study comprehensive CMR was performed a median of 3.3 days after STEMI in 383 patients treated by primary percutaneous coronary intervention. Aortic forward flow (AFF) was assessed by phase contrast imaging, left ventricular ejection fraction (LVEF) and stroke volume (SV) by cine MR. Major adverse cardiac events (MACE) were defined as the composite of death, myocardial infarction or hospitalization for heart failure.

Results: Median age of the total population was 56 years (IQR 16), 83 % were male. Mean Body Surface Area was 2 ± 0.2 m[2]. Median LVEF was 53 % (IQR 14), median AFF 75 ml (IQR 27) and median SV 76 ml (IQR 25). AFF and SV were moderately correlated ($r = 0.71$; $p < 0.0001$). During a median follow-up of 13.5 (IQR 24) months, 36 patients experienced a MACE event. Patients with AFF of less than 68 ml had a significantly higher risk of MACE during follow up ($p = 0.001$; log rank). In multivariate Cox regression analysis adjusted for age, sex and LVEF an AFF lower than 68mls was significantly predictive of MACE (HR 2.4 95 % CI 1.1–4.9, $p = 0.02$) whereas a LVEF of < 53 % was not (HR 1.7 95 % CI 0.8–3.5, $p = 0.07$).

Conclusion: At baseline after acute STEMI an AFF of < 68 ml assessed with phase-contrast imaging was a stronger prognostic marker for MACE than a LVEF of < 53 %. Absolute, "per beat" values performed better than indexed hemodynamic markers.

PS 4/4-5

Multimodal imaging appraoch and 3D rendering to map cardiac Vagus Nerve anatomy and morphology

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Introduction: Although heart transplantation (HTx) is known as a well-established gold standard in the treatment of patients with end-stage heart failure (HF), patients still suffer from various negative side-effects that are caused by the consequences of complete allograft denervation during operation including higher heart rate (HR) at rest and slow acceleration of HR during exercise. Therefore, early parasympathetic reinner-

Fig 1: VN isolation from sheep (A) and human (B) cadaver.
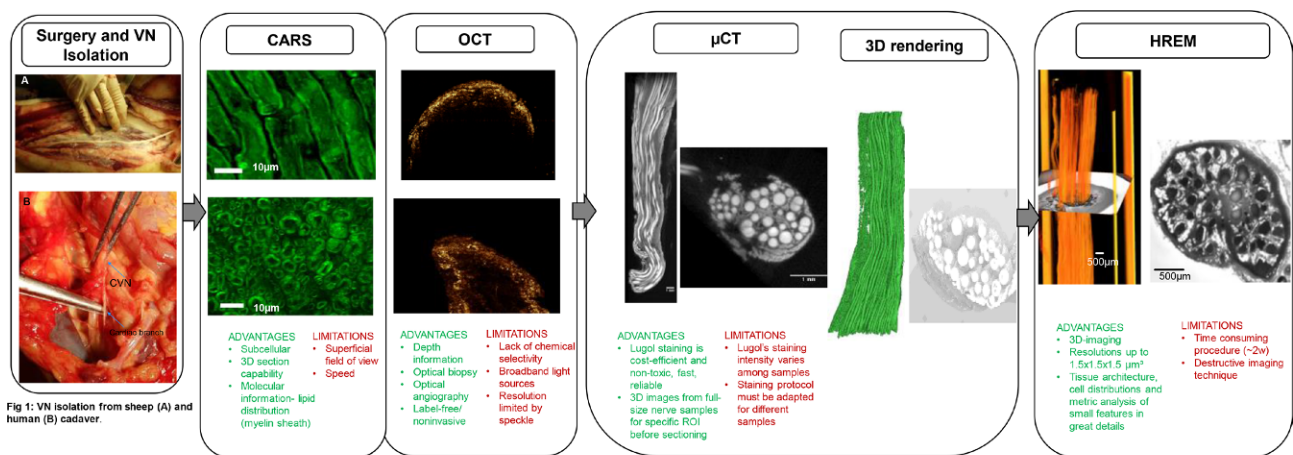
Fig2. Multimodal Imaging Pipeline to map Vagus Nerve anatomy.

Fig. 1 | PS 4/4-5 Vagus Nerve (VN) transplantation and Multimodal Imaging Pipeline (MMIP)

vation is a desirable goal and interesting area of research in HTx patients1. Within this project, we aim to study the Vagus Nerve (VN) anatomy and morphology, with a focus on its cardiac branches, using a multimodal imaging approach that allows the creation of an anatomical 3D model as basis for a regenerative neural interface of the VN.

Methods: Cervical Vagus Nerve (CVN) and subsequent cardiac branches were freshly isolated from New-Zeeland female rabbits ($n=8$) and female sheep ($n=4$). In addition, CVN (fixed) were obtained from human cadaver ($n=2$; with cardiac branches) and pigs ($n=2$, without cardiac branches). Fresh samples were first imaged using a multimodal optical setup including coherent anti-Stokes Raman scattering (CARS) microscopy and optical coherence tomography (OCT)2. The combination of OCT and CARS provides access to detailed information of tissue structure and molecular composition in a fast, label-free and non-invasive manner. CARS was used to image myelin content of the nerve and OCT served as a guiding tool for localization of CARS information within the VN. After fixation in formalin, samples were analyzed via contrast-enhanced micro-CT (μCT)3 and high-resolution episcopic microscopy (HREM). Based on the data obtained, a 3D model of the VN anatomy was created. A preliminary evaluation of the multiple imaging modalities has been investigated to characterize the anatomy of the cardiac VN in greater detail.

Results: First proof-of-concept results showed the feasibility of the multimodal imaging pipeline to map the VN anatomy including cardiac branches. Multimodal imaging in combination with 3D reconstructions of the VN could display the anatomical structure of single fascicles from cardiac branches up to CVN as well as their main features, such as myelin sheaths, fascicle number, fascicle area, internal branching, and twisting (Fig. 1 + 2). With an average fascicle number of 7 fascicles per nerve in the CVN and a mean nerve area of 5.2 mm^2, the pig models were closest to the humans (A = 4.6 mm^2) as also described in literature. However, the branching patterns from the CVN to the heart showed certain differences between species (pigs, sheep, rabbit, humans), among which the pigs were also most similar to humans in this aspect.

Conclusion: Our multimodal imaging approach has the potential to display the cardiac VN in multiple anatomical aspects that enable the creation of anatomical models in greater details including its branching. Results showed that large animal models, in particular pigs, represent the most suitable models for anatomical studies because of the similarity in size and morphology of its VN compared to the human one. These models can provide a wealth of information to support the design, development and test of a novel, smart cardiac neuroprosthesis for HTx patients. This work was funded by the EU-project "A neuroprosthesis to restore the vagal-cardiac closed-loop connection after heart transplantation, NeuHeart" H2020-EU.1.2.2. Grant agreement ID: 824071

PS 4/4-6

Novel echocardiographic strain parameters for the estimation of myocardial infarct size in STEMI

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Introduction: Prognosis and outcome of patients after ST-elevation myocardial infarction (STEMI) is primarily determined by final infarct size. Gold standard for the calculation of infarct size is cardiac magnet resonance (CMR), which is expensive, laborious and has only limited availability. In comparison, transthoracic echocardiography (TTE) is widely available, cost
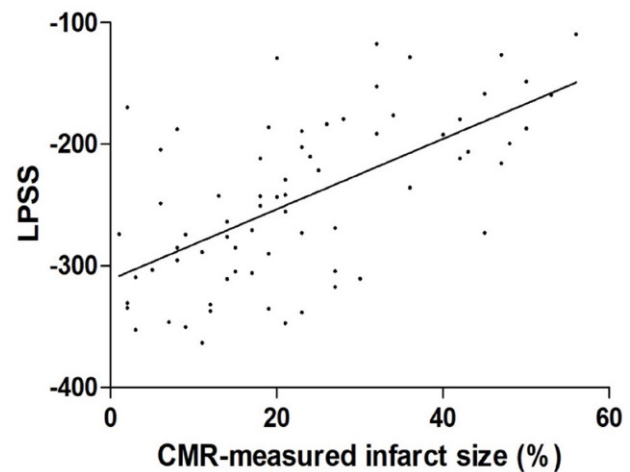
Fig. 1 | PS 4/4-6 Correlation of cumulative longitudinal peak systolic strain (LPSS) and cardiac magnet resonance (CMR)-measured infarct size (%). (n = 70, r = 0.620; p < 0.0001)
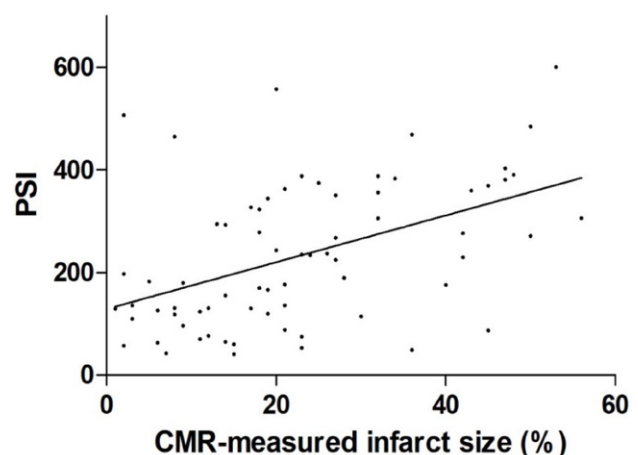
Fig. 2 | PS 4/4-6 Correlation of cumulative post-systolic shortening index (PSI) and cardiac magnet resonance (CMR)-measured infarct size (%). (n = 70, r = 0.472; p < 0.0001)
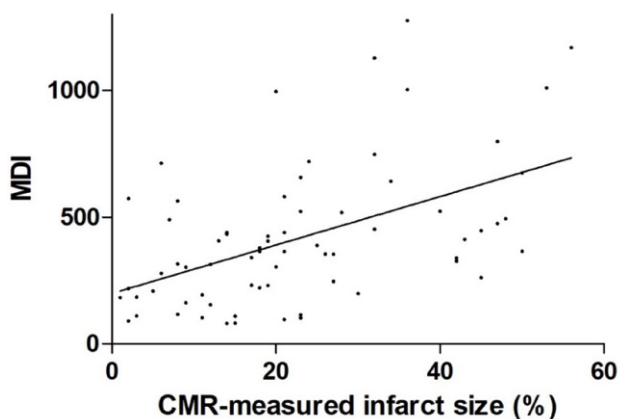
Fig. 3 | PS 4/4-6 Correlation of cumulative myocardial dysfunction index (MDI) and cardiac magnet resonance (CMR)-measured infarct size (%). (n = 70, r = 0.500; p < 0.0001)

efficient, and practicable at the bedside. Still, standard TTE is not very accurate in the detection of ischemic wall segments. Global longitudinal peak systolic strain (GLPSS) measured by speckle tracking is already used in clinical routine to estimate cardiac function.

Methods: STEMI patients ($n = 70$), who were included in a prospective, controlled trial at the General Hospital of Vienna between 2013 and 2016, were analyzed in the study. Analysis was performed using speckle tracking software (EchoPacs, GE healthcare). The following values were measured in each segment: longitudinal peak systolic strain (LPSS, maximum strain during systole), post-systolic shortening (PSS, amount of shortening after systole), early systolic lengthening (ESL, amount of lengthening in systole), early systolic lengthening time (ELT, time to peak lengthening) and time to peak event (TTP, time to overall shortening). Using these measurements, the following index parameters were computed: PSI ([PSS/maximal strain in the cardiac cycle (maxStrain)]*100), early systolic lengthening index ([ESLI/maxStrain]*100), myocardial dysfunction index (MDI [(ESL amplitude + PSS amplitude)/maxStrain]*100). All segment values of each parameter were added up in each patient. These cumulative values were correlated with infarct size, obtained by CMR 4 ±2 days post STEMI, using Pearson's correlation. Furthermore, the GLPSS (highest average strain at one point in time) was correlated with CMR.

Results: We included 70 STEMI patients (55.5 ±10 years, 18.6 % female), in which CMR (mean infarct size 22.79 ± 14.5 %; 4 ±2 days post admission) and TTE (5 ± 5 days post admission) data were available. LPSS (r = 0.620; $p < 0.0001$), GLPSS (r = 0.588; $p < 0.0001$), MDI (r = 0.500; $p < 0.0001$), PSI (r = 0.472; $p < 0.0001$) and ESLI (r = 0.426; $p < 0.0001$) were positively correlated with infarct size obtained by CMR. ELT, TTP and ESL were not correlated with CMR-measured infarct size.

Conclusion: Novel strain parameters significantly correlate with infarct size and could be useful measurements to predict final infarct size and possibly outcome of STEMI patients.

Cardiac remodeling in ambitious endurance-trained amateur athletes older than 50 years

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Introduction: Data on cardiac remodeling in veteran athletes are scarce. Therefore, in this study we aimed to define echocardiographic features of healthy endurance athletes older than 50 years.

Methods: We included probands aged >50 years, who have performed endurance sports at 70 % of their maximum heart rate for at least 1 h 3 times per week over the previous 5 years.

Results: Between November 2018 and May 2019, 69 probands were recruited. Median age was 57 years (IQR 52–64) and 26 % were female. Groups were formed according to the median training time of 6 h per week: (A) 45 probands with >6 h (6–10) and (B) 24 probands with <6 h (3.5–5). Age, sex and body mass index were similar. Group A demonstrated slightly smaller right atrial and left atrial (LA) diameters when compared to probands in B (53 mm (49–55) vs. 53 mm (51–58), $p = 0.045$, and 52 mm (49–55) vs. 53 mm (52–58), $p = 0.039$, respectively) and showed preserved diastolic function ($p = 0.026$) with lower E/E' ratio (7 (6–9) vs. 9 (7–10), $p = 0.039$). LA volumes, interventricular septal thickness and relative wall thickness ratio were similar. Global right and left ventricular (LV) strain were similar, but LA reservoir strain was higher in group A than in B (27 % (22–34) vs. 20 % (15–29), $p = 0.016$).

Conclusion: Endurance training in healthy athletes older than 50 years does not result in relevant chamber dilatation or LV hypertrophy and a weekly training duration of >6 h seems beneficial to preserve diastolic function associated with an increased LA reservoir function.

Assoziation von Early-Repolarization und aberranten Sehnenfäden bei Patienten mit linksventrikulärer Hypertrabekulierung/Noncompaction

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Einleitung: Aberrante Sehnenfäden des linken Ventrikels (LVAS) sind fibromuskuläre Strukturen, die im linken Ventrikel verlaufen und von einer ventrikulären Struktur zur anderen ziehen. LVAS lassen sich in der Echokardiographie bildgebend darstellen, jedoch besteht keine Empfehlung sie im Befund anzuführen. Assoziiert werden LVAS mit Herzgeräuschen, ventrikulären Arrhythmien und Störungen der Repolarisation im Ruhe-EKG. Early Repolarisation (ER) ist eine häufige EKG-Veränderung, definiert als J-Punkt Elevationen in mindestens zwei zusammengehörenden Ableitungen. Es wird vermutet, dass LVAS eine Rolle in der Entstehung von J-Punkt Erhöhungen und als Ursache von ER spielen. ER wird unter Patienten mit linksventrikulärer Hypertrabekulierung/Noncompaction (LVHT) häufiger gefunden als in der Normalbevölkerung (48 % vs. 5–13 %). Der Grund dafür ist noch unklar. Ziel dieser retro-

spektiven Studie war, herauszufinden, ob es einen Zusammenhang zwischen ER und LVAS bei Patienten mit LVHT gibt.

Methoden: Für diese Studie wurden Echokardiographiebilder von LVHT-Patienten auf das Vorliegen von LVAS retrospektiv analysiert. Eingeschlossen wurden Patienten, von denen ein lesbares EKG vorhanden war und deren Echobilder von ausreichender Qualität waren um LVAS diagnostizieren zu können.

Resultate: Es wurden 134 Patienten eingeschlossen (mittleres Alter: 55 Jahre, 32,8 % weiblich). Bei 36 (26,9 %) Patienten konnten LVAS gefunden werden. Auf Grundlage unserer Ergebnisse erfolgte die Einteilung der LVAS in 4 Typen: Typ 1 verbindet das basale interventrikulare Septum (IVS) mit der apikalen freien Ventrikelwand ($n=1$, 2,8 %); Typ 2: ist eine Verbindung zwischen mittleren IVS und mittleren freien Ventrikelwand ($n=7$, 19,4 %); Typ 3: verbindet das mittleren IVS und die apikaler freien Ventrikelwand ($n=3$, 33,3 %); Typ 4: ist eine Verbindung zwischen dem apikalen IVS und der apikalen freien Ventrikelwand ($n=16$, 44,4 %). Achtzehn Patienten (13,4 %) hatten LVAS und ER. Es konnte kein Zusammenhang zwischen LVAS und ER unter Patienten mit LVHT festgestellt werden. Unter den 18 Patienten mit LVAS und ER war ER in 7 Fällen (38,9 %) ausschließlich anterior und in 3 Fällen (16,7 %) ausschließlich inferior lokalisiert. Es gab keinen Zusammenhang zwischen den LVAS Typen und der ER Lokalisation. Der Vergleich von Patienten mit LVAS und Patienten ohne LVAS ergab keine signifikanten Unterschiede bezüglich Alter, Geschlecht, Komorbiditäten, EKG-Befunden, Symptomen, Therapien und Todesursachen. Patienten mit LVAS hatten signifikant niedrigere Durchmesser des IVS, als Patienten ohne LVAS (11 mm vs. 12 mm; $p=0,05$). Die mediane Follow-Up Zeit betrug 5 Jahre (IQR 2,5–8,3). Während dieser Zeit konnte kein Unterschied bezüglich Mortalität zwischen Patienten mit und ohne LVAS gezeigt werden

Schlussfolgerungen: Diese Studie konnte keinen Zusammenhang zwischen LVAS und ER unter Patienten mit LVHT feststellen.

POSTERSITZUNG 5 – HERZINSUFFIZIENZ 1

PS 5/5-1

Significant impact of prescription bias in the treatment of chronic systolic heart failure on outcome

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Introduction: Background and aim. There is irrevocable evidence that medical therapy with beta-blockers (BB) and renin-angiotensin-system inhibitors, uptitrated to target dosage (TD) decreases mortality and hospitalizations in heart failure with reduced ejection fraction (HFrEF). However, physicians seemingly do not sufficiently follow the guidelines and a great number of patients do not receive recommended dosages. As humans are visually deceivable, it might be that absolute numerical values of equipotent recommended TD and the
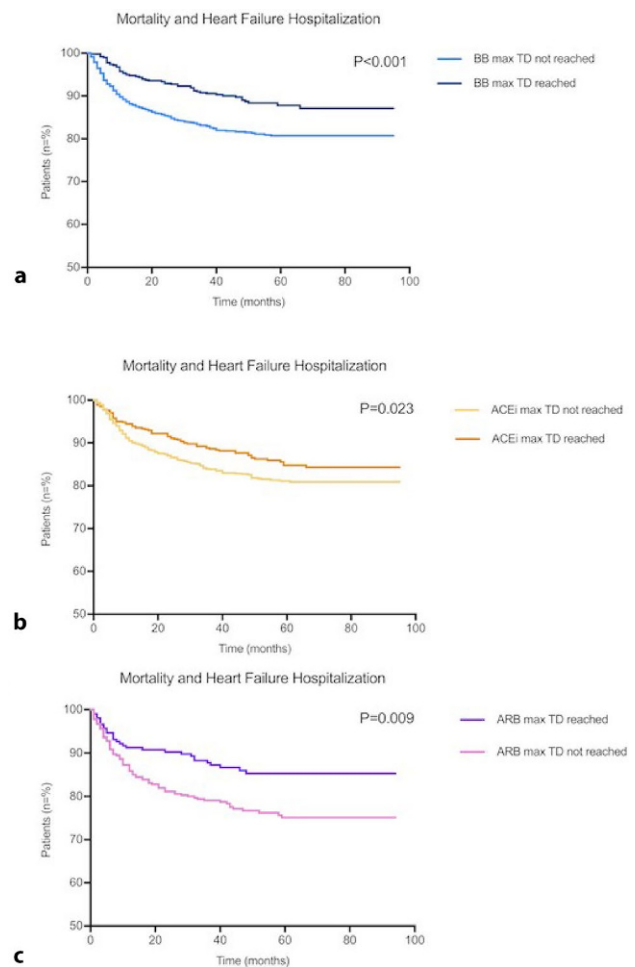
Abb. 1 | PS 5/5-1

"milligram-based" subjective weighting of tolerability by the individual physician influences the maximal prescribed dosage. We sought to assess whether different numerical TD of equipotent medications affect prescription patterns, potentially biases uptitration and impacts outcome.

Methods: Methods. Data of 3737 HFrEF outpatients were identified from a prospective registry. Maximal achieved dosages of BB, angiotensin-converting-enzyme inhibitors (ACEi) and angiotensin-receptor blockers (ARB) after one year of repeated visits for uptitration were assessed. BB, ACEi and ARB with largest numerical differences in TD (10 mg/d bisoprolol/nebivolol versus 200 mg/d metoprolol; 10 mg/d ramipril versus 40 mg/d lisinopril/enalapril/fosinopril; and 32 mg/d candesartan versus 320 mg/d valsartan) were compared using the individually achieved dose as percentage of the defined TD at one year of follow-up (FUP). The association of maximal TD achieved with HF hospitalization and overall survival were determined.

Results: Results. Median age was 65 years (IQR: 55–74), 2720 patients (73 %) were male. Within the whole study population, 1434 patients (38 %) received bisoprolol/nebivolol, 280 (8 %) metoprolol. Ramipril was prescribed in 599 (16 %) patients, lisinopril/enalapril/fosinopril in 1138 (30 %). 409 patients (11 %) were on candesartan, 173 (5 %) on valsartan. Significant differences at baseline were not clinically meaningful and there were no contraindications for uptitration in all subgroups. After one year aiming for up-titration dosages increased significantly in all medication-groups ($p < 0.001$ for all). How-

ever, significantly less patients were treated with the TD when TD was numerically higher (BB: metoprolol (57 (20 %)) versus bisoprolol/nebivolol (446 (31 %)), *p* <0.001; ACEi: lisinopril/enalapril/fosinopril (231 (20 %)) versus ramipril (313 (52 %)), *p* <0.001; ARB: valsartan (45 (26 %)) vs candesartan (166 (41 %), *p* <0.001)). At 45 (IQR: 32–68) months of FUP, 859 (23 %) of the patients were hospitalized for HF or died. Achievement of TD significantly improved outcome (Fig. 1A–C) and showed association with mortality and HF-hospitalization (BB: adj.HR: 0.87, 95 %CI 0.79–0.95; ACEi adj.HR 0.89, 95 %CI 0.81–0.97; ARB: adj. HR 0.74, 95 %CI 0.69–0.91; $P \leq 0.009$).

Conclusion: Conclusion. The present analysis described the influence of the numerical values of recommended TD of equipotent drugs on the prescription behavior of the treating physician and on outcome. This psychological phenomenon was identified a substantial confounder at least partly responsible for the underuse of BB, ACEi and ARB in HF. Moreover, it was never shown that this cognitive illusion based on risk aversion is linked to outcome.

PS 5/5-2

A novel SGLT-2 Score to identify HFpEF patients who may benefit from SGLT-2 inhibitors

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Introduction: Established therapies for the treatment of heart failure have shown no effects in patients with heart failure and preserved ejection fraction (HFpEF). However, subgroup analyses of the HFpEF patient populations of drug trials suggest that certain patients might benefit from certain heart failure treatments. This underlines the importance of thorough patient characterization and individualized therapy regimens, especially in the heterogeneous HFpEF population. Sodium-glucose transporter 2 (SGLT-2) inhibitors are emerging as a promising novel treatment of heart failure. The proposed mechanisms of action leading to improved outcomes in patients with heart failure include 1) treatment of diabetes as a cardiovascular risk factor, 2) osmodiuresis preventing volume overload, 3) enhancement of the cardio protective Angiotensin 1–7 pathway, instead of Angiotensin II which has a multitude of negative effects on the cardiovascular system. Our aim was to characterize subgroups of HFpEF patients by risk factors, which can be modified by SGLT-2 inhibitors in order to identify individuals who may especially benefit from these drugs.

Methods: HFpEF patients were included in a single center registry. Baseline evaluation included standard laboratory testing, assessment of HbA1c, fluid status measured by body composition monitor (BCM) and plasma angiotensin concentration using RAS-Fingerprint® (Attoquant Diagnostics, Vienna Austria). We calculated an "SGLT-2 score" with a possible maximum of 3 points each of the following parameters: 1) baseline HbA1c >6.5 %, 2) overhydration, defined as a fluid overload of >1.5L and 3) plasma renin activity (PRA) levels above the median of 365.8 pmol/L as a parameter off over-all RAS activity. All-cause death or heart failure hospitalization was defined as the primary outcome parameter. All parameters used for the "SGLT-2 score" were independently predictive for the chosen endpoint in a multivariable Cox regression analysis (table 2). Kaplan
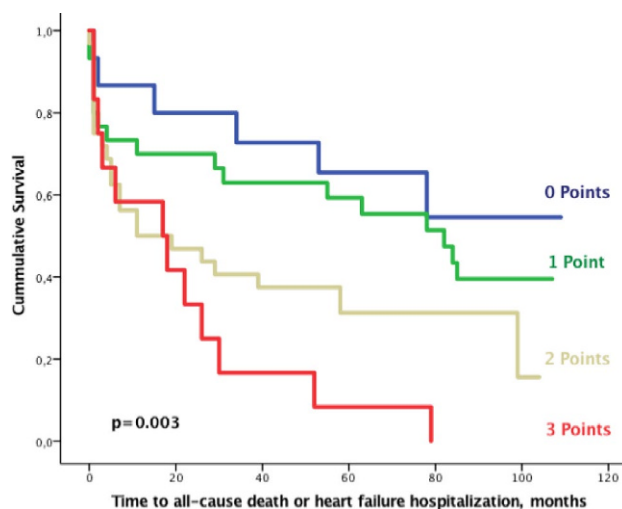
Abb. 1 | PS 5/5-2 Kaplan Meier analysis showing outcomes according to SGLT-2 score

		All-cause death or heart failure hospitalization		
		No	Yes	Total
SGLT-2 Score	0	9	6	15
	1	12	17	29
	2	9	26	35
	3	0	11	11
Total		30	60	90

Table 2- Multivariable Cox regression analysis for the effects of parameters of the SGLT-2 Score on the combined endpoint of all-cause death and heart failure hospitalization

	Multivariable HR	p-value
Age, years	1.02 (0.98-1.06)	0.342
Sex,	1.19 (0.67-2.13)	0.555
HbA1c, %	1.57 (1.20-2.06)	0.001
Overhydration, litres	1.24 (1.02-1.52)	0.032
PRA surrogate*, pmol/l	1.50 (1.07-2.09)	0.018

*PRA levels were categorized by tertiles

Abb. 2 | PS 5/5-2

Meier analyses were used to show the association between the SGLT-2 score and outcomes.

Results: In total, 90 patients were included into this registry. Median HbA1c was 6.0 %, (IQR 5.6–6.5) median fluid status was 1.2L (IQR 0.1–2.2) and the median angiotensin II levels of patients in the "high PRA-group" were 5.35.1 pmol/L (IQR 174.6–1589.5). Patient scores are shown in table 1. After a mean follow up time of 44.0 ± 38.7 months, 60 patients (66.6 %) reached the primary endpoint. Kaplan Meier analysis showed a significant association between number of points reached on the SGLT-2 Score and outcome ($p = 0.003$, figure 1).

Conclusion: Patients with HbA1c >6.5 %, overhydration and high RAS activity have poor outcomes. We propose the future use of this score to identify a subgroup of HFpEF patients who may benefit from treatment with SGLT-2 inhibitors.

PS 5/5-3

Echocardiograhic parameters and serum levels of cardioprotective angiotensin 1-7 in patients with heart failure and preserved ejection fraction

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Introduction: Angiotensin 1-7 (Ang 1-7) is a peptide of the renin-angiotensin system and counteracts the detrimental effects of Angiotensin II. Previously published data show that cardioprotective angiotensins, such as Ang 1-7 are up-regulated in more pronounced heart failure. So far, the association between echocardiographic parameters and Ang 1-7 levels is not known. We therefore sought to describe structural and functional parameters of the left and right ventricle in patients with high and low serum levels of Ang 1-7 in a cohort of heart failure and preserved ejection fraction (HFpEF).

Methods: Patients with HFpEF were consecutively included in a clinical registry. A comprehensive echocardiography exam was performed at baseline. In addition serum samples were taken and levels of Ang 1-7 were measured using mass spectroscopy. Patients were divided by median Ang 1-7 serum levels into a "high"- and "low Ang 1-7" group and echocardiographic parameters were compared between the two groups (Table 1).

Results: In total, 154 patients were included for data analysis. Patients in the "high Ang 1-7" group had larger right ventri-
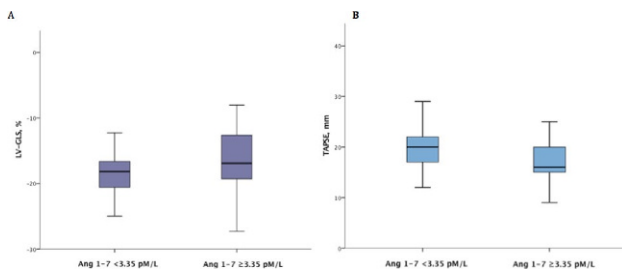
Fig. 1 | PS 5/5-3 A Left ventricular global longitudinal strain (LV-GLS) in the „low Angiotensin (Ang) 1-7" versus the „high Ang 1-7" group (p = 0.025). B Tricuspid annular plane systolic excursion (TAPSE) in the „low Angiotensin (Ang) 1-7" versus the „high Ang 1-7" group (p = 0.004)

	Ang 1-7 <3.35pM/L (n= 79)	Ang 1-7 ≥ 3.35 pM/L (n=75)	p-value
LVEDD, mm	45.0 (42.0-48.0)	43.0 (40.0-48.0)	0.181
LA length, mm	60.0 (56.0-65.0)	63.0 (58.0-67.0)	0.069
LVEF, %	57.0 (53.0-64.0)	58.0 (52.0-65.0)	0.714
LV-GLS, -%	18.0 (21.0-17.0)	17.0 (19.0-13.0)	**0.025**
E/A ratio	1.5 (1.1-2.0)	1.1 (0.9-2.3)	0.598
E/e' ratio	11.8 (10.6-14.0)	17.2 (12.0-21.0)	**0.017**
RVEDD, mm	35.0 (31.0-40.0)	39.0 (34.0-43.0)	**0.014**
RA length, mm	59.0 (56.0-64.0)	62.0 (58.0-70.0)	**0.024**
TAPSE, mm	20.0 (17.0-22.0)	16.0 (15.0-20.0)	**0.004**
RV-TDI, m/s	0.12 (0.10-0.16)	0.13 (0.10-0.15)	0.409
sPAP, mmHg	56.0 (41.0-71.0)	59.0 (48.0-75.0)	0.141

Ang indicates angiotensin; LVEDD, left ventricular end-diastolic diameter; LA, left atrium; LVEF, left ventricular ejection fraction; LV-GLS, left ventricular global longitudinal strain; RVEDD, right ventricular end-diastolic diameter; RA, right atrium; TAPSE, tricuspid annular plane systolic excursion; RV-TDI, right ventricular tissue Doppler index and sPAP, systolic pulmonary artery pressure.

Fig. 2 | PS 5/5-3 Left and right heart structural and functional echocardiography parameters in patients in the „low Angiotensin (Ang) 1-7" versus the „high Ang 1-7" group

cles and right atria and more pronounced diastolic dysfunction (E/e'). Left- and right ventricular function was more impaired in patients with higher serum levels of Ang 1-7, reflected by left ventricular global longitudinal strain (LV-GLS) and tricuspid plane systolic excursion (TAPSE), respectively (Fig. 1A and 1B).

Conclusion: In patients with HFpEF, left and right ventricular function is significantly poorer in patients with higher serum levels of Ang 1-7. This could support the previously described findings that cardioprotective angiotensins are increased in patients with more severe heart failure.

PS 5/5-4

Sacubitril/Valsartan in heart failure with preserved ejection fraction in women and lower-range preserved ejection fraction

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Introduction: Heart failure with preserved ejection fraction (HFpEF) affects half of patients suffering from symptoms of heart failure, yet to date no treatment could achieve a significant reduction in morbidity and mortality. In the recently published PARAGON trial Sacubitril/Valsartan did not reduce overall morbidity and mortality in HFpEF, however analysis of subgroups suggested a potential benefit in female patients and those with lower-range preserved EF (≤57 %).

Methods: A prospective study was initiated treating patients with a diagnosis of HFpEF, fulfilling either criteria–female sex or lower-range preserved EF–with Sacubitril/Valsartan. Patients were up-titrated to the maximum tolerated dose and regularly followed-up regarding potential adverse events necessitating cessation of Sacubitril/Valsartan. After completion of a three-month treatment period the effects of Sacubitril/Valsartan from baseline to follow-up were evaluated. Analysis focused on changes in *N*-terminal prohormone of brain natriuretic peptide (NT-proBNP), 6-minute walking distance (6MWD), New York Heart Association (NYHA) functional class, quality of life

		Sacubitril/Valsartan n=8	p-Value
CARDIAC BIOMARKERS			
NT.proBNP, ng/L	Baseline, median	802,5 (276,5-1669,8)	317,8 0,16
	Follow Up 3 months	(186,6-1545,5)	
	CFB to 3 months, median	-484,7	
FUNCTIONAL STATUS			
6MWT, m	Baseline, mean	335,5±156,0	0,40
	Follow Up 3 months	283,0±105,1	
	CFB to 3 months, mean	-52,3	
NYHA functional class, n (%)			
- improvement	0 (0)		
- stability	8 (100)		
- worsening	0 (0)		
EQ5D-3L	Baseline, mean	8,5±1,8	0,16
	Follow Up 3 months	7,9±1,6	
	CFB to 3 months, mean	-0,6	
Health Score	Baseline, mean	45,7±22,3	0,17
	Follow Up 3 months	60,6±21,8	
	CFB to 3 months, mean	+14,9	
BCM-OH, L	Baseline, mean	0,6±1,2	0,67
	Follow Up 3 months	0,8±0,8	
	CFB to 3 months, mean	+0,2	

BCM-OH = body composition monitoring – overhydration, CFB = Change from baseline, NT-proBNP = N-terminal prohormone of brain natriuretic peptide, 6MWT = six-minute walk test, NYHA = New York Heart Association. Plus–minus values are means ±SD.

Fig. 1 | PS 5/5-4 Changes from baseline

quantified using the EQ5D-3L questionnaire and Body-Composition-Monitoring (BCM).

Results: Starting November 2019, 15 patients commenced treatment with Sacubitril/Valsartan. In course of the study period 5 patients (33.0 %) had to be excluded prematurely due to symptomatic hypotension (4 patients, 26.7 %) or pruritus (1 patient, 6.7 %). Analysis from baseline to follow-up after completion of a 3-month treatment period was performed on 8 patients. A promising decrease of N-terminal prohormone of brain natriuretic peptide (NT-proBNP) (median difference, –484.7 pg/mL, $p = 0.16$) could be observed. 6MWD did not change significantly (mean difference –52.3, $p = 0.40$), neither did overhydration (OH) (+0.2, $p = 0.67$) in body composition monitoring (BCM). Likewise all patients (100 %) reported stability of NYHA functional class. Lastly, interpretation of the EQ5D-3L questionnaire displayed a modest improvement of quality of life (–0.6, $p = 0.16$).

Conclusion: The brief observation of a small patient collective suggested subtle improvements regarding NT-proBNP levels and quality of life. NYHA functional class remained stable during the study period, as did 6MWD and BCM-OH. According to our results female patients and those with lower-range preserved EF displayed stability in response to Sacubitril/Valsartan. Weather a longer study period might procure significant improvement remains a matter open to discussion.

PS 5/5-5

Renin-angiotensin-aldosterone fingerprint in treatment of heart failure with preserved ejection fraction with sacubitril/valsartan

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Introduction: Sacubitril/Valsartan did not reduce overall morbidity and mortality in heart failure with preserved ejection fraction (HFpEF) in the recent PARAGON trial, however analysis of two subgroups–female patients and those with lower-range HFpEF suggested a potential benefit. Analysis of the effect of Sacubitril/Valsartan on the Renin-Angiotensin-Aldosterone system (RAAS) could provide new insight into the yet unrewarding treatment of HFpEF.

Methods: Starting November 2019 a study was initiated treating 5 patients with a diagnosis of HFpEF with Sacubitril/Valsartan, up-titrated to the maximum tolerated dose. Before and after a treatment period of three months RAAS-Fingerprints were performed including the serum concentrations of Angiotensin I (1-10), Angiotensin II (1-8), Angiotensin 1-7, Angiotensin 1-5 and Aldosterone as well as the deducted ratios of Aldosterone/Angiotensin II (AA2R), Plasma-Renin-Activity (ANG I + ANG II) and Angiotensin-Converting-Enzyme Activity (ANG II/ANG I). Moreover the Aldosterone–Renin Ratio (Aldosterone/PRA) and activity of the N-domain of ACE (Ang 1-5/Ang 1-7) were monitored.

Results: Serum concentration of the the observed analytes did not differ significantly from baseline after completion of a three month treatment period. Concerning the deducted ratios, the already low AA2R further decreased ($p = 0.22$). While PRA did not show a significant tendency pertaining an increase or decrease ($p = 0.89$). ARR was reduced ($p = 0.22$). ACE activity was enhanced during the observed period ($p = 0.14$). No increase could be seen for the N-domain of ACE ($p = 0.89$).

Conclusion: The observed decrease in AA2R and ARR can be explained by angiotensin receptor blockade (ARB) via Valsartan. Stability in PRA was expected due to patients not being naïve to ARB treatment at baseline. In contrast the observed increase in ACE activity cannot be explained by the same mechanism. Overall the short treatment period and low patient number were insufficient to procure a level of statistical significance. While showcasing typical changes in RAAS in response to ARB we encountered inexplicable effects that might be caused by additional Neprilysin inhibition.

PS 5/5-6

Comparison of three different methods to measure heart rate in patients with chronic heart failure

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Introduction: Heart rate (HR) control in heart failure (HF) patients is commonly based on single 12-lead ECG-measurement in supine position (ECG-HR). However, mean office heart rate in the sitting position or 24-hours ambulatory heart rate (24 h-HR) measurement may represent more accurate parameters of individual HR than ECG-HR. In a prospective, single-centered study, we investigated associations between ECG-HR, office-HR and 24 h-HR in clinically stable chronic HF patients.

Methods: Patients with stable chronic heart-failure and a left ventricular ejection fraction (LVEF) < 50 % were prospectively enrolled in the outpatient clinic of a tertiary care hospital in Graz, Austria. ECG-HR was measured in a supine position after 5 min of rest. Office-HR rate was measured in a sitting position after 5 min of rest and the mean of three measurements was taken. Mean 24 h-HR was determined using a certified ambulatory blood pressure monitoring device (Mobil-O-Graph, I. E. M. GmbH, Stolberg, Germany). This measurement was subdivided into mean day 24 h-HR and mean nocturnal 24 h-HR measurements.

Results: Ninety-nine patients with a mean age of 64.8 ± 9.6 years were included in the study. 20 % of the study population was female ($n = 20$). Mean LVEF was 35.1 ± 9 % and median NT-proBNP was 964 pg/mL (IQR 335–2172 pg/mL). Comparing the three methods differences were not significant, however ECG-HR tended to be slightly higher than office-HR (71.9 ± 15.9 bpm vs. 68.5 ± 13.3 bpm, $p = 0.088$), mean overall and daytime 24 h-HR (69.8 ± 11.1 bpm, $p = 0.343$ and 70.2 ± 10.9 bpm, $p = 0.5$). 24 h-HR was significantly higher during the day than during the night (70.2 ± 10.9 bpm vs. 64.3 ± 11.4 bpm, $p < 0.001$). ECG-HR did neither correlate with parameters of 24 h-HR measurement ($p > 0.2$ for all) nor with office-HR (r = 0.142, $p = 0.166$). However, there was a significant correlation between office-HR and 24 h-HR measurements (mean 24 h-HR: r = 0.549, $p < 0.001$; daytime 24 h-HR: r = 0.715, $p < 0.001$; nocturnal 24 h-HR: r = 0.477, $p < 0.001$). Interestingly, among patients in sinus rhythm ($n = 44$) with elevated ECG-HR ≥ 70 bpm ($n = 20$), only 6 had elevated office-HR ≥ 70 bpm. There was no significant association

between elevated HR as measured by ECG-HR compared with office-HR using Fisher's exact test ($P = 0.6$). There was no significant correlation between any of the HR measurements and LVEF or NT-proBNP.

Conclusion: Current guidelines suggest HR monitoring and HR control in patients with HF and reduced LVEF. However, the ambulatory heart rate monitoring is only comparable with office heart rate measurement in sitting position in the outpatient clinic and not with ECG-HR measurements. This finding should be investigated further, in order to find the appropriate method of HR measurement to guide HR control in HF patients.

PS 5/5-7

Kardiale ATTR Amyloidose – Eine Fallserie

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Einleitung: Bei der kardialen Amyloidose kommt es zur Ablagerung von fehlgefaltetem Transthyretin im Herzen. Die ATTR Amyloidose tritt mit zunehmendem Alter gehäuft auf und betrifft überwiegend Männer. Das Amyloid kann alle Strukturen des Herzens infiltrieren und im Verlauf zu Herzinsuffizienz (87 %) und Herzrhythmusstörungen (65 %) führen. Die Symptome der Erkrankung sind üblicherweise unspezifisch, was die Diagnosestellung verzögern kann. Es gibt jedoch typische „red flags" (in erster Linie typische Veränderungen im EKG und in der Echokardiographie), die an eine Amyloidose denken lassen und zu einer entsprechenden Abklärung führen sollten. In Studien liegt die Mortalität für Patienten mit ATTR Amyloidose bei über 30 % innerhalb von 2 Jahren.

Methoden: Wir berichten über 12 Patienten, bei denen in der Zeit von Dezember 2018 bis Februar 2020 an unserer Klinik die Diagnose einer kardialen ATTR Amyloidose gestellt werden konnte. Von diesen 12 Patienten waren 11 Männer (91,6 %), das Durchschnittsalter bei Diagnosestellung lag bei 78,8 Jahren (62–85 Jahre). Alle Patienten wiesen eine Herzinsuffizienzsympto-

			Sacubitril/Valsartan n=5	p-Value
ANALYTE CONCENTRATIONS				
Angiotensin II (1-8)	Baseline, median		1077,5±1666,5	0,89
	Follow Up 3 months		1007,2±1187,7	
Angiotensin 1-7	Baseline, mean		43,0±72,6	1,0
	Follow Up 3 months		18,6±25,6	
Angiotensin 1 (1-10)	Baseline, median		376,0±572,9	0,89
	Follow Up 3 months		313,1±407,4	
Angiotensin 1-5	Baseline, median		78,4±110,7	0,89
	Follow Up 3 months		53,3±73,2	
Aldosterone	Baseline, median		468,6±442,6	0,23
	Follow Up 3 months		372,7±281,4	
RAAS Triple-A Evaluation				
AA2R	Baseline, median		3,85±6,5	0,22
	Follow Up 3 months		1,0±1,1	
PRA	Baseline, median		1453,6±2236,9	0,89
	Follow Up 3 months		1320,3±1593,9	
ACE	Baseline, median		3,1±0,7	0,14
	Follow Up 3 months		4,1±1,2	
Other Ratios				
Aldo/PRA	Baseline, median		2,8±4,6	0,22
	Follow Up 3 months		0,8±0,9	
N-ACE	Baseline, median		3,8±3,0	0,89
	Follow Up 3 months		3,3±1,4	

AA2R = Aldosterone/Angiotensin II ratio, ACE = Angiotensin-Converting-Enzyme Activity (ANG II/ANG I), Aldo/PRA = Aldosterone–Renin Ratio , N-ACE = acitivity of the N-domaine of ACE (Ang 1-5/Ang 1-7), PRA = Plasma-Renin-Activity (ANG I + ANG II).

Fig. 1 | PS 5/5-7 Change from baseline

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matik im Stadium NYHA II oder III auf. Eine arterielle Hypertonie war bei keinem der Patienten vordokumentiert.

Resultate: Zwei von 12 Patienten zeigten ein völlig unauffälliges EKG, 6 hatten Vorhofflimmern, bei 3 Patienten zeigte sich ein AV Block Grad I, ein Patient hatte ein Schrittmacher EKG. Der Sokolow-Index für Hypertrophie war bei allen 12 Patienten negativ. Echokardiographisch war bei allen 12 Patienten eine linksventrikuläre Hypertrophie auffällig mit einer mittleren Septumdicke von 19 mm (14–24 mm). Die systolische Pumpfunktion (EF) war bei 5 Patienten normal, bei 4 Patienten geringgradig und bei 3 mäßig bis höhergradig reduziert. Laborchemisch waren sowohl proBNP als auch hs Troponin T bei allen Patienten erhöht, proBNP im Schnitt auf 2890 pg/ml (169–8734 pg/ml, Normwert bis 125 pg/ml), hs Troponin im Schnitt auf 119 ng/l (9–867 ng/l, Normwert bis 5 ng/l), wobei sich bei beiden Werten eine relativ große Bandbreite zeigte. Bei 11 Patienten wurde zur Diagnosestellung eine Knochenszintigraphie durchgeführt (die Sensitivität/Spezifität der Untersuchung liegt in der Literatur bei jeweils ca. 90 % für eine kardiale ATTR Amyloidose), die bei allen Patienten positiv war. Die Diagnose wurde bei 11 Patienten mit Myokardbiopsie gesichert. Alle Patienten erhielten eine Therapie mit Tafamidis, die ohne relevante Nebenwirkungen vertragen wurde. Insgesamt zeigten die meisten Patienten einen stabilen oder leicht progredienten Verlauf. Während des Follow-Ups (durchschnittlich 7,2 Monate, 0–14 Monate) wurde ein Patient wegen kardialer Dekompensation hospitalisiert.

Schlussfolgerungen: Bei einer linksventrikulären Hypertrophie in der Echokardiographie ohne Hypertrophiezeichen im EKG und ohne Anamnese für eine arterielle Hypertonie sollte unabhängig von der systolischen Pumpfunktion vor allem bei älteren männlichen Patienten eine Amyloidose als Differentialdoagnose in Betracht gezogen und die entsprechende Abklärung bezüglich einer ATTR mittels Knochenszintigraphie durchgeführt werden.

PS 5/5-8

Diagnostic and prognostic value of scoring systems in heart failure with preserved ejection fraction

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Introduction: Almost half of all heart failure patients present with a preserved ejection fraction (HFpEF). However, due to concomitant diseases the definite diagnosis of HFpEF is often challenging. Two recent publications tried to assess this issue by presenting scores consisting of clinical and echocardiographic parameters intending to assess the probability of HFpEF diagnosis. [1, 2] We, therefore, aimed to evaluate the diagnostic as well as prognostic value of these new scoring systems.

Methods: This was a prospective observational study performed at a national HFpEF referral center. For the validation part of the study inclusion criteria were left ventricular ejection fraction ≥50 % and invasively measured elevated intracardiac pressures (pulmonary arterial wedge pressure ≥15 mm Hg or left ventricular end-diastolic pressure ≥16 mm Hg). Patients were excluded in case of missing variables needed for the cal-

Abb. 1 | PS 5/5-8 Kaplan Meier curves for event-free survival stratified by median HFA PEFF score (panel A), median H2FPEF score (panel B), and median H2FPEF probability score (panel C)

culation of the H2FPEF or the HFA PEFF score. After validation, the prognostic significance of the scores were tested. The H2FPEF and HFA PEFF scores consist of clinical as well as echocardiographic parameters with points being allocated for each parameter. In case of H2FPEF, the total score correlates with the probability of HFpEF, whereas a HFA PEFF score ≥5 allows for a definite diagnosis of HFpEF.

Results: In total, 427 patients with HFpEF were included into the present study. The H2FPEF and HFA PEFF validation cohorts consisted of 100 (23.4 %) and 162 (37.9 %) patients, respectively. In the total HFpEF cohort median age was 74.0 years [Interquartile range (IQR): 68.0–77.0], median *N*-terminal pro-B-type natriuretic peptide (NT-proBNP) was 1064 pg/mL (IQR: 438–2002), and median LVEF was 59.0 % (IQR: 54.0–65.0). 300 (70.3 %) patients were female and 256 (60.0 %) were in New York Heart Association (NYHA) class ≥III. Median H2FPEF score was 6.0 (IQR: 4.0–7.0), with a respective HFpEF probability of 82.2 % (IQR: 50.1–94.1). Median HFA PEFF score was 4.0 (3.0–5.0). In the validation cohort median H2FPEF score was 6.0 (IQR: 4.0–8.0), with a HFpEF probability of 85.0 % (IQR: 55.4–93.7). In the HFA PEFF validation cohort the median score was 5.0 (3.0–6.0). The scores were not statistically different between the validation and total HFpEF cohorts (*p*:1.000, *p*:1.000, and *p*=0.922). Contrary to the HFA PEFF score, the H2FPEF score and H2FPEF probability score were, predictors of adverse outcome in our univariable Cox regression analysis [hazard ratio (HR): 1.069, 95 % confidence interval (CI): 0.966–

1.182, *p*=0.192; HR: 1.113, 95 % CI: 1.029–1.204, *p*=0.007; HR: 1.016, 95 %CI: 1.009–1.023, *p*<0.001, Fig. 1A-C]. In a multivariable model adjusted for NT-proBNP, NYHA class, age and sex the H2FPEF as well as the H2FPEF probability score remained independently associated with outcome.

Conclusion: In our cohort, the H2FPEF score showed high HFpEF probability values and the majority of patients could have been diagnosed with the HFA PEFF score without further hemodynamic testing. In addition, the H2FPEF scores were independent predictors of outcome. Therefore, these scores seem suitable for detecting, as well as diagnosing patients with HFpEF and in the case of the H2FPEF scores also for risk stratification.

POSTERSITZUNG 6 – VITIEN 1

PS 6/6-1

Prognosis of patients with severe isolated tricuspid regurgitation

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Introduction: A graded relationship of mild vs moderate vs severe tricuspid regurgitation (TR) with survival has been documented in previous large studies. Recently, new classification schemes were suggested introducing additional grades beyond third grade TR. We sought to investigate the prognosis of patients in these additional subgroups.

Methods: In this retrospective observational cohort-study, consecutive patients were analyzed who had been classified at our echo laboratory as severe isolated TR. All patients had normal left ventricular function, none had more than mild-to-moderate regurgitation or stenosis of the mitral and aortic valve. Vena contracta (VC), regurgitant volume (RV), and effective regurgitant orifice area (EROA) of the tricuspid valve were measured. TR was considered severe or massive if (1) EROA ≥0.4 cm2 and ≥0.6 cm2, and (2) if VC ≥7 mm and ≥14 mm, respectively. A multivariate Cox regression model was calculated adjusting for age, sex, creatinine, coronary artery disease, diabetes mellitus, arterial hypertension, and maximal tricuspid regurgitation velocity. One-year and two-year all-cause mortality were retrieved from the national death registry as end-points.

Results: A total of 220 patients were included in the final analysis, median age was 69.5 years (IQR 55–79), 39 % were male. One-year survival was 77 %, 2-year survival was 70.5 %. Median VC was 10 mm (8–12), median EROA was 0.4 cm2 (0.25–0.54), and median regurgitant volume was 44 ml (32–60). Considering (1) EROA classification, 59 patients had severe TR with a 1-year survival of 78 %, and 46 patients had massive TR with a 1-year-survival of 78 %, 2-year survival was 73 % and 70 %, respectively. Considering (2) VC classification, 191 patients had severe TR with a 1-year survival of 79 %, and 29 patients had massive TR with a 1-year-survival of 62 % (*p*=0.046), 2-year survival was 76 % and 58 % (*p*=0.111), respectively. In multivariate regression analysis, only VC as a continuous variable was significantly associated with 2-year mortality with a HR of 1.08 (95 %CI 1.01-1.16, *p*=0.03). Neither the established cutoffs for VC nor for EROA method as categorical variables showed a significant difference in one or two year survival in patients with severe and massive TR.

Conclusion: Classification of significant TR differs tremendously depending on the grading scheme applied. Considering mortality, the VC method differentiates best between the groups. However, with the current cutoffs, in multivariate regression analysis there was no significant difference in survival between severe and massive TR.

PS 6/6-2

Long-term evaluation of the Ross procedure in acute infective endocarditis

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Introduction: There is ongoing debate about the ideal valve substitute in young patients with aortic valve endocarditis. Among various other beneficial characteristics, resistance towards infection is another feature to make the Ross procedure superior compared to other valve replacement techniques. This study puts special focus on analysis of the long-term results of the Ross procedure in patients suffering from acute infective endocarditis.

Methods: Between 1991 and 2017 a total of 191 patients underwent a Ross procedure at our institution. Acute endocarditis was the indication for operation in 19 patients. Completeness of clinical follow up is 100 % with a mean of 12.0 ± 5.7 years.

Results: The mean age of the study cohort was 35.9 ± 11.5 years. Moderate or severe aortic regurgitation (grade III–IV) was present in 84.2 % of patients. Systemic embolization had occurred in 36.8 % of patients. In all patients the pulmonary autograft was implanted as free-standing root replacement with a mean aortic cross clamping time of 126 ± 24 min. Early mortality at 30-days was 5.3 % (one patient). The median stay on the intensive care unit was 1 day. All patients were discharged with no or trivial aortic regurgitation (grade 0–I). The mean length of hospital stay was 19.3 ± 8.7 days. No case of recurrent endocarditis affecting the autograft occurred. In one drug-addicted patient the homograft was replaced 1.8 years after the Ross procedure for recurrent endocarditis. Aneurysmal autograft dilatation lead to reoperation in three patients late after the initial procedure.

Conclusion: The Ross procedure can be performed with low morbidity and mortality, an excellent valve function and a low recurrence rate in selected young patients with infective endocarditis. Reoperations for autograft dilatation remain a major concern after implantation of the autograft as free-standing root replacement.

PS 6/6-3

Natural course of non-severe secondary tricuspid regurgitation–insights from quantitative Doppler assessment

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Introduction: sTR is a frequent finding in patients with heart failure with reduced ejection fraction (HFrEF) and is associated with adverse outcome despite guideline directed therapy (GDT). However, little is known about the natural course of non-severe sTR and its relation to cardiac remodeling and outcome. This study sought (i) to investigate the natural course of secondary tricuspid regurgitation (sTR) progression and regression by quantitative measurements, (ii) to assess the prognostic impact on long-term mortality and (iii) to identify morphologic and functional risk factors associated with sTR progression.

Methods: We included 216 patients with HFrEF under GDT and quantitatively defined sTR progression as well as regression.

Results: Among patients with non-severe TR at baseline, 62 patients (32 %) experienced sTR progression. Progressive sTR was accompanied by larger left and right atrial volumes ($P = 0.022$ and $P = 0.019$ respectively) and a higher prevalence of atrial fibrillation ($P = 0.039$). During a median follow-up of 60 months (IQR 37–60), 82 patients died. Progression of sTR conveyed an increased risk of long-term mortality (HR 1.80, 95 % CI 1.1–2.83; $P = 0.018$), even after multivariate adjustment for a bootstrap-selected (adj. HR 1.70, 95 % CI 1.06–2.74; $P = 0.028$) and clinical confounder model (adj. HR 1.80, 95 % CI 1.07–3.05; $P = 0.027$). In contrast, regression of sTR was observed in 6 % of patients and was not associated with improved long-term survival (HR 1.62, 95 % CI 0.64–4.11; $P = 0.311$).
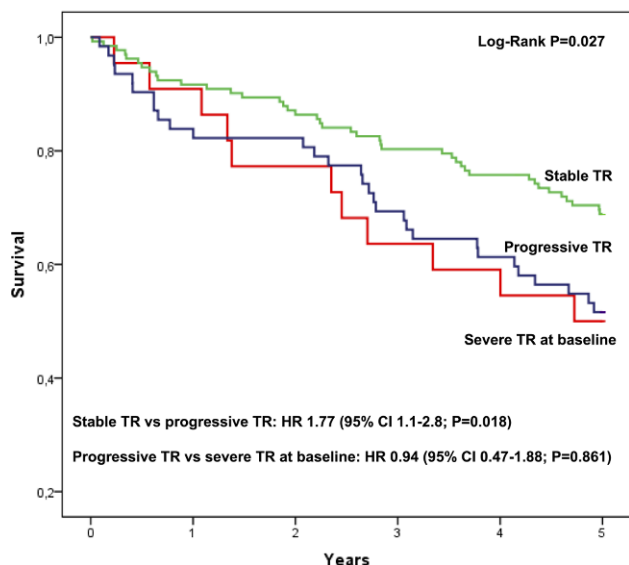
Abb. 1 | PS 6/6-3

Conclusion: A third of patients with non-severe sTR at baseline develop subsequent sTR progression despite GDT. This drives adverse cardiac and valvular remodelling and is associated with a significant increase in long-term mortality. Bi-atrial enlargement as well as atrial fibrillation are determinants for the development of subsequent progressive sTR and may help to identify patients at risk for sTR progression, potentially creating a window of opportunity for closer follow-up and newly arising minimal invasive transcatheter repair therapies.

PS 6/6-4

Right ventricular strain predicts survial in patients with tricuspid regurgitation

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Introduction: Tricuspid regurgitation (TR) is a frequent finding in echocardiography. Significant reflux may be well tolerated for years. However, recent data indicates that right ventricular (RV) function is crucial in these patients and associated with poor prognosis. This study aimed to evaluate the prevalence of RV dysfunction and its prognostic value in patients with different degrees of TR.

Methods: In this retrospective observational cohort study, data from 1,656 consecutive patients (56.9 % male) undergoing echocardiography at a tertiary care center from 2010 to 2016 was analyzed. Tricuspid annular plain systolic excursion (TAPSE <17 mm), right ventricular free lateral wall strain (RV strain > –23 %), and fractional area change (FAC <35 %) were used to evaluate RV dysfunction. Kaplan-Meier analysis was performed to estimate mortality and Cox-regression to identify predictors for long-term survival.

Table 1 | PS 6/6-4

	Survivor	Non-survivor	*p*-value
TAPSE [mm]	20.41 ± 5.26	18.73 ± 4.82	< 0.001*
RV free lateral strain [%]	−21.30 ± 8.01	−17.33 ± 7.64	< 0.001*
FAC [%]	38.18 ± 19.69	34.33 ± 14.10	0.003*

Students' t-test for differences in right ventricular function regarding survivor status. TAPSE = tricuspid annular plain systolic excursion, RV = right ventricular, FAC = fractional area change. * p-value ≤ 0.05 was considered significant.

Results: 234 (14.1 %) patients showed no, 1,055 (63.7 %) mild, 290 (17.5 %) moderate, and 77 (4.6 %) severe TR. The number of patients with RV dysfunction varied according to the different echocardiographic approaches: TAPSE: N = 408/1,511 (27.0 %), RV strain: N = 633/992 (63.8 %), and FAC: N = 571/1,303 (43.8 %). Survivors showed significantly better RV function compared to non-survivors (Table 1). Kaplan-Meier analysis revealed significantly worse survival in patients with RV strain > –23 % in patients overall (Fig. 1A) as well as in subgroups consisting of patients with TR grade ≤ I and ≥ II (Fig. 1B and C). RV strain, but not TAPSE or FAC, was independently associated with all-cause mortality (Table 2).

Conclusion: Prevalence of RV dysfunction varies according to echocardiographic parameter used. In patients with different degrees of TR, RV dysfunction assessed by RV strain is independently associated with worse survival.

Fig. 1 | PS 6/6-4 Kaplan-Meier analysis according to right ventricular free wall strain (RV strain) > − 23 % and ≤ –23 % in **a)** all patients (Log rank, *p* < 0.001; *N* = 991), **b)** patients with tricuspid regurgitation (TR) ≤ I (Log rank, *p* = 0.001, N = 780) and **c)** patients with TR ≥ II (*p* = 0.046, N = 210). Impaired RV function is associated with an increased mortality

Table 2 | PS 6/6-4

Variable		Hazard ratio	Confidence interval		p-value
TAPSE		0.975	0.942	1.009	0.142
RV free lateral strain		1.033	1.010	1.055	0.005*
FAC		1.000	0.989	1.011	0.999
Age		1.023	1.004	1.042	0.016
Type II diabetes		1.417	1.034	1.942	0.030*
CKD					0.024*
	eGFR 30–60 ml/min	2.035	1.001	4.135	0.050
	eGFR < 30 ml/min	3.155	1.082	9.198	0.035*
Hypertension		1.143	0.557	2.343	0.716
COPD		1.101	0.760	1.593	0.612
TR Vmax		0.999	0.961	1.037	0.942
CAD					0.282
	one vessel disease	0.581	0.337	1.002	0.581
	two vessel disease	0.773	0.430	1.391	0.390
	three vessel disease	0.763	0.477	1.220	0.258
Atrial fibrillation					0.128
	paroxysmal	1.444	0.931	2.241	0.101
	persistent	1.065	0.601	1.889	0.829
	permanent	1.504	1.025	2.206	0.037(*)

Cox-Regression including cardiac and non-cardiac risk factors in patients with different grades of TR ≥ II. TAPSE = tricuspid annular plain systolic excursion, RV = right ventricular, FAC = fractional area change, CKD = chronic kidney disease, eGFR = estimated glomerular filtration rate, COPD = chronic obstructive pulmonary disease, TR = tricuspid regurgitation, CAD = coronary artery disease. * a p-value ≤ 0.05 was considered significant (N = 618).

PS 6/6-5

Echocardiographic parameters of right ventricular function in patients with isolated severe tricuspid regurgitation

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Introduction: In patients with severe tricuspid regurgitation (TR), gradation of right ventricular function (RVF) by echocardiography is often impeded through misleading ventricular motion caused by the pendulous volume. In our cohort of patients with isolated TR, we sought to analyze the prognostic value of traditional and new echocardiographic measures of RVF.

Methods: In this retrospective observational cohort study, consecutive patients were analyzed who had been classified as severe isolated TR. All patients had normal left ventricular function, none had more than mild-to-moderate left-sided valve disease. Vena contracta (VC) was measured and only patients with VC ≥ 7 mm were included in the final analysis. Tricuspid annular plane systolic excursion (TAPSE), fractional area change (FAC), and right ventricular free lateral wall longitudinal strain (RVFW-LS) were analyzed as continuous variables. Two-year all-cause mortality was retrieved from the national death registry as end-point. Multivariate Cox regression analysis was calculated adjusting for age, sex, creatinine, maximal tricuspid regurgitation velocity, presence of coronary artery disease, diabetes mellitus, and arterial hypertension.

Results: A total of 220 patients were included in the final analysis, median age was 69.5 years (IQR 55–79), 39 % were male. Two-year survival was 70.5 %. Median TAPSE was 19 mm (15–22), median FAC 42 % (30–52), and median RVFW-LS –20 % (–11 to –27). TAPSE (HR 0.94, 95 %CI 0.88–0.99, $p = 0.035$), FAC (HR 0.97, 95 %CI 0.94–0.99, $p = 0.015$), and RVFW-LS (HR 0.95, 95 %CI 0.89–0.99, $p = 0.046$) were significant predictors for all-cause 2-year-mortality in the multivariate regression model.

Conclusion: TAPSE, RVFW-LS, and FAC predict 2-year-mortality even in the presence of severe pendulous volume through isolated TR.

PS 6/6-6

Resting echo parameters in classical low-flow, low-gradient aortic stenosis for prediction of a true severe subform

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Introduction: This study sought to assess the diagnostic accuracy of resting peak aortic jet velocity (Vmax) and mean transvalvular gradient (MPG) to predict true hemodynamic severity in patients with classical low-flow, low-gradient (LF/LG) aortic stenosis (AS). Dobutamine stress echocardiography (DSE) helps to differentiate true-severe LF/LG AS from pseudo-severe LF/LG AS in patients with classical LF/LG AS.

Vmax ≥4 m/s or mean transvalvular gradient MPG ≥40 mm Hg are regarded indicative for true-severity during DSE. However, borderline values of resting Vmax and MPG below these cut-offs may be representative of true-severity.

Methods: Ninety-two patients with classical LF/LG AS who underwent subclassification using DSE were studied. For various cut-points of rest Vmax and MPG, sensitivity/specificity values were calculated and receiver operating curves (ROC) constructed.

Results: Vmax showed a ROC-AUC of 0.716 ($p < 0.05$) and MPG showed a ROC-AUC of 0.699 ($p < 0.05$). The cut-offs >3.5 m/s for Vmax and >35 mm Hg for MPG yielded a positive predictive value (PPV) of 100 % irrespectively of the presence of contractile reserve.

Conclusion: The presence of a rest Vmax >3.5 m/s or a rest MPG >35 mm Hg is likely to reflect true hemodynamic severity and might be sufficient to diagnose TS LF/LG.

POSTERSITZUNG 7 – RISIKOFAKTOREN/STOFFWECHSEL/ LIPIDE 1

PS 7/7-1

Hand grip strength and type 2 diabetes are mutually independent predictors of cardiovascular events and of mortality in patients with established cardiovascular disease

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Introduction: Impaired skeletal muscle function is involved in the pathogenesis of type 2 diabetes (T2 DM) as well as of cardiovascular disease (CVD). Hand grip strength (HGS) is a simple, inexpensive and non-invasive diagnostic parameter for muscle function. Its association with cardiovascular events and mortality in patients with established CVD is unclear and is addressed in the present study.

Methods: We prospectively recorded cardiovascular events over a mean follow up time of 8.9 ± 3.2 years in a cohort of 876 patients with established CVD (672 patients with angiographi-

cally proven coronary artery disease and 204 patients with sonographically proven peripheral artery disease).

Results: From our patients, 274 (31.3 %) had T2 DM at baseline; HGS did not differ significantly between patients with T2 DM and non-diabetic subjects (35.3 ± 11.9 vs. 36.6 ± 11.9 kg; $p = 0.082$). Prospectively, cardiovascular events occurred in 387 patients, and 278 patients died during follow-up. T2 DM and low HGS after multivariate adjustment in Cox regression models proved to be mutually independent predictors of cardiovascular events (adjusted HR 1.52 [1.23–1.89]; $p < 0.001$ and standardized adjusted HR 0.85 [0.74–0.99]; $p = 0.032$, respectively) as well as mortality (adjusted HR 1.79 [1.39–2.29]; $p < 0.001$ and standardized adjusted HR 0.69 [0.58–0.83]; $p < 0.001$, respectively).

Conclusion: We conclude that low HGS and T2 DM are mutually independent predictors of cardiovascular events as well as of mortality in patients with established CVD.

PS 7/7-2

Comparison of two recent ceramide-based coronary risk prediction scores: CERT and CERT-2

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Introduction: The Coronary Event Risk Test (CERT) is a validated cardiovascular risk predictor that uses circulating ceramide concentrations to allocate patients into one of four risk categories. This test has recently been updated (CERT-2), now additionally including phosphatidylcholine concentrations.

Methods: We investigated the power of CERT and CERT-2 to predict cardiovascular mortality in 999 patients with cardiovascular disease (CVD).

Results: Overall, comparing survival curves (figure) for over 12 years of follow up and the predictive power of survival models using net reclassification improvement (NRI), CERT-2 was the best predictor of cardiovascular mortality, surpassing CERT (NRI = 0.456; $p = 0.01$) and also the 2019 ESC-SCORE

Abb. 1 | PS 7/7-2 Cardiocascular survival of CVD patients with respect to CERT, CERT-2 and the ESC-SCORE
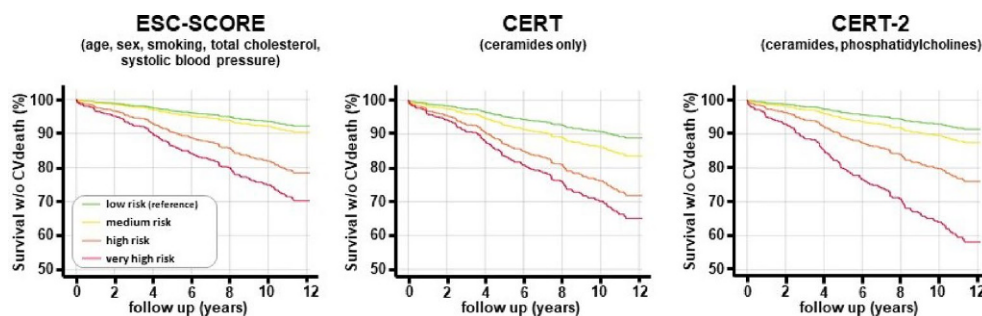
Cardiovascular (CV) survival is shown according to ESC-SCORE categories and according to the four risk categories of the ceramide-based Coronary Event Risk Test (CERT) and of CERT-2. ESC-SCORE categories were built according to current ESC/EAS guidelines, using SCORE charts for low risk countries, which is based on age, sex, smoking, total cholesterol, and systolic blood pressure.

(NRI = 0.163; p = 0.04). Patients in the highest risk category of CERT as compared to the lowest category had a HR of 3.63 [2.09–6.30] for cardiovascular death; for CERT-2 the corresponding HR was 6.02 [2.47–14.64]. Among patients with T2 DM (n = 322), the HR for cardiovascular death was 3.00 [1.44–6.23] using CERT and 7.06 [1.64–30.50] using CERT 2; the corresponding HRs among non-diabetic subjects were 2.99 [1.20–7.46] and 3.43 [1.03–11.43], respectively.

Conclusion: We conclude that both, CERT and CERT-2 scores are powerful predictors of cardiovascular mortality in CVD patients, especially in those patients with T2D. Performance is even higher with CERT-2.

PS 7/7-3

Value of total cholesterol earlier versus later in life to predict cardiovascular death

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Introduction: Prognostic implications of blood cholesterol may differ at different stages of life. This study compares the value of total cholesterol (TC) earlier versus later in life to predict coronary atherosclerosis and fatal as well as non-fatal cardiovascular events.

Methods: In a cardiovascular observation study (CVOS) we performed coronary angiography and prospectively recorded cardiovascular events in 1090 patients over up to 19 years. These patients had participated in a health survey 15 years prior to the CVOS baseline. TC was measured both at the health survey and at the baseline of the CVOS and categorized into four groups, according to SCORE charts of the current ESC/EAS guidelines.

Results: Patients in the highest versus the lowest TC-category of the health survey had an OR of 4.38 [2.46–7.81]; p = 0.001 for significant CAD at angiography, a HR of 1.80 [1.13–2.85]; p = 0.013 for cardiovascular events, and a HR of 8.03 [1.11–57.98]; p = 0.039 for cardiovascular death after multivariate adjustment. In contrast, TC as measured at the baseline of the CVOS was neither significantly associated with significant CAD nor with cardiovascular events or death during follow-up. In addition, the ESC/EAS-SCORE was found to be more powerful in predicting cardiovascular death when using earlier instead of later TC, with a continuous net reclassification improvement of 0.301 (p < 0.001).

Conclusion: We conclude that TC assessed earlier in life is a better predictor of cardiovascular risk than TC assessed later in life, over and above other cardiovascular risk factors, enabling earlier therapy of patients at risk.

PS 7/7-4

Type 2 diabetes, congestive heart failure and non-alcoholic fatty liver disease

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Introduction: Non-Alcoholic fatty liver disease (NAFLD) is associated with both type 2 diabetes (T2 DM) and congestive heart failure (CHF). T2 DM is highly prevalent in CHF patients; however, the single and joint associations of T2 DM and CHF with NAFLD have not been investigated yet. This issue therefore is addressed in the present study.

Methods: We investigated 202 patients with CHF and 670 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 47.0 % in CHF patients and 22.1 % in controls (p < 0.001). FLI and prevalence of NAFLD (FLI ≥60) in non-CHF subjects without T2 DM were 49 ± 28 and 38.6 %, respectively. They were significantly higher in non-CHF T2 DM patients (70 ± 25, p < 0.001 and 68.5 %, p < 0.001, respectively), in CHF patients without T2 DM (63 ± 23, p < 0.001 and 58.6 %, p < 0.001, respectively) and in CHF patients with T2 DM (73 ± 24, p < 0.001 and 78.0 %, 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 (F = 21.47; p < 0.001 and F = 53.92; 0 < 0.001, respectively); concordantly, T2 DM and CHF independently predicted the presence of NAFLD in logistic regression analyses, with adjusted odds ratios of 2.49 [1.55–3.99]; p < 0.001 and 6.97 [3.95–12.29]; p < 0.001, respectively.

Conclusion: We conclude that T2 DM and CHF are mutually independent predictors of NAFLD.

PS 7/7-5

Effects of exercise training on lipid accumulation product and matrix metalloproteinase 9 in patients with small abdominal aortic aneurysms

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Introduction: Currently, no medical therapy is effective in limiting progression of small abdominal aortic aneurysms (AAA; ≤ 5.5 cm). Previously, we have demonstrated the safety and efficacy of exercise training in patients with AAA. We now assessed possible associations between AAA diameter, lipid accumulation product (LAP) and matrix metalloproteinase 9 (MMP-9). Whereas LAP has been shown in previous studies to outperform body mass index (BMI) for identifying US adults at cardiovascular risk, MMP-9 has been linked to destruction of the aortic matrix.

Methods: 140 patients with AAA ≤ 5.5 cm were included in this randomized trial and complete data of ninety-six patients (male $n = 87$, female $n = 9$; exercise training (EX) $n = 42$, usual care (UC) $n = 54$) were available for analysis. EX performed in-hospital and/or home-based endurance and resistance training (3×55min/week) for one year. Statistical analyses of in-group and between-group changes of AAA diameter, parameters of exercise capacity, LAP (LAPmen = (waist circumference (WC)—65) × fasting triglycerides (TG); LAPwomen = (WC—58) × TG) and MMP-9 were performed.

Results: During maximal ergometry EX showed a significant increase in maximal exercise time and estimated metabolic equivalent of task (METs). LAP decreased in EX and increased in UC (both $p = 0.004$); MMP-9 remained statistically unchanged in EX, but increased significantly in UC ($p = 0.005$), resulting in a trend towards a difference between groups ($p = 0.094$).

Conclusion: This is the first study to demonstrate that exercise training in AAA beneficially modifies LAP and MMP-9, both markers of vascular disease, without inducing aneurysmal growth beyond what is otherwise observed during usual care.

PS 7/7-6

Correlation of food and alcohol consumption with peripheral atherosclerotic plaque volume measured by 3D-volumetry

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Introduction: Cardiovascular diseases (CVD) represent the leading cause of death in western countries. Food patterns influence the risk for CVD and a healthy nutrition is essential for the prevention of CVD. Light to moderate alcohol consumption (1–2 units per day) reduces cardiovascular risk showing a U-or J-shaped relation between alcohol consumption and CVD. The aim of this study was to determine the influence of nutrition and alcohol consumption on peripheral atherosclerotic plaque volume measured by 3D-ultrasound technique.

Methods: In this prospective, single centre study, we included 339 patients (median age 66; 57.9 % men) with at least one CVRF or established cardiovascular disease. Plaque volume was measured using an automated software. Information's on food and alcohol consumption of the participants were collected using an internationally acknowledged standardized questionnaire, named DEGS1 Statistical analyses were performed using SPSS Statistic (version 24.0).

Results: Patients with low total plaque volume consumed significantly ($p < 0.05$) more vegetables and vegetable juice per day. Instead, patients with higher total plaque volume had

reported a higher alcohol consumption compared to patients with lower total plaque volume, with borderline statistical significance ($p = 0.044$). Patients without cardiovascular diseases reported a significantly higher fish consumption per day ($p = 0.03$) compared to patient with known cardiovascular disease. This association was also observed for the subgroup of peripheral arterial disease

Conclusion: In our study, patients with more vegetables and vegetable juice consumption per day had significant less plaque volume. Instead, patients with higher alcohol consumption had also a higher total plaque volume. To our knowledge, this is the first study investigating the association of food consumption with peripheral atherosclerotic plaque volume measured by 3D-ultrasonography.

COVID-19 UND HERZ 1 POSTERSITZUNG

CO-1-1

Comparison of number and clinical outcome of acute coronary syndromes before, during and after the lockdown in the COVID-19 Pandemic

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Introduction: COVID-19 has affected care of patients with acute coronary syndromes (ACS) worldwide. The majority of analyses compared the lockdown phase during the pandemic with pre-pandemic times and less is known about the post-lockdown phase when restrictions were loosen. It was aim of this analysis to compare the number as well the clinical outcome of ACS-patients admitted to our hospital pre, during and post lockdown due to the pandemic.

Methods: We analysed consecutive patients presenting with ACS during January to June 2020 and compared these with patients from the same time period in 2019, focusing on 6 weeks of the pre (February 3rd to March 15th), during (March16th to April 26th) and post (April 27th to June 7th) lockdown times, respectively. We recorded the patient number, type of ACS of patients, patient characteristics, door-to-balloon time (for STEMI only), hospitalization length, and clinical outcome, including MACE rate (in-hospital mortality, admission to ICU), peak levels of myocyte necrosis markers, BNP and peri-procedural cardiogenic shock symptoms.

Results: Patient numbers: A total of 96 patients presented to our institution with an ACS during the pre, during and post lockdown phase in 2020, compared to 86 cases in the corresponding time period in 2019. During the official COVID-19 associated lockdown phase a total of 28 patients were admitted with an ACS (11 STEMIs and 17 NSTEMIs), which is slightly reduced compared to the 31 patients admitted in the corresponding time period in 2019 (16 STEMIs and 15 NSTEMIs). However, a striking difference in ACS cases was observed in the post-lock down phase: In 2020 37 patients were admitted with an ACS, compared to only 23 in the same time period in 2019, largely driven by a significantly higher number of NSTEMIs during the post-lock down phase (23 vs. 7 NSTEMIs 2020 vs. 2019; $p = 0.017$) and a significantly reduced number of STEMIs in relation to total ACS admissions (69.5 % STEMIs in 2020 vs. 37.8 % in 2019; $p = 0.018$) Patient characteristics: Significantly more men were admitted to our hospital in the lockdown phase 2020 (81 % males) com-

pared to 2019 (66 % males; $p = 0.002$). Regarding the patients age at admission, there were no significant differences between all phases of 2019 and 2020. Significantly more ACS patients were admitted 2020 with a history of coronary artery disease (53.5 % vs. 22.5 %; $p = 0.014$) or previous MI (43 % vs. 19 %; $p = 0.05$). No statistical difference in the severity of coronary artery disease was evident between pre- and post- lockdown intervals and the respective data 2019

Conclusion: Clinical outcome: There was no statistically significant difference between peak values of markers of myocyte necrosis (Troponin, CK, CK-MB), BNP or MACE rate (including in-hospital mortality) for all time intervals. Admission to ICU was non-statistically higher in the lockdown phase 2020 compared to pre- and post-lockdown intervals and also higher compared to the same time span in 2019. Furthermore, no difference was found in STEMIs with respect to first medical contact-to-balloon times, respectively. Hospitalization length was similar for all phases 2020 and comparable to 2019. There was also no significant difference in patients who were in peri-procedural cardiogenic shock between all compared phases of 2019 and 2020 Conclusion: In our hands the lockdown during the COVID-19 pandemic led to a reduction of ACS patients (mainly STEMI) admitted to our hospital compared with the same time interval 2019 and to the pre-and post-lockdown phases 2020. Moreover, there was a trend to more severe ACS cases during the lockdown phase 2020. Interestingly, we fonud a "rebound" of NSTEMI patients admitted in the post-lockdown

phase 2020, possibly indicating that CAD patients tried to avoid hospital admission during the lockdown phase. Finally. clinical outcome data were comparable between years and the investigated time intervals within years, which might be explained to some extent by the low total number of ACS patients admitted to our department.

CO-1-1

Vergleich der Katheterzahlen für chronische Patienten und akutes Koronarsyndrom (STEMI/NSTEMI) im Zeitraum Anfang März bis Ende März 2020 in Relation zu den entsprechenden Zahlen aus dem Vergleichszeitraum des Vorjahres.

G. Lamm[1], J. Auer[2], R. Berger[3], G. Christ[4], G. Delle Karth[5], M. Frick[6], A. Geppert[7], K. Huber[8], M. Grund[9], M. Haas[10], U. Hoppe[11], L. Kaltenbach[12], K. Kaspar[13], S. Papai[14], H. Ulmer[12], V. Mühlberger[15]

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Tab. 1 | CO-1-1

Anzahl		Phase 1-2019		Phase 2-2019		Phase 3-2019		Phase 1-2020 (Pre-lockdown)		Phase 2-2020 (Lockdown)		Phase 3-2020 (Post-lockdown)
		Mittel-wert	Anzahl	Mittel-wert	Anzahl	Mittel-wert	Anzahl	Mittel-wert	Anzahl	Mittel-wert	Anzahl	Mittel-wert
ACS type	NSTEMI	14		15		7		13		17		23
	STEMI	18		16		16		18		11		14
	Total	32		31		23		31		28		37
Hospitalization length (days)			7		5		11		7		6	6
Vessel disease (n)	0	0		2		0		0		0		2
	1	10		12		7		15		11		11
	2	8		5		5		7		3		9
	3	9		8		11		7		13		13
	1+LM	0		0		0		1		0		0
	2+LM	1		2		0		0		0		0
	3+LM	4		2		0		1		1		2
Peri-procedural cardiogenic shock (n)		2		2		3		0		2		3
MACE rate (n)		3		4		8		4		6		7
In-hospital death (%)		6,3%		3,2%		8,7%		3,2%		3,6%		8,1%
Admission to ICU (%)		3,1%		9,7%		26,1%		9,7%		17,9%		10,8%
Troponin hs peak (ng/L)			27.775		36.138		117.222		30.927		46.048	61.607
CK peak (U/L)			970		926		1372		1095		1213	1251
CK-MB peak (U/L)			146		147		160		179		174	161
BNP peak (ng/L)			7049		3029		8685		6541		10.284	4892
FMC-balloon time (STEMI only) (hrs)			2:22		2:02		11:43		2:58		1:53	2:17

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Einleitung: Die COVID-19 Pandemie hat weltweit zu einer Reduktion der Hospitalisierungsrate an akuten Koronarsyndromen geführt. Die entsprechenden Daten aus Österreich sind in diesem Zusammenhang von Interesse.

Methoden: In einer Blitzumfrage aus dem Austrian National Cath-Lab Registry (ANCALAR) wurden die Herzkatheterzahlen von 11 österreichischen Zentren vom März 2020 mit Daten aus dem März des Vorjahres verglichen.

Resultate: Die Auswertung der 11 teilnehmenden Herzkatheterzentren vom März 2020 ergab im Gegensatz zum Vergleichszeitraum des Vorjahres eine Abnahme sowohl der diagnostischen Koronarangiografien (CAG) um 26 % ($p < 0{,}001$) als auch der perkutanen Koronarinterventionen (PCIs) um 22 % ($p < 0{,}001$), wobei die Anzahl der elektiven PCIs um 29 % ($p < 0{,}001$) und der akuten PCIs um 14 % ($p = 0{,}029$) zurückgingen. Die Abnahme der STEMI-PCIs betrug 18 %, der NSTEMI-PCIs 15 %. Hierbei wurden regionale Unterschiede beobachtet: In 5 Zentren war kein Rückgang der STEMI-PCIs zu verzeichnen, während 6 Zentren eine Abnahme um 31,3 % ($p < 0{,}01$) berichteten, darunter 2 Zentren mit einer >50 % Reduktion der STEMI-PCIs. Das Verhältnis PCI/CAG verschob sich mit 52/50 % (mean/median) im Vergleich zu 2019 (48/49 %) zugunsten der PCIs, d. h. es wurden insgesamt mehr therapeutische Eingriffe durchgeführt.

Schlussfolgerungen: Wie international beobachtet kam es auch österreichweit zu Beginn der COVID-19 Pandemie zu einem deutlichen Rückgang von Herzkathetereingriffen, wobei interessanterweise nicht nur die Zahl der elektiv durchgeführten Eingriffe sondern auch die Anzahl der PCIs bei akuten Koronarsyndromen deutlich abnahm, wobei die Ursache hierfür vielschichtig sein dürfte.

CO-1-3

COVID-19 treatment by anti-coagulation: anti-thrombotic and anti-inflammatory?

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Introduction: In comparison to influenza, an infection with the newly emerged coronavirus SARS-CoV-2 (severe acute respiratory syndrome coronavirus-2) is associated with a higher risk of mortality. This is mainly due to the high susceptibility for secondary diseases, such as the acute respiratory distress syndrome (ARDS). In turn, ARDS is characterized by an acute inflammation and an excessive activity of the coagulation system, leading to an increased vulnerability for thromboembolic events. Previous studies have already outlined that heparin has the potential to lower the SARS-CoV-2 infection rates by 50 % in vitro and to significantly reduce the rate of mortality in vivo.

Methods: As a consequence thereof, we suggested a link between the coagulation pathway and the inflammatory response. In order to analyse this issue, human peripheral mononuclear blood cells (PBMCs) from healthy volunteers were isolated and treated with serum, heparinized plasma, EDTA plasma and different doses of fibrin, for 24 h. Thereafter, the pro-inflammatory cytokine and chemokine levels in the secretome were measured using enzyme-linked immunosorbent assay (ELISA).

Results: Our results outline that serum has the greatest impact on the secretion of cytokines and chemokines measured resulting in a significant increase of their supernatant levels within 24 h. Furthermore, the coagulation factor fibrin markedly rises the cytokine and chemokine supernatant concentrations in a dose-dependent manner.

Conclusion: Therefore, in accordance with previous studies, our results indicate that anti-coagulation may serve as a promising tool for the treatment of severe COVID-19 infections, reducing both, the cytokine storm, as well as the risk for thromboembolic events, such as pulmonary embolism.

CO-1-4

The impact of the global COVID-19 pandemic on incidences of atrial fibrillation and electrical cardioversion at a tertiary care Emergency Department: An inter- and intra-year analysis

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Introduction: As recommended by international organizations, national authorities have introduced measures against further spreading of COVID-19, such as lockdowns and isolation. It was shown that for multiple non-COVID-19 diseases, documented incidences have dropped, and treatment timeframes were stretched. Yet, detailed data on the workload dynamics concerning atrial fibrillation and electrical cardioversion whilst a national lockdown are scarce, and may be helpful for future planning.

Methods: All documented cases of atrial fibrillation and all respective electrical cardioversion episodes at the Emergency Department of the Medical University of Vienna, Austria, within 01/01/2020 and 31/05/2020 were assessed. As reference groups, those incidences were also calculated from the years of 2017,

2018 and 2019. Inter- and intra-year analyses were conducted through Chi-square test and Poisson regression.

Results: A total of 2310 atrial fibrillation-, and 511 electrical cardioversion episodes were included. We found no significant differences in the respective incidences in inter-year analyses of the overall time periods from January to May, or in those of the weeks pre- and post the national lockdown due to SARS-CoV-2 pandemic. However, an intra-year analysis of the year 2020 showed a trend towards a decrease in atrial fibrillation incidences (rate ratio 0.982, CI 0.964–1.001, $p = 0.060$), and a significant increase of electrical cardioversion incidences towards the post-lockdown period (rate ratio 1.051, CI 1.008–10.96, $p = 0.020$).

Conclusion: The decreased atrial fibrillation incidences are in line with international data. However, an increased demand of electrical cardioversions during the lockdown period was observed. A higher threshold of patients to seek medical attention may result in a subsequently selected group with potentially more severe clinical courses. In addition, lifestyle modifications during isolation and a higher stress level may promote atrial fibrillation episodes to be refractory to other therapeutic approaches than electrical cardioversion.

CO-1-5

Pacemaker implantation rates during COVID-19

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Introduction: Studies showed significantly reduced rates of patients presenting with acute coronary syndrome during the COVID-19 crisis. However, there is only limited data showing the trend of pacemaker implantations.

Methods: We evaluated the weekly rates of pacemaker implantation at our centre during the national lockdown for COVID-19 at the between 16 March 2020 and 29 April 2020 (weeks 12–17/2020), compared to the implantation rates 6 weeks before (weeks 6–11/2020), 6 weeks afterwards (weeks 18–23/2020), and the same time frame in 2017–2019. To reduce bias due to postponed planned procedures, we stratified pacemaker implantations into the following groups: total implantations (including box changes), new pacemaker implantations, implantation due to AV block, implantation due to supraventricular conduction disturbances, and other implantations.

Results: The total number of total weekly implantations was reduced from 10.7 (weeks 6–11/2020) to 4.2 (weeks 12–17/2020; –60.1 %, $p = 0.02$). We found no significant reduction in the same time frame in 2017–2019 (6.5 vs. 6.1 per week, $p = 0.29$). We found a similar effect in "new" pacemaker implantations (8.5 vs. 3.2 per week, –62.7 %, $p = 0.02$) and AV block (5.0 vs. 1.5 per week, –70 %, $p = 0.03$). There was no reduction in pacemaker implantation due to sick sinus syndrome (2.5 vs. 0.8 per week, –66.7 %, $p = 0.12$) and other indications (1.0 vs. 0.8 per week, –16.7 %, $p = 0.86$). In the six following weeks (18–23/2020), the total numbers (6.0 per week) and indications other than AV block rose to baseline ($p > 0.05$), but patients with AV block were still less prevalent (1.7 per week, $p = 0.04$).

Conclusion: The reduction of total and new pacemaker implantations during the COVID-19 lockdown was mainly based on a reduced pacemaker implantation rate for AV block. This effect persisted even after the national lockdown. This analysis implies that a significant number of patients with AV block may have avoided medical contact during and after the lockdown and therefore may have experienced increased mortality.

CO-1-6

Impact of COVID-19 on ablation numbers in the Austrian Ablation Registry

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Introduction: Coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has emerged as a pandemic crisis and poses major challenges to health care systems globally. COVID-19 is related to significant morbidity and mortality but has also been linked with a decrease in emergency and total hospital admissions, i. e. for acute coronary syndromes or stroke. The impact of this "collateral" damage of COVID-19 on outcomes is not yet foreseeable and may also impact patients with arrhythmias. Objective: We assessed the impact of COVID-19 on admission rates for elective ablations as well as emergent ablations for ventricular tachycardia (VT) in Austria.
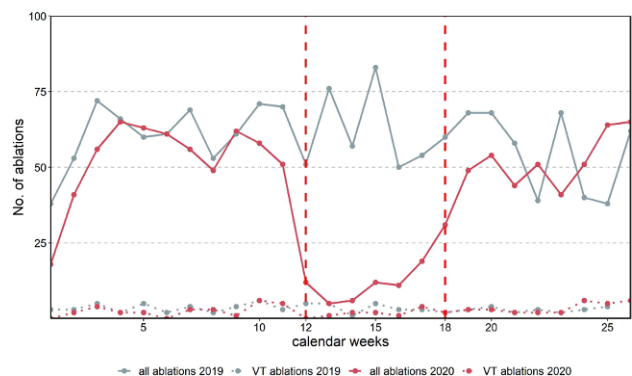
Fig. 1 | CO-1-6 Impact of COVID-19 on ablation procedures in Austria. The absolute numbers of all ablations from January to June 2019 (grey, solid line), of emergent ablations for ventricular tachycardia (VT) from January to June 2019 (grey, dotted line), of all ablations from January to June 2020 (red, solid line), and of emergent ablations for ventricular tachycardia from January to June 2020 (red, dotted line) are shown. Dashed, vertical lines depict the begin and end of lockdown in Austria

Methods: Our data derive from the prospective Austrian ablation registry including all consecutive patients entered in the database until July 1 2020. Austrian public authorities announced measures of social restriction ("lockdown") between March 16th and April 30th, 2020. We compared the number of ablation procedures for any arrhythmia (supraventricular tachycardia, atrial fibrillation or VT) and of emergent ablations VT for the time period of social restrictions with the same time period of 2019.

Results: In total, 500 patients were eligible for this analysis. Mean age was 59.6 ± 14.0 years, 187 (37.4%) were female. Compared to 2019 there was a relative reduction of 76.3% ($n = 404$ vs. $n = 96$) in overall ablation procedures, and a relative reduction of 47.1% in VT ablations ($n = 23$ vs. $n = 12$) (Fig. 1). Of note, the proportion of VT ablations increased from 5.7% in 2019 to 12.5% in 2020.

Conclusion: COVID-19 was associated with a significant decline in total ablations as well as emergent VT cases, although the relative proportion of VT ablations increased. Nevertheless, if the absolute decrease in acute VT ablation procedures translates into elevated morbidity and mortality has to be further elucidated.

POSTERSITZUNG 8 – RHYTHMOLOGIE 2

PS 8/8-1

Cardiac sympathetic denervation for the control of refractory ventricular arrhythmia: a single center experience

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Introduction: Cardiac sympathetic denervation (CSD) alters the autonomic tone of the heart by reducing its sympathetic input. In patients with refractory ventricular arrhythmia not amendable to medical control and radiofrequency ablation, CSD could be considered as an adjunct therapy. Unlike in patient with underlying long QT syndrome (LQTS) and catecholaminergic polymorphic ventricular tachycardia (CPVT) where evidence and experience for CSD use are strong, the efficacy of CSD in ventricular arrhythmia associated with ischemic and non-ischemic cardiomyopathy is relatively undefined. This case series aimed to add to the current understanding of the use of CSD in patients with ischemic and non-ischemic cardiomyopathy and refractory ventricular arrhythmia by following up their post-operative clinical course.

Methods: All patients referred to the Ordensklinikum Linz Elisabethinen from the period December 2018 to December 2019 for the management of refractory ventricular arrhythmia (VAR) and deemed CSD candidates were recruited. A total of 11 patients were identified, four with ischemic cardiomyopathy, one with coronary spasm, one with dilated cardiomyopathy, one with non-compaction and four with arrhythmogenic right ventricular cardiomyopathy. Transthoracic endoscopic bilateral sympathicotomy surgery was performed under general anesthesia. Post-operatively, these patients were contacted by phone at regular interval to update on their ventricular arrhythmia control and their vital status.

Results: The mean monitoring period was 277 days. As of March, 2020, two patients were lost to contact. Concerning the remaining 9 patients for whom follow up information was available, six had no implantable cardioverter defibrillator (ICD) shocks (66%), three had VAR and shocks (33%). In addition, two patients had passed away (22%). One patient had no ICD shock prior to death. Another patient with advanced disease and residual VAR had deceased after heart transplantation. Accepted control of VAR was noted in the two remaining patients with residual ICD shocks, one patient had three ICD shocks in one day followed by no shocks in the following 10 months, and one patient had post-operative ventricular tachycardia followed by no additional ICD shocks. Limitation: Subclinical VAR terminated by anti-tachycardia pacing not reported by patients was not reflected in this study.

Conclusion: CSD was a valuable adjunct therapy to refractory VAR in ischemic and non-ischemic cardiomyopathy patients.

PS 8/8-2

High degree atrial ventricular block and sinus arrest during cryoballoon for atrial fibrillation

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Introduction: A 56-year-old man was referred for pulmonary vein isolation for symptomatic lone paroxysmal atrial fibrillation. There was no history of bradycardia including sinus node dysfunction or heart block. His baseline ECG during sinus rhythm was unremarkable with PR interval of 194 ms and QRS 106 ms. He was on dabigatran 110 mg twice daily and was not on any rate controlling agents.

Methods: Cryoballoon ablation using 28 mm second generation Arctic Front Cryoballoon (Medtronic) was arranged for the patient's pulmonary vein isolation. Transseptal puncture and pulmonary venous access were achieved under fluoroscopic guidance. Adequate sealing of the pulmonary vein ostia by the cryoballoon was confirmed by contrast injection. Isolation of the veins were monitored by local pulmonary vein signals by the Achieve catheter. Left superior pulmonary vein was the first to be isolated. A 240 s freeze was performed uneventfully with stable heart rate, satisfactory target temperature and loss of local venous signal. During thawing, the patient developed prolong sinus pause and complete atrioventricular block that took a total of 42 s to resolved spontaneously (Fig 1). Pacing was started from the coronary sinus catheter, which was immediately available at that juncture. There was atrial capture without ventricular conduction (Fig 2). Right ventricular catheter was inserted for ventricular pacing. Further cryoablation was performed to the remaining left inferior (LIPV), right inferior (RIPV) and right superior pulmonary veins (RSPV) in this order and all resulted in bradycardia. Complete heart block occurred during ablation of all four veins and sinus arrest occurred during LSPV and LIPV ablation. Pulmonary vein isolation in the patient was successful achieved with ventricular backup.

Results: Cryoballoon ablation for pulmonary vein isolation leads to vagal reaction in 24% to 42% of patients, with response ranges from bradycardia to asystole and high degree atrioventricular block. 1–2 This is mainly perceived to be due to the autonomic nervous system modification secondary to cryoablation of the ganglionic plexi located near the junctions of left atrium and the ostium of pulmonary veins. The area of ablation created by second generation 28 mm cryoballoon catheter during PVI is wide and antral, leaving only 27% of posterior left atrial wall

Fig. 1 | PS 8/8-2 Complete atrioventricular block followed by sinus pause during thawing
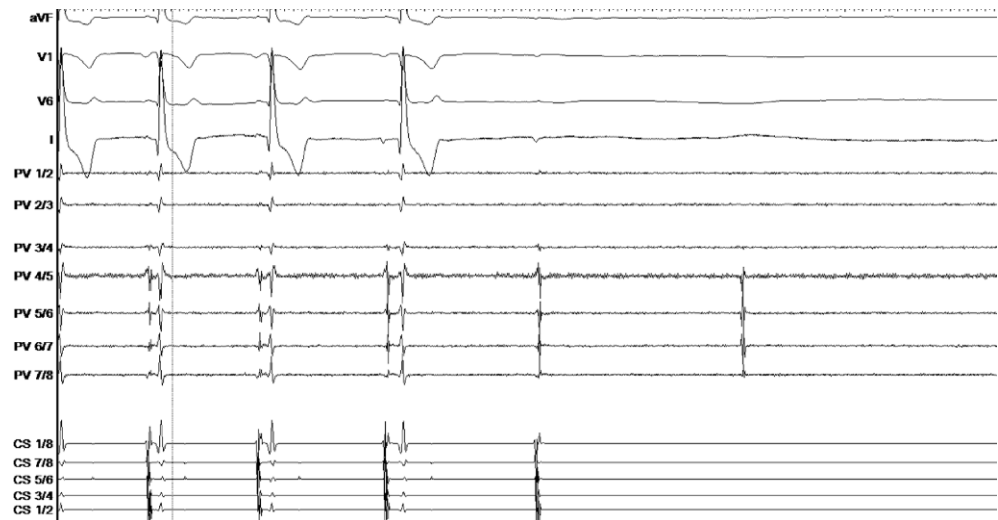
Abb. 2 | PS 8/8-2 Atrial capture without atrioventricular conduction
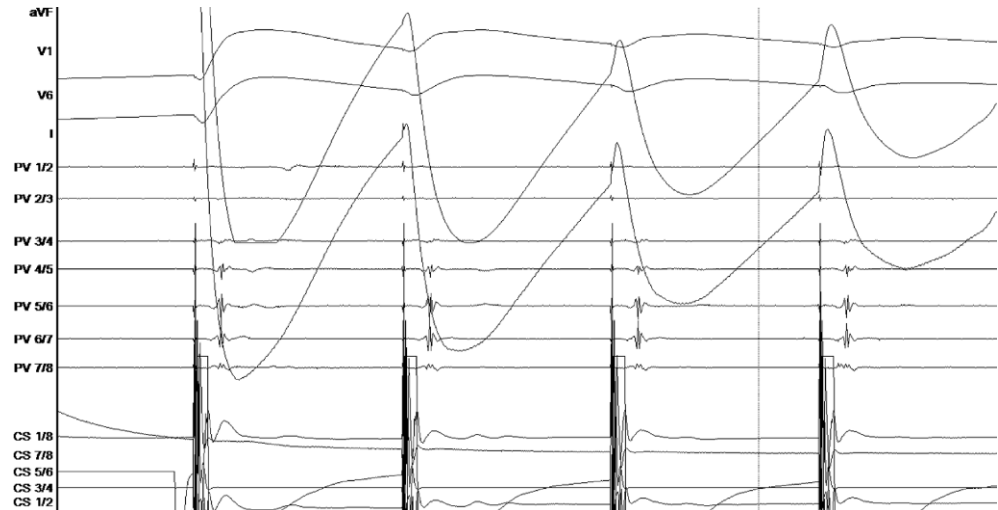
unablated. 3 Thus, the bradycardiac phenomenon observed in our patient could be compatible with vagal response during individual pulmonary vein isolation. Interestingly all bradycardias (AV block or sinus arrest) occurred after the thawing phase and deflation of the balloon. It remains unexplained, why this time latency would occur if bradycardia is caused by irritation of the ganglionated plexi. In addition, hypothermia or even a cardioplegic effect could lead to depression of pacemaker cells automaticity and myocardial conduction delay, giving rise to a multifactorial picture. 4–6

Conclusion: Sinus pause and complete atrioventricular block can occur following cryoablation of all four pulmonary veins, not limited to the more commonly involved left superior pulmonary and right superior pulmonary veins. Modification of the ganglionated plexi, as well as hypothermia itself (cardioplegia) may be the cause of bradycardia.

PS 8/8-3

Mid-term outcome after ablation of paroxysmal and persistent atrial fibrillation using the CLOSE protocol

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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). The novel ablation tool Ablation Index (AI) combines information of contact force sensing, stability and energy output monitoring to predict lesion formation. Standardisation of inter-lesion distance (ILD) and differential AI threshold for the anterior and posterior wall within the CLOSE protocol have shown to increase proce-
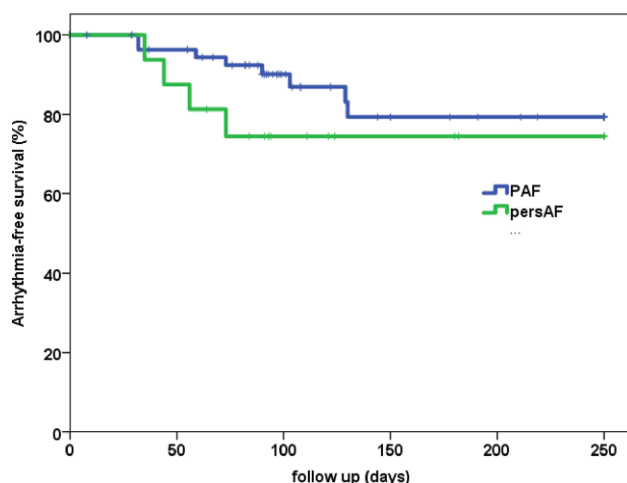
Abb. 1 | PS 8/8-3 Multiple procedure arrhythmia-free survival in patients with paroxysmal (PAF) and persistent atrial fibrillation (persAF) after CLOSE-guided PVI

dural outcome in single centre studies. We aimed to describe mid-term outcome of CLOSE protocol guided ablation.

Methods: 218 consecutive patients (167 PAF and 51 persAF) underwent pulmonary vein isolation (PVI) using a contact force sensing catheter targeting an ILD ≤6 mm and AI ≥380 at the posterior and ≥500 at the anterior wall.

Results: Mean age was 59 ±11 years, 29 % were female, mean BMI was 30 ±3.2 kg/m^2, median CHA2DS2-VASc Score 1 (0, 2), median HAS-BLED Score 1 (0, 2), history of AF was 53 (12, 70) months, mean left ventricular ejection fraction was 59 ±6 %. Median follow up duration was 71 (0, 250) days. 21 % of PAF and 28 % of persAF patients had additional ablation of typical right atrial flutter (p = n. s.). Age (59 ±11 years in PAF patients vs. 60 ±11 years in persAF patients, p = n. s.), gender distribution (31 vs. 22 % females, p = n. s.), CHA2DSs-VASc-Scores (1 [0.1] vs. 2 [1.3], p = n. s.) and AF duration (55 [13.67] vs. 47 [9.72] months, p = n. s.) did not differ between both groups. Patients with PAF had lower BMI (27 [24.30] vs. 29 [26.32] kg/m2, p < 0.05), HAS-BLED scores (1 [0.1] vs. 1 [1.2], p < 0.05) and higher left ventricular ejection fraction (60 ±5 vs. 56 ±8 %, p < 0.001). Primary success rate to meet CLOSE protocol criteria as well as pulmonary vein isolation was achieved in all patients. Eight PAF (5 %) and 4 persAF (8 %) patients had recurrences after the blanking period of 3 months. After multiple procedures (6 re-do procedures in PAF and 2 in persAF patients), arrhythmia-free survival did not differ between both groups (logrank p = 0.26, Fig. 1). There were no procedure-related complications or complications during follow-up.

Conclusion: Strict application of criteria for contiguity and ablation index using the CLOSE protocol is safe and results in a high success rate after PVI. A randomized controlled multicentre trial is needed to compare outcome to conventional PVI approaches.

PS 8/8-4

EHRA physician-based survey: catheter ablation of atrial fibrillation–patient selection, peri- and post-procedural management, and ablation technique

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Introduction: Catheter ablation of atrial fibrillation (AF) is a well-established and widely used therapy option for patients with symptomatic AF. However, management of patients undergoing AF ablation varies in daily practice, especially with the increasing number of available imaging and ablation modalities. The aim of this European Heart Rhythm Association (EHRA) survey was to evaluate current clinical practice regarding management of patients undergoing catheter ablation of AF, in particular the adherence to the 2017 EHRA/HRS/ECAS expert consensus statement on catheter ablation of AF.

Methods: A 25-item questionnaire was presented to 258 EHRA centers from 35 European countries. Among those, 16 Austrian centers participated in the survey.

Results: In patients with symptomatic AF without structural heart disease or other relevant co-morbidities, in whom a rhythm control strategy is feasible (i. e. small LA size), 42 % and 7 % of centers perform first-line AF ablation in paroxysmal and persistent AF, respectively. In patients with symptomatic AF, LVEF <35 %, and a LA diameter <50 mm (CASTLE-AF patients), 72 % routinely perform first-line ablation. AF ablation. 60 % still perform TEE in all patients undergoing ablation. 48 % perform pre-procedural CT/MRI. Only 25 % perform AF ablation without withholding the NOAC morning dose. 61 % perform AF ablation in mild sedation only. In 58 %, the preferred ablation strategy during first-time PVI is radiofrequency ablation. In patients undergoing redo ablation for AF, without any PV reconnections at the beginning of the procedure, 57 % ablate low-voltage areas, 46 % empirical lines, and 31 % CFAEs. 28 % of centers perform an EP study in selected patients undergoing AF ablation. 56 % of centers routinely prescribe antiarrhythmic drugs for three months after ablation. 71 % prescribe PPIs post ablation.

Conclusion: The EHRA survey on AF ablation, with strong Austrian participation, demonstrates a diverse practice in patient selection, peri- and post-procedural management, and ablation technique.

PS 8/8-5

Ventricular storm in a patient with Wolf-Parkinson-White syndrome

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Introduction: Paroxysmal atrial fibrillation (AF) is found in half of the patients with Wolf-Parkinson-White (WPW) syndrome. AF with fast ventricular response due to a fast conducting accessory pathway may lead to life-threatening arrhythmias. Patients presenting with FBI (fast-broad-irregular) are typically young and have no structural heart disease. Risk of sudden cardiac death in patients with WPW syndrome is estimated to be 0.25 %/year.

Methods: Case presentation

Results: We present a case of a 64-year-old patient with previously undiagnosed WPW syndrome. He presented the outpatient clinic with hemodynamically relevant FBI-tachycardia which was interpreted as polymorphic ventricular tachycardia (Fig. 1). He was intubated, cardioverted and coronary angiography was performed ruling out obstructive coronary artery disease. Within five days at the intensive care unit, he required >10 cardioversions, antiarrhythmic drug therapy was started with amiodarone, lidocaine and landiolol resulting in inadequate rhythm control and sinus arrest requiring temporary pacing. The patient was then transferred to our institution for acute ablation. We identified two bidirectionally conducting pathways: a left lateral and a left posteroseptal pathway. Both pathways could be successfully ablated. The patient was extubated two days later and had recurrences of AF and sinus node dysfunction requiring permanent dual chamber pacemaker implantation. The patient could be dismissed from hospital one week after ablation therapy.

Conclusion: Atrial fibrillation in patients with WPW syndrome may lead ventricular storm requiring acute ablation therapy.

PS 8/8-6

100 zero fluoroscopy procedures in electrophysiology–a single center experience

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Introduction: Millions of patients worldwide are suffering from symptomatic tachycardias causing reduced quality of life and frequently requiring hospital treatment. In most cases

Herzfrequenz	211/min
PQ Intervall	ms
QRS Dauer	140 ms
QT/QTc	296/555 ms
P-QRS-T Winkel	/-55/133°
P Dauer	ms
RR/PP Intervall	282/1425 ms
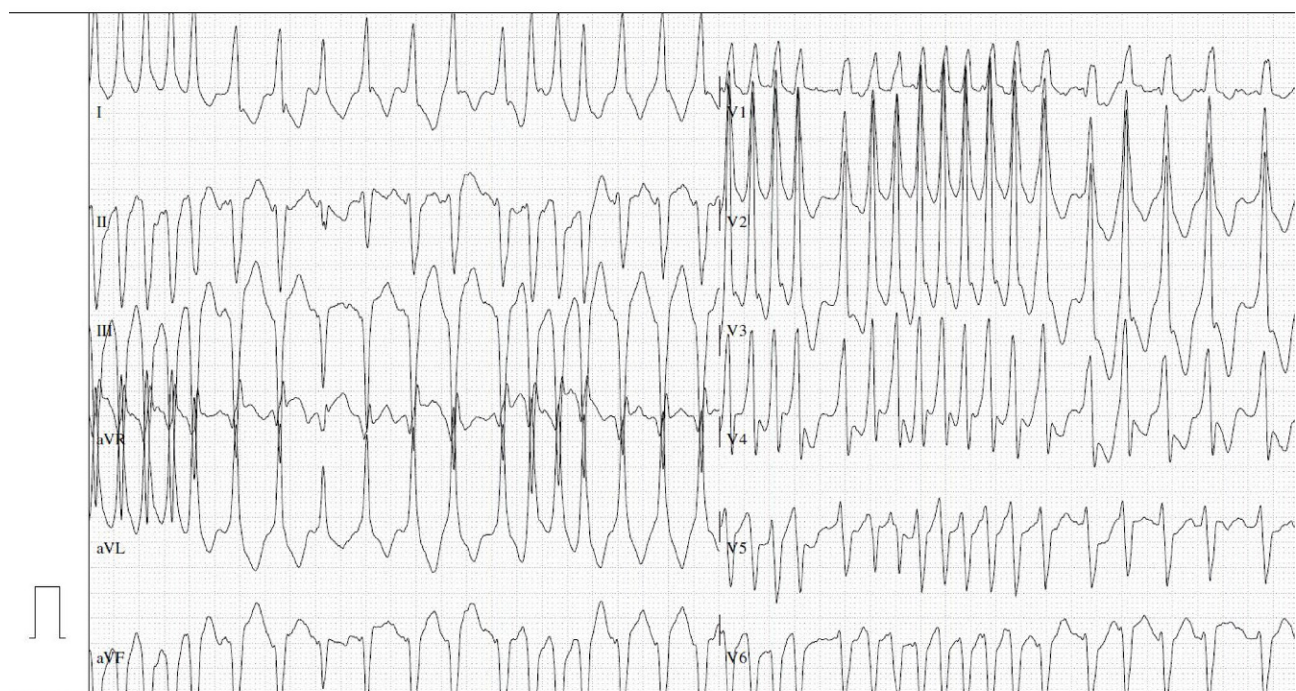
GE CardioSoft V6.73(2)   nicht befundet
25mm/s 10mm/mV 0.01-40Hz 50Hz ADS 12SL V21   Behand. Arzt:

Abb. 1 | PS 8/8-5 12-lead ECG showing pre-excited AF with a ventricular rate of 211/min

of recurrent supraventricular tachycardias, ablation is the recommended treatment. Fluoroscopy is essential for adequate endocardial ablation catheter positioning. Though fluoroscopy duration is low in the majority of ablation procedures, it is well known that radiation can cause severe side effects for the entire cathlab-team on account by cumulating doses over the years. In addition, the weight of radiation protecting coats can give rise to various musculoskeletal disorders. The rapid advancements of 3D mapping technology over the last few years enable highly accurate and thus safe catheter positioning inside the heart without fluoroscopy (zero-fluoro procedure, ZF). However, only few centers worldwide have yet started to perform ablations of arrhythmias using ZF on a routine daily basis. Our department started to perform ZF procedures on a routine basis for electrophysiology (EP) testing and radiofrequency (RF) ablations of arrhythmias. The main aim was to show the feasibility, safety and efficacy of ZF procedures in a consecutive series of patients. As ZF procedure, EP testing and/or ablation without any fluoroscopy ("true" ZF procedure) was defined.

Methods: From September 2018 till February 2020 all right-sided procedures were planned to be performed as true ZF procedures. Only patients with implanted intracardiac electronic devices were excluded, in whom fluoroscopy was used for safety reasons. The guiding and positioning of all catheters was performed using the NAVX Ensite Precision System (Abbott). A monoplane fluoroscopy system (Artis Q Zen, Siemens) was available and ready to use at every time of the procedure, if deemed necessary.

Results: During the 18 months, we performed 100 consecutive true ZF procedures (without any fluoroscopy). The mean age of patients was 54 ±16.7 years with 46 % percent of them being female. Co-morbidities were hypertension (40 %), cardiomyopathy (20 %), coronary artery disease (20 %) and diabetes (12 %) We performed 12 EP testings and 88 ablations (12 % and 88 %, respectively). The following arrhythmias were successfully ablated: Typical atrial flutter ($n=31$), AV-nodal reentrant tachycardia ($n=30$), AV reentrant tachycardia involving accessory pathways ($n=8$), focal right atrial tachycardia ($n=7$), extrasystoles or tachycardias originating from the right ventricular outflow tract ($n=12$). The overall mean procedure time was 105.7 ±43.2 min. Fluoroscopy back-up was not needed in any case (100 % true ZF). Only one complication occurred, a patient with clinical signs of pericarditis after ablation of typical isthmus dependent flutter but neither elevated troponin levels nor pericardial effusion. After a 10 day treatment with ibuprofen the condition resolved without sequelae.

Conclusion: Zero-fluoroscopy procedures for EP testing and RF ablations of right-sided arrhythmias are feasible, effective and safe, when performed by a dedicated and experienced team.

PS 8/8-7

Impact of different three-dimensional electro-anatomical mapping systems on procedural aspects in pulmonary vein isolation

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Introduction: Pulmonary vein isolation (PVI) is a well-established therapeutic approach in symptomatic atrial fibrillation (AF). The introduction of three-dimensional electro-anatomical mapping (EAM) systems has improved success rates and has reduced fluoroscopy times and radiation exposure. Several EAM systems are available with distinctive technological features. Objectives: We investigated the impact of different three-dimensional EAM systems on procedural characteristics during PVI.

Methods: This is a sub-analysis of a prospective ablation registry. For this analysis we included patients undergoing first successful PVI for symptomatic AF between October 2017 and February 2020. For EAM either EnSite NavX™ (Endocardial Solutions, St. Jude Medical, Inc., St. Paul, MN, USA) or CARTO3® system (Biosense Webster, Diamond Bar, CA, USA) was used. As primary endpoint, we investigated the impact of both EAM systems on procedural aspects (ablation points, total procedural time, radiofrequency time, fluoroscopy time). As secondary endpoint, we investigated the impact of both EAM systems on the incidence of redo procedures.

Results: In total, 335 patients were eligible for analysis. Median age was 62 (55–69) years, 220 (65.5 %) were male. For the overall cohort, 198 (59.1 %) patients were referred for paroxysmal AF and 137 (40.9 %) for persistent AF. NavX™ was used in 177 (52.8 %) patients, CARTO3® in 158 (47.2 %). Baseline characteristics were comparable for both groups (Table 1). For all patients, the number of ablation points (80 [IQR 62–98] vs. 73 [IQR 49–93]; $p=0.004$) and total procedural time (136 min [IQR 120–173] vs. 130 min [IQR 112–155]; $p=0.036$) was higher in CARTO3® compared to NavX™, whereas radiofrequency time (27 min (IQR 24–33) vs.30 min (24–39); $p=0.056$) and fluoroscopy time was shorter in CARTO3® (15 min [IQR 11–22] vs. 18 min [IQR 13–24]; $p=0.020$), as depicted in Table 2. These findings were comparable for patients with paroxysmal and persistent AF (Table 3, Table 4). The rate of redo procedures was similar between NavX™ and CARTO3® (15 [9.5 %] vs. 16 [9.0 %]; $\chi2$ $p=0.886$). In binary logistic regression analysis adjusting for CHA2DS2-VASc score and type of AF, there was no significant difference between both EAM systems concerning redo procedures (OR 0.910 [95 %CI 0.428–1.935]; $p=0.910$).

Conclusion: Although, there were significant differences in procedural characteristics comparing both EAM systems, these differences were small and negligible. The use of different EAM systems had no impact on the rate of redo procedures.

Table 1 | PS 8/8-7 Procedural characteristics for all patients stratified for both three-dimensional mapping systems

Characteristics	NavX No.=177	CARTO3 No.=158	*p*-value
Number of ablations, No., median (IQR)	73 (49–93)	80 (62–98)	0.004
Total procedural duration, min., median (IQR)	130 (112–155)	136 (120–173)	0.036
Fluoroscopy time, min., median (IQR)	18 (13–24)	15 (11–22)	0.020
Radiofrequency time, min., median (IQR)	30 (24–39)	27 (24–33)	0.056
Data were compared using Mann-Whitney-U-test.			

POSTERSITZUNG 9 – DIVERSE 1

PS 9/9-1

Quantification of fluid status by bioelectrical impedance spectroscopy in patients with valvular heart disease: sex matters

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Introduction: Fluid overload, which may finally lead to cardiac decompensation, is a major threat in valvular heart disease (VHD) patients. In clinical practice, leg edema, pulmonary congestion, and rapid weight gain indicate fluid overload. However, these parameters lack both specificity and sensitivity. Bioelectrical impedance spectroscopy (BIS) is an easy, non-invasive and reliable way to determine the extent of fluid overload. BIS it already used in patients on chronic haemodialysis to guide therapy. Whether fluid status as measured by BIS is associated with outcome in VHD patients is unknown.

Methods: Stable patients with moderate or severe VHD as diagnosed by transthoracic echocardiography underwent fluid status assessment by BIS at baseline and were prospectively followed. The primary endpoint was a composition of heart failure hospitalisation and cardiovascular death. Patients with overt cardiac decompensation or on intra-venous diuretic therapy were excluded from this study. Kaplan-Meier estimates and multivariable Cox-regression analysis were used to identify sex-specific factors associated with outcome. This study was registered at clinicaltrials.gov (NCT03372512).

Results: 336 patients (51.8 % female, 76 ± 13 years) were included in the study. 26.2 % (3.5 % moderate, 22.7 % severe) suffered from aortic stenosis, 50.9 % from mitral regurgitation (16.1 % moderate, 34.2 % severe) and 11.8 % (6.2 % moderate, 5.6 % severe) from aortic regurgitation. A total of 68.5 % of the patients additionally presented with tricuspid regurgitation. Mean overhydration was +0.6 l with no significant differences between men and women ($p = 0.076$). We did not observe sex-specific differences in baseline characteristics with the exception of higher left-ventricular ejection fraction ($p = 0.007$) as well as better renal function ($p = 0.003$) in women compared to men. During a follow-up of 433 ± 364 days, a total of 153 events (45.7 %) occurred. 102 patients (30.4 %) underwent valve intervention, which was not considered as an event, and were cen-

sored from the analysis. Sex-specific stratification of patients based on OH tertiles revealed that overhydration was associated with significantly higher event-rates in men (log-rank $p = 0.002$, see Fig. 1), but not in women ($p = 0.127$, see Fig. 2). Similarly, in the multivariate cox-regression, OH was significantly associated with outcome only in men ($p = 0.009$) after adjustment for cardiac size and function, NT-proBNP, diabetes, coronary artery disease, NYHA functional class, renal function, and history of cardiac decompensation. In female patients, only NT-proBNP ($p = 0.001$) was significantly associated with outcome whereas OH was not ($p = 0.849$).

Conclusion: Fluid status, as determined with BIS, is significantly associated with outcome in male but not in female patients with VHD. Sex-specific approaches for risk assessment and fluid management should be further examined.

PS 9/9-2

Falsch-negative Ergometrien durch zu frühes Abbrechen: Gefahr nicht nur für Sportler und sportliche Patienten

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Einleitung: Die Quantifizierung und Objektivierung der körperlichen Leistungsfähigkeit (Pmax) spielt nicht nur im Rahmen der Leistungsdiagnostik bei Sportlern, sondern auch als diagnostische Untersuchung in der gesamten Medizin eine wichtige Rolle. Um die körperliche Leistungsfähigkeit korrekt beurteilen und etwaige Pathologien identifizieren zu können, werden Personen auf einem Ergometer, beispielsweise einem Fahrrad- oder Laufbandergometer, maximal ausbelastet. Zur Interpretation der Pmax dienen Referenzwerte – in Österreich häufig nach Niederberger et al. [1]. In der Praxis wird die Belastung entgegen gültiger Leitlinien oft bereits bei Erreichen derartiger Referenzwerte abgebrochen. Liegt der Referenzwert niedriger als die tatsächliche Pmax, ist die Belastung lediglich submaximal und es besteht somit die Gefahr, Pathologien zu übersehen, die klinisch und diagnostisch erst unmittelbar vor oder getriggert durch maximale Belastung auftreten könnten. Deshalb ist es Ziel dieser Studie, zu vergleichen, inwiefern sich die Leistungsfähigkeit eines sportlichen Untersuchungskollektivs von Normwerten aus der Literatur unterscheidet.
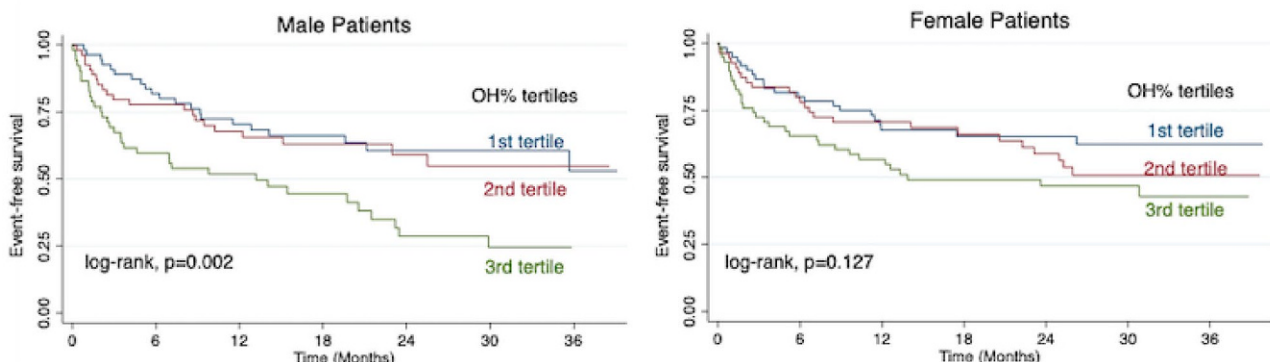
Fig. 1 | PS 9/9-1
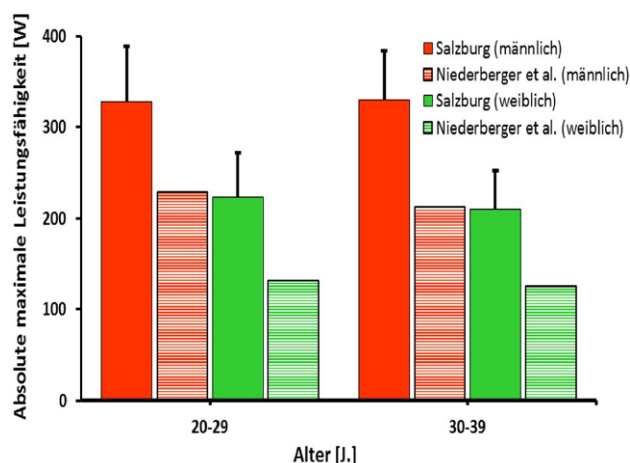
Abb. 1 | PS 9/9-2
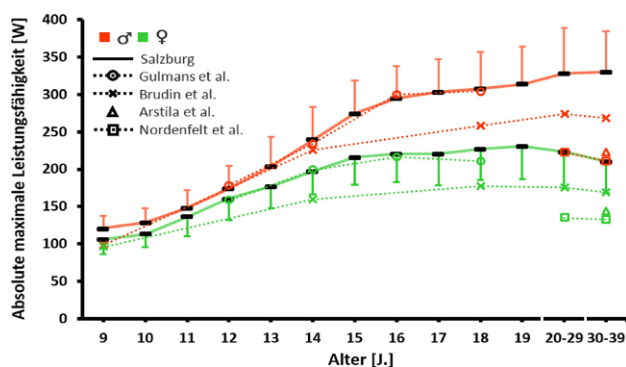
Abb. 2 | PS 9/9-2

Methoden: Zwischen 1998 und 2018 wurden 39.172 Personen ergometriert. Davon absolvierten 12.745 Personen (67 % männlich) im Alter von 9 bis 39 Jahren mit einer wöchentlichen Trainingszeit von über drei Stunden eine Fahrradergometrie und 9757 (73 % männlich) eine Laufbandergometrie.

Resultate: Bei der Fahrradergometrie erreichten männliche Personen eine Pmax von durchschnittlich 277 ± 78 W, weibliche von 194 ± 51 W. Das ergibt bei Männern eine relative Pmax von $4{,}2 \pm 0{,}7$ W kg^{-1} und bei Frauen von $3{,}6 \pm 0{,}6$ W kg^{-1}. Im Vergleich zu Referenzwerten aus der Literatur erreichten die in Salzburg untersuchten Erwachsenen absolut um 66 ± 27 W und relativ um $1{,}0 \pm 0{,}5$ W kg^{-1} höhere Werte. Bei uns untersuchte Kinder und junge Erwachsenen (≤ 19 Jahre) lagen hinsichtlich der absoluten Pmax ebenfalls deutlich über den Literaturwerten. Bei der Laufbandergometrie erzielten männliche Personen im Durchschnitt eine maximale Geschwindigkeit von 15,5 ± 1,9 km/h und weibliche von 13,2 ± 1,6 km/h.

Schlussfolgerungen: Die körperliche Leistungsfähigkeit sportlicher Personen ist weit größer als die in der Literatur angegebenen Referenzwerte. Da eine Ergometrie nur dann diagnostisch aussagekräftig ist, wenn die Personen maximal ausbelastet werden, besteht bei zu früh abgebrochenen Ergometrien die Gefahr, Pathologien wie Ischämien und Arrhythmien nicht zu induzieren. Solche falsch-negativen Ergometrien sind nicht nur gefährlich, sondern in ihrem Ergebnis potentiell lebensbedrohlich. Folglich ist eine maximale Ausbelastung bei Ergometrien, wie in den Leitlinien gefordert, unumgänglich.

Sacubitril/valsartan in patients with heart failure and a history of cancer

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Introduction: Sacubitril/valsartan has been shown to significantly reduce cardiovascular mortality and hospitalizations due to heart failure in patients with reduced ejection fraction when compared to enalapril. Data about sacubitril/valsartan in patients with a history of cancer are scarce, as these patients were excluded from the pivotal trial, PARADIGM-HF. The aim of the current study was to assess tolerability of sacubitril/valsartan in patients with a history of cancer.

Methods: We identified 225 patients at our heart failure out-patient unit who fulfilled the indication criteria to receive sacubitril/valsartan. Out of these, 9.3 % ($n = 21$) had a history of histologically confirmed cancer: 23.8 % breast cancer ($n = 5$), 14.3 % colorectal cancer ($n = 3$), 14.3 % non-Hodgkin lymphoma ($n = 3$), 9.5 % osteosarcoma ($n = 2$), 9.5 % renal cell carcinoma ($n = 2$), lung cancer, Hodgkin lymphoma, prostate cancer, bladder carcinoma, pancreas carcinoma, multiple myeloma, acute leukaemia and myeoloproliferative syndrome (each 4.8 %, $n = 1$). Surgery due to cancer was performed in 76.2 % of patients ($n = 16$), 42.9 % previously received chemotherapy ($n = 9$) and 42.9 % radiation therapy ($n = 9$).

Results: Sacubitril/valsartan was withdrawn in 3 patients (14.3 %) because of dizziness or pruritus. After a median follow-up of 12 months (range 1–34 months), NYHA functional class improved significantly (mean –0.6, $p = 0.006$), left ventricular ejection fraction as assessed by echocardiography increased (mean +9 %, $p = 0.004$) and NT-proBNP was significantly decreased (mean –1927 pg/ml, $p = 0.015$). There was no significant change in creatinine levels (mean +0.094 mg/dl, $p = 0.256$).

Conclusion: In this pilot study we were able to show that sacubitril/valsartan is generally well tolerated in patients with a history of cancer. Patients with cardiotoxicity induced heart failure can be treated and up-titrated with sacubitril/valsartan to usual dosages similarly as in other causes of heart failure. Larger studies are needed to confirm these findings in cancer patients with cardiotoxicity.

Gastric regurgitation predicts neurological outcome in out-of-hospital cardiac arrest survivors

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Introduction: Hypoxic-ischemic brain injury can affect and disturb the autonomous nervous system (ANS), which regulates various visceral systems including the gastro-intestinal and emetic system. The present study aimed to analyze the predictive value of gastric regurgitation (Greg) for neurological outcome in out-of-hospital cardiac arrest (OHCA) survivors.

Methods: In this prospective, single-center study, 79 OHCA survivors treated at a university-affiliated tertiary care centre were included and GReg was measured at the first day after successful cardiopulmonary resuscitation. Neurological outcome was assessed by the Cerebral Performance Categories score at discharge.

Results: Seventy-six percent of the study population had a poor neurological outcome. GReg was found to be associated with poor neurological outcome with an adjusted OR of 5.89 (95 % CI 1.56–22.25; $p = 0.009$). The area under the ROC curve for GReg was 0.69 (95 % CI, 0.56–0.81) for poor neurological outcome.

Conclusion: GReg on the first day after OHCA is an early, strong and independent predictor for poor neurological outcome in comatose OHCA survivors. These results are particularly compelling because measurement of GReg is inexpensive and routinely performed in critical care units.

PS 9/9-5

Simultaneous bilateral ultrasound assisted catheter based local thrombolysis in acute pulmonary embolism

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Introduction: The prognosis of acute pulmonary embolism (PE) is determined by the hemodynamic status and right ventricular function. Systemic thrombolysis is the treatment of choice for high-risk PE with hemodynamic instability, but is associated with an increased risk of bleeding. Therefore it is not recommended for the treatment of intermediate high-risk PE. Catheter-based therapies for PE are promising alternatives with a better risk-benefit-ratio due to a lower bleeding risk. We herewith report a series of successful simultaneous bilateral use of the EkoSonic® ultrasound assisted catheter-directed system (EKOS) in patients with intermediate high-risk PE who were not candidates for systemic fibrinolysis.

Methods: From July to December 2019 patients with PE admitted to our institution were screened for eligibility for ultrasound assisted catheter based local thrombolysis with EKOS. EKOS uses high frequency (2 MHz) sound waves to augment the penetration of local thrombolytics while causing the reversible disaggregation of uncrosslinked fibrinogen. Patient selection for local thrombolysis was based on proximal thrombus location, hemodynamic status and PE severity index. PE was diagnosed by CT-pulmonalis angio (CTPA). From a right femoral
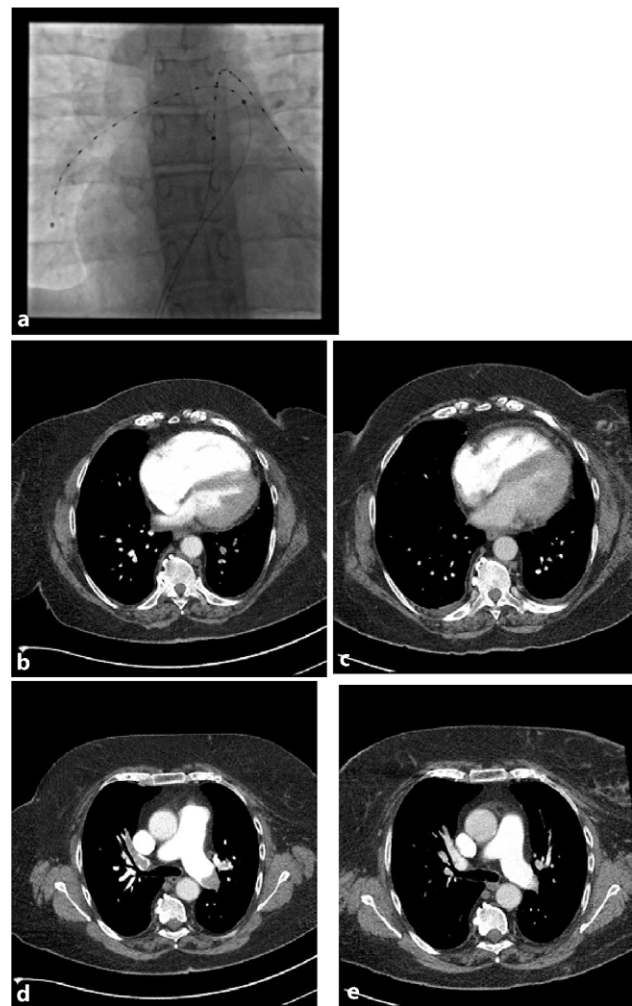
Abb. 1 | PS 9/9-5 Flouroscopy of bilateral catheters in the pulmonary arteries. Change in right ventricular and left ventriculare size before (*left*) and after (*right*) treatment. Thrombus burden before (*left*) and after (*right*) catheter treatment of acute pulmonary embolism

approach 5.4-Fr catheters with microscopic side holes and an ultrasound core catheter were placed under local anesthesia in the main branches of left and right pulmonary arteries, dependent on thrombus location in CTPA. Based on the ULTIMA trial, 10 mg tissue plasminogen activator (rTPA) were infused locally at each pulmonary artery over 10 h in combination with unfractionated heparin aiming at an activated partial thromboplastin time (aPTT) of 50–70s. We analysed hemodynamic parameters (systolic blood pressure, heart rate), right ventricular to left ventricular (RV/LV) ratio in echocardiography and change of thrombus-burden in pre- and post-procedural CTPA.

Results: From July to December 2019, 105 patients were diagnosed with acute PE in our institution. 51 patients had central PE, 5 patients were considered "high-risk", 17 patients were stratified as "intermediate high-risk". 4.8 % of PE patients (2 male, 3 female, mean age 54.8 years) were considered eligible for catheter based treatment. One patient was classified "high-risk" because of hemodynamic instability, but had an absolute contraindication for systemic thrombolysis because of polytrauma. The other patients were considered "intermediate high-risk" with right heart dilatation and elevation of cardiac biomarkers. All patients received simultaneous bilateral treatment with EKOS catheters in the right and left pulmonary artery

Tab. 1 | PS 9/9-5 Baseline characteristics and outcomes of patients treated with ultrasound assisted catheter based local thrombolysis for acute pulmonary embolism

Age, gender	Risk	hsTrop admission ng/L	RV/LV ratio before	RRsyst before-> after mmHg	Heartrate before -> after/ min	RV/LV Ratio after	CTPA central thrombi	complication
72, female	IMHR	329	>1	110->125	110->83	<1	clear	no
15, female	IMHR	188	>1	100->110	130->105	<1	—	no
50, male	IMHR	120	>1	105->130	106->70	<1	reduced	no
59, male	HR	660	>1	75->95	120->87	<1	—-	bleeding
78, female	IMHR	161	>1	100->110	110->85	= 1	reduced	no
IMHR = intermediate high-risk; HR = high-risk; CTPA = computed tomography pulmonary angiogram; RV = right ventricle; LV = left ventricle								

(figure 1A). In all 5 patients an increase in systolic blood pressure and decrease in heart rate was documented within the onset of treatment. In 4 patients a change in RV/LV ratio from more than 1 to less than 1 was documented within 12 h of therapy (Fig. 1B). In one patient RV/LV ratio chanced from >1 to equal 1, whereas this patient was also thought to have chronic pulmonary hypertension (Table 1). 3 patients had another CTPA 24 to 48 h after local thrombolysis: 2 showed marked reduction and 1 showed moderate reduction in thrombus burden (Fig. 1C). One patient experienced an adverse event with bleeding in his thigh due to hip fracture after the polytrauma. He needed to undergo surgery and also received a transient vena cava filter for 14 days.

Conclusion: Ultrasound assisted simultaneous bilateral catheter based local thrombolysis with the EKOS system is a feasible and effective treatment option for intermediate high risk PE patients and unstable PE patients with contraindications for systemic thrombolysis. Clinical improvement occurs within hours, due to a reduction in pulmonary artery resistance documented by a reduction in thrombus burden and improvement in RV/LV-ratio.

PS 9/9-6

Sentinel device in use during revision of a pacemaker probe: a case report

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Introduction: Probe malposition is a rare but nevertheless occurring complication in the implantation of an ICD. At present, it is recommended to apply systemic anticoagulation with warfarin in such a situation. Complications of left ventricular probe placement can range from aortic valve endocarditis, pericardial effusion, peripheral artery thrombosis and thrombotic events (TE). The Heart Rhythm Society recommended in 2009 that surgical extraction of the misplaced probe is a class III recommendation. However, other experts recommended its removal under cardiopulmonary bypass. The most important complication during extraction of the probe is TE, which in the worst case can lead to stroke. [1] The Sentinel device is currently only recommended for TAVI. A meta-analysis has shown that 30 days after TAVI the risk of suffering a stroke or TIA is 3.3 % +/– 1.8 %. Therefore, neuroprotection is almost indispensable. [2] However, here we show a case where the sentinel device was also successfully used in another transcatheter intervention.

Methods: In a transthoracic echocardiography the misplaced RV-ICD probe was detected. This could be confirmed by a CT. After it was also shown in the TTE that "floating" deposits were present on the misplaced probe, the decision was made for neuroprotection during the revision surgery of the probe. The sentinel device was positioned in the brachiocephalic trunk and in the left common carotid artery. Afterwards, the probe, which had been incorrectly positioned via the persistent foramen ovale (PFO), was retracted. A new RV-ICD probe could then be correctly positioned in the right ventricle. Washing out the sentinel device was successful in recovering a thrombus, thus confirming the successful use of the device.

Results: The successful use of the sentinel device as neuroprotection in a revision surgery of the misplaced RV-ICD probe could be shown. Thus, the Sentinel device is not only limited to TAVI. In the appendix "Document 1" we have shown the thrombus that could be recovered in the Sentinel device.

Conclusion: After TAVI 1/10 patients show overt signs of ischemic brain injury. There is also a 6 × increased risk in stroke-related 30-day-mortality post-TAVI. Sentinel is a cerebral protection system to capture and remove thrombus while performing TAVI procedures. In 99 % of TAVI procedures Sentinel has shown to capture and remove stroke-causing embolic debris. In the randomized SENTINAL trial overt strokes were reduced from 8.2 % in unprotected TAVI patients to 3 % in patients undergoing protected TAVI within the first 72 h. [3] In the first 72 h after a TAVI, 85 % of all strokes are diagnosed. It is assumed that these are mainly induced by detached thrombi. New cerebral lesions were found in almost 100 % of patients after TAVI on MRI, most of them do not cause a clinically significant stroke. [4] Thus, the sentinel device has an essential role in neuroprotection during a TAVI. However, in the context of this case we were able to show that the Sentinel device can be used as successful neuroprotection not only during TAVI.
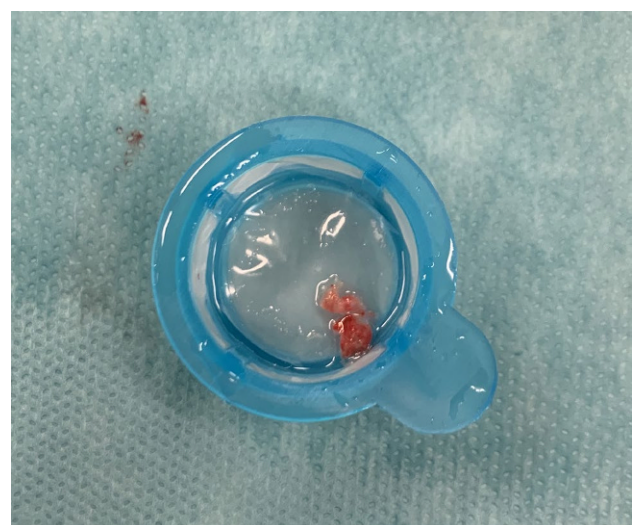
Abb. 1 | PS 9/9-6 Thrombus in the sentinel device

PS 9/9-7

Rehabilitation von PatientInnen mit symptomatischer peripherer arterieller Verschlusskrankheit – Ergebnisse einer prospektiven Beobachtungsstudie

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Einleitung: In den ESC Guidelines 2017 wird der Empfehlungsgrad für ein überwachtes Gefäßtraining von symptomatischen PatientInnen (Pat) mit peripherer arterieller Verschlusskrankheit (PAVK) als IA Indikation geführt. Diese Form des Trainings ist in der Gefäßrehabilitation ein wesentlicher Bestandteil. Grundsätzlich werden 4 Rehabilitationsphasen unterschieden. Die Phase 1 beginnt im Akutkrankenhaus im Rahmen der Akutbehandlung, die Phase 2 kann stationär und ambulant fortgesetzt werden und die Phase 3 erfolgt ausschließlich ambulant mit dem Bestreben die erzielten Effekte zu stabilisieren und langfristige positive Veränderungen des Lebensstils zu erreichen. In der Phase 4 steht die selbstständige Festigung der Veränderungen im Vordergrund. Ziel der Studie war es, die Effekte bei Pat am Ende der Phase 3 im Anschluss an eine Phase 2 zu vergleichen mit Pat, die nur eine Phase 2 absolvierten .

Methoden: Wir untersuchten prospektiv Pat, die eine Phase 2 entweder ambulant im Zentrum für ambulante Rehabilitation in Wien über 6 Wochen oder stationär in der Sonderkrankenanstalt Felbring über 4 Wochen in den Jahren 2013 bis 2015 absolvierten. In weiterer Folge konnten alle Pat, die in Wien und Umgebung wohnten an einer ambulanten Phase 3 über 12 Monate 2x/Woche über je 1 h teilnehmen. Die Ergebnisse wurden am Beginn (T0) und am Ende (T1) der Phase 2 und am Ende der Phase 3 bzw. 1 Jahr nach Beendigung der Phase 2 (Patienten ohne Phase 3) (T3) in Hinsicht auf die absolute Gehstrecke (ACD – absolute claudication distance), die Anzahl der durchgeführten Zehenstände und die krankheitsbezogene Lebensqualität mittels eines Fragebogens (Vascuqol) erhoben.

Resultate: 162 Pat (104 Männer) absolvierten entweder eine stationäre oder ambulante Rehabilitation, davon setzten 54 Pat (30 Männer) diese in Form einer ambulanten Phase 3 fort. In der Phase 2 (T0 bis T1) kam es zu einer signifikanten Verbesserung der ACD (429,1 ±349,1 auf 549,9 ±399,4 m, $p = ,000$), der Anzahl der durchgeführten Zehenstände (42,8 ±27,1 auf 55,1 ±31,5, $p = ,000$), so auch der Ergebnisse des Vascquol (4,79 ±1,1 auf 5,33 ±1,11 $p = ,000$). Pat, die die Rehabilitation in der Phase 3 fortsetzten konnten weiter von T1 auf T3 die ACD von 598,8 ±417,0 auf 863,1 ±629 m, $p = ,000$ verlängern und mehr Zehenstände absolvieren (60,0 ±41,0 auf 79,2 ±40,5, $p = ,000$). Die 31 Patienten (23 Männer) der Kontrollgruppe ohne Phase 3 hingegen zeigten keine weiteren signifikanten Veränderungen – ACD 648,5 ±536,4 auf 784,3 ±628,3 m, $p = ,126$, Zehenstände 53,7 ±28,0 auf 52,6 ±35,6, $p = ,844$. Dies spricht dafür, dass die Effekte auch 1 Jahr nach Ende der Phase 2 erhalten blieben. Weder bei den Pat mit absolvierter Phase 3 (5,39 ±1,12 auf 5,24 ±1,33, $p = ,337$) noch ohne Phase 3 (5,49 ±1,2 zu 5,36 ±1,15, $p = ,552$) veränderte sich nach Ende der Phase 2 (T1 zu T3) signifikant die krankheitsbezogene Lebensqualität.

Schlussfolgerungen: Die Gefäßrehabilitation der Phase 2 bewirkt eine Verlängerung der ACD, eine Erhöhung der Anzahl der absolvierten Zehenstände und auch eine Verbesserung der krankheitsbezogenen Lebensqualität. Diese Effekte blieben auch ohne Phase 3 über 1 Jahr erhalten. Pat, die die Rehabilitation mit einer Phase 3 fortsetzten, konnten im Gegensatz zur Kontrollgruppe ohne Phase 3 weiter die ACD verlängern und die Anzahl der absolvierten Zehenstände erhöhen. Bei der krankheitsbezogenen Lebensqualität konnte nach der Verbesserung in der Phase 2 keine weitere Veränderung weder bei den Pat mit Phase 3 noch ohne Phase 3 dokumentiert werden.

PS 9/9-8

The potential of objective physical activity measurements in cardiac rehabilitation–A scoping review

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Introduction: Promoting regular physical activity and thus improving exercise capacity are primary goals of cardiac rehabilitation (CR), which is usually executed as a facility-based multidisciplinary and multiphase program. Modern digital technologies enable monitoring peoples' physical activity and thus hold promise to help patients achieve their goals of increasing physical activity during CR and lifelong home-based exercise training. The aim of this scoping review was to identify studies where digital interventions are employed to objectively monitor and promote physical activity during CR.

Methods: PubMed, Cochrane Central Register of Controlled Trials and Cochrane Database of Systematic Reviews were searched for efficacy studies published between December 2014 and December 2019, since we aimed to identify studies that employed current technology. Randomized controlled trials that include technologies to objectively measure physical activity as part of an intervention to improve exercise capacity were included. In addition, technologies and behavior change strategies were compared across the different studies. We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement.

Results: A total of 964 publications were identified and 13 studies met all inclusion criteria. All 13 studies employed complex interventions, using multiple and diverse digital technologies and behavior change strategies, such as feedback on exercise, goal setting and communication with physician.

Table 1 | PS 9/9-8 Strategies and effects.

	Strategies for behavior change							Effects[a]		
	Education	Feedback on exercise	Goal setting	Physician/expert involved	Real-time monitoring	Self-monitoring	Tailored prescription	IG: within-group	CG: within-group	Δ IG vs. Δ CG: between-group
Digital technology during home-based CR vs. outpatient CR										
Avila 2018[b] [1]		√	√	√		√	√	–	–	–
Kraal 2017 [6]		√	√	√		√	√	↑	↑	–
Maddison 2019 [7	√	√	√	√	√	√	√	⊗	⊗	⊙
Digital technology during home-based CR immediately following outpatient CR vs. usual care										
Avila 2018[c] [1]		√	√	√		√	√	–	–	↑
Duscha 2018 [2]	√	√	√	√		√	√	↑	↓	↑
Fang 2019 [3]	√	√	√	√	√	√	√	↑	↑	↑
Frederix 2015 [4]		√	√	√		√	√	↑	–	↑
Frederix 2017[d] [5]	√	√	√	√		√	√	↑	–	↑
Piotrowicz 2015[e] [8]		√	√	√		√	√	↑	–	↑
Piotrowicz 2019[e] [9]	√	√	√	√		√	√	↑	↑	↑
Skobel 2017 [11]	√	√	√	√	√	√	√	–	–	↑
Snoek 2019 [12]		√	√	√		√	√	↑	↑	–
Vogel 2017[f] [3]		√	√	√		√		↑	↓	↑
Outpatient CR with vs. without digital technology										
Rosario 2018 [10]		√	√			√		–	–	–
Vogel 2017[g] [13]		√	√	√		√		↑	↑	–

↑: statistically significant improvement; ↓: statistically significant deterioration; -: no statistically significant difference; ⊗: no within-group statistical comparison reported, but similar improvements observed in both groups descriptively; ⊙: statistically non-inferior.
[a]Changes in exercise capacity directly after intervention compared to baseline
[b]IG: home-based CR, CG: outpatient CR.
[c]IG: home-based CR, CG: usual care.
[d]First 6 of 18 weeks of study in outpatient CR for both IG and CG.
[e]Effects for both VO2 peaks and 6MWT.
[f]Last 6 of 12 weeks of study: home-based continuation vs. usual care.
[g]First 6 of 12 weeks of study: both IG and CG underwent an outpatient CR.

Studies were grouped according to study design; there were 15 between-group comparisons: (a) digital technology during home-based CR vs. outpatient CR ($n=3$); (b) outpatient CR followed by home-based CR with digital technology vs. outpatient CR followed by usual care ($n=10$); and (c) outpatient CR with vs. without digital technology ($n=2$). Digital technology during home-based vs. outpatient CR as well as outpatient CR with vs. without digital technology did not lead to statistically significant differences in exercise capacity. In contrast, digital technology during home-based CR immediately following outpatient CR led to improvements in exercise capacity as compared to usual care.

Conclusion: Supplying patients with digital technologies that monitor physical activity during home-based and/or outpatient CR does not necessarily lead to improvement in physical exercise capacity. However, after an outpatient CR, home-based CR with digital technology to monitor and promote physical activity is superior to usual care. Future research should evaluate the contribution of digital technology and behavior change strategy components in complex CR interventions to effectiveness.

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POSTERSITZUNG 10 – KORONARE HERZKRANKHEIT

PS 10/10-1

SWEATY HEARTS–A collaborative partnership to develop, implement and evaluate a model of long-term physical activity and behavioral change in CHD European patients

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Introduction: Exercise training and increased physical activity is a cornerstone in the rehabilitation of cardiac diseases. Unfortunately, after completion of rehabilitation programs many patients fall back into previous routines and thus a rather sedentary lifestyle. This increases their risk of new cardiac events. Sweaty Hearts, a European funded project (ERA-COPART-2017-2778) set out to apply new technologies, like smartphone or podcasts, in order to help patients with coronary artery disease maintain an active lifestyle after cardiac rehabilitation.

Methods: In this demonstration project, 103 participants (63.7 years ±8.9; 79.6 % males) with underlying coronary artery disease from the five participating countries, participated in this trial. First, patients participated in a center-based cardiac rehabilitation for 24 weeks with exercise and education. Hereafter, patients were monitored by a two-weekly or monthly transmission of their step count measured with smartphone applications. Patients received feedback and new goals via e-mail or by telephone based on the transmitted step counts. All subjects underwent exercise testing and filled in questionnaires at baseline, after the completion of the first phase (24 weeks) and again after another 24 weeks. The main goal was to assess sustainability of this model as well as to test if this model was feasible in different European countries.

Results: 74 participants finished both phases of the trial. Quality of life increased in every country after 48 weeks. The overall knowledge about CAD increased during 24 weeks in-center but decreased after 48 weeks. Peak power during exercise testing increased significantly after 12 months (Peak power baseline 144 W; 24 weeks 155 W ($p < 0.001$); 48 weeks 156 W (p-value vs. 24 weeks = 0.576; p-value vs. baseline <0.001)). Exercise capacity was significantly higher after the first phase [VO2peak baseline: 22.5 ml/min/kg; VO2peak 24 weeks 23.6 ml/min/kg (p = 0.011)] and decreased again after 48 weeks [VO2peak 48 weeks 22.9 ml/min/kg (p-value vs. 24 weeks = 0.056; p-value vs. baseline = 0.437)].

Conclusion: This demonstration project showed that smartphone based step count monitoring helped preserve the initially gained physical fitness during a phase III cardiac rehabilitation program. In all participating countries, the intervention had a positive impact on quality of life, flexibility and muscle strength and physical fitness. However, knowledge of CAD decreased once patients where unsupervised. This suggests that regular booster sessions might be helpful. Lastly, even though the project showed positive results, more research is needed to find alternative ways to motivate especially women to stay physically active.

PS 10/10-2

Dose reduction of NOACs during triple antithrombotic therapy after percutaneous coronary intervention from a real-life perspective

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Introduction: The current 2019 ESC guidelines for the management of chronic coronary syndromes (CCS) recommend at least one month of triple antithrombotic therapy (TAT) after percutaneous coronary intervention (PCI) in patients with an indication for oral anticoagulation (OAC) when the risk of stent thrombosis outweighs the bleeding risk. Dose reduction of NOACs, particularly during TAT, is discussed controversially and CCS guidelines recommend a reduction only when formal reduction criteria apply. We therefore aimed to picture the antithrombotic management and application of dose reduction criteria during TAT from a real-life perspective.

Methods: Patients presenting with the indication for NOACs who underwent PCI between 01/2017 and 10/2019 were enrolled in our clinical PREDICT-OAC registry. Duration of TAT was evaluated according medical prescription at time of discharge and during follow-up visits. Patients were followed prospectively until the primary study endpoint (= cardiovascular mortality) was reached.

Results: We identified 238 patients (median age 77 [IQR: 71–83]; male: 70.6 %) with a median CHA2DS2-VASc-Score of 4 (IQR 3–4). Elective PCI was more frequently observed (65.1 %) than acute coronary syndrome (34.9 %). 39.9 % ($n = 95$) patients received apixaban, 12.6 % ($n = 30$) dabigatran, 20.6 % ($n = 49$) edoxaban and 26.9 % ($n = 64$) rivaroxaban during TAT. 86.6 % ($n = 206$) were discharged with a reduced dose of a respective agent. Out of these, 44.7 % were discharged with a transient [34.8 % ($n = 32$) during TAT; 65.2 % ($n = 60$) during dual antithrombotic therapy (DAT)] and 55.3 % ($n = 114$) with a permanent dose reduction. When applying the recommended respective criteria for dose reduction, 74.3 % of all patients with dose reduction received a reduced therapeutic regimen without any indication for dose reduction with 84.6 % in the apixaban arm, 74.1 % dabigatran, 57.1 % edoxaban and 72.9 % rivaroxaban, respectively. A prolonged dual anti-platelet therapy (DAPT) of >1 month was found in 33.2 % of all cases, resulting in a median time for TAT of 30 days (IQR: 30–68). 97.7 % were discharged with clopidogrel. During a median follow-up time of 19 months (IQR: 12–27) 15 patients experienced the primary study endpoint (12 individuals receiving apixaban and 3 edoxaban). There was no impact on outcome observed when comparing NOAC agents with justified vs. unjustified dose reduction (HR 0.69 [95 %CI: 0.24–2.02;$p = 0.498$]) and transient dose reduction during TAT vs. permanent reduction during DAT (HR 0.43[95 %CI:0.14–1.1.36;$p = 0.151$]).

Conclusion: Nearly 50 % of all patients received a transient dose reduction during TAT. Two third of patients experienced a dose reduction of NOACs that could not be justified via applying formal criteria. Therefore, awareness in terms of adequate dosage of NOACs and its duration should be promoted, in order to prevent fatal atherothrombotic and thromboembolic events in these patients.

enrolled in our clinical PREDICT-OAC registry. Duration of TAT was evaluated according medical prescription at time of discharge and during follow-up visits. Patients were followed prospectively until the primary study endpoint (= cardiovascular mortality) was reached.

Results: We identified 264 patients receiving TAT (median age 77 years [IQR: 65–89]; male: 70.5 %) with a median CHA2DS2-VASc-Score of 4 (IQR 3–4). Elective PCI was more frequently observed (65.9 %) than acute coronary syndrome (34.1 %). As expected, the majority of individuals received a NOAC (90.1 %) for TAT as compared to vitamin-K-antagonists (VKA; 9.9 %) and 97.7 % were discharged with the P2Y12 inhibitor clopidogrel. Interestingly, a prolonged dual anti-platelet therapy (DAPT) of longer than one month was found in 37.1 % of all cases, resulting in a median time of TAT of 30 days (IQR: 30–68). Considering the choice of the respective NOAC agent, 39.9 % ($n = 95$) patients received apixaban, 12.6 % ($n = 30$) dabigatran, 20.6 % ($n = 49$) edoxaban and 26.9 % ($n = 64$) rivaroxaban. When applying the recommended respective criteria for dose reduction, 64.3 % of all patients received a reduced therapeutic regimen without any indication for dose reduction during TAT and DAT. Only one out of 26 patients in the VKA group presented with an eGFR <15 ml/h, the major contraindication for NOACs. During a median follow-up time of 19 months (IQR: 12–27) 18 patients experienced the primary study endpoint with 12 individuals receiving apixaban, 3 edoxaban and 3 a VKA respectively. Notably, there was no impact on outcome observed when comparing different NOAC agents with unjustified vs. justified dose reduction (HR 1.12 [95 %CI: 0.57–1.67; $p = 0.838$]).

Conclusion: The present data highlighted that the majority of patient presenting with the indication for TAT received a NOAC agent, as recommended by the latest CCS guidelines. Of utmost importance two third of patients experienced a dose reduction of NOACs that could not be justified via applying formal criteria. Therefore, awareness in terms of adequate dosage of NOACs and its duration should be promoted, in order to prevent fatal atherothrombotic and thromboembolic events in these patients.

PS 10/10-3

Triple antithrombotic therapy after percutaneous coronary intervention from a real-life perspective–duration and dose reduction

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Introduction: The current 2019 ESC guidelines for the management of chronic coronary syndromes (CCS) recommend at least one month of triple antithrombotic therapy (TAT) after percutaneous coronary intervention (PCI) in patients with an indication for oral anticoagulation (OAC) and when the risk of stent thrombosis outweighs the bleeding risk. Dose reduction of NOACs, particularly during TAT, is discussed controversially and CCS guidelines recommend a reduction only when formal reduction criteria apply. We therefore aimed to picture the antithrombotic management and application of dose reduction criteria during TAT from a real-life perspective.

Methods: Patients presenting with the indication for OAC who underwent PCI between 01/2017 and 10/2019 were

PS 10/10-4

Comparability of C-reactive protein and high-sensitive C-reactive protein for cardiovascular risk prediction

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Introduction: CRP is a highly sensitive, though unspecific, biomarker for inflammation. Increased CRP indicates inflammatory processes, but can also be found during chronic inflammation, such as atherosclerosis. The more sensitive high-sensitivity CRP (hs-CRP) is used for risk estimation of patients at risk for cardiovascular diseases with the degree of <0.1 mg/dL for low, 0.1–0.3 mg/dL for average, and >0.3 mg/dL for high risk. CRP is a more commonly available and cost-effective biomarker, when compared to hs-CRP. The aim of this study was to assess the agreement between CRP and hs-CRP measurements, especially in the low detection range.

Methods: 590 consecutive adult patients from 11/2017 to 10/2018 of our cardiology outpatient clinic were retrospectively
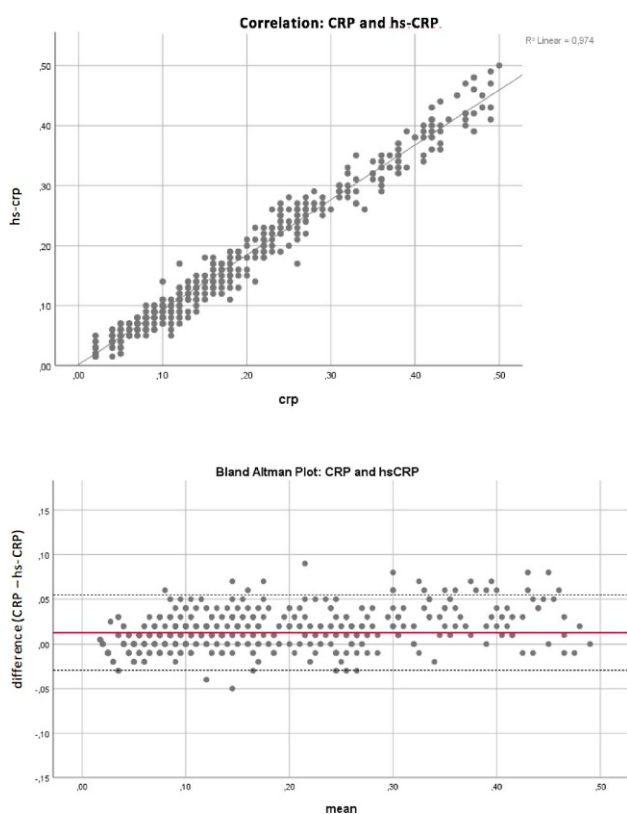
Abb. 1 | PS 10/10-4 Highly significant correlation between CRP and hs-CRP

	n (total 590)	%
male	335	57
hypertension	465	81
diabetes mellitus	104	18
hyperlipidemia	409	70
smoking	136	24
positive family history	148	26
coronary artery disease	251	43
peripheral artery disease	30	5
acute myocardial infarction	118	20
percutaneous coronary intervention	169	29
coronary artery bypass graft	39	7
chronic kidney disease	132	23

Abb. 2 | PS 10/10-4 Baseline data

enrolled in this study. Laboratory values and medical charts were assessed (Tab. 1). In order to compare CRP and hs-CRP values, a linear regression and Bland-Altman plot, with mean plotted against difference (CRP—hs-CRP) were calculated.

Results: The correlation between CRP and hs-CRP was significant ($p < 0.01$), with a regression coefficient of 0.97. Bland-Altman plot displayed a fixed bias with a mean difference between the 2 laboratory tests of 0.01 ± 0.16 SD with only sporadic outliers; hence, the mean difference value is clinically insignificant, not allowing further stratification of the patients (Fig. 1).

Conclusion: Usual laboratory test for CRP values in lower range correlates with the hs-CRP test, therefore can replace the costlier hs-CRP measurements.

PS 10/10-5

Quality of life in patients with stable angina pectoris (sAP) in Austria–Minor differences between patients with or without revascularisation procedures

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Introduction: Revascularisation is a frequently used procedure in patients with coronary artery disease (CAD). In 2018 24.462 Percutaneous Coronary Interventions (PCI) were performed in Austrian clinics. Thereof 14,439 cases (59 %) have been elective non-acute PCIs[1]. Recent studies such as ISCHEMIA[2] re-discuss the benefits of intervention versus optimal medical therapy in CAD patients. Here, we compare in a post-hoc analysis the quality of life of patients with stable angina pectoris in Austria with or without a history of revascularisation procedures.

Methods: Data are taken from the cross-sectional study LENA[3] as recently published. 660 patients with sAP from 70 sites of general practitioner, specialists for internal medicine or outpatient clinics across Austria were included. Data collection was done between September and November 2017 and included demographic data, sAP disease history and quality of life. Patients were stratified for revascularisation (prior PCI or CABG) or non-revascularisation (no PCI or CABG in patient's history). Quality of life was assessed using the patient rated standardised Seattle Angina Questionnaire (SAQ) in German language. The SAQ assesses in 5 domains physical limitations (Part A), angina stability (Part B) and frequency (Part C), treatment satisfaction (Part D) and disease perception (Part E). Comparison were done calculating percentages and using the Mann-Whitney U Test defining significant differences when $p <= 0.05$.

Results: Out of a total of 660 sAP patients, 520 (78.8 %) had undergone prior revascularisation procedures while 140 (21.2 %) had not. Notably, more men were stratified to the revascularisation group (74.5 % versus 25.5 % women) whereas gender distribution was balanced in the non-revascularisation stratum (54.5 % vs. 45.5 %). 54 % of the patients in the revascularisation stratum had a history of myocardial infarction compared to only 28 % in the non-revascularisation group. Median age was significantly lower in the revascularisation stratum compared to the non-revascularisation stratum (70.0 versus 71.5 years; $p = 0.0367$). The only significant difference in the SAQ scoring was observed for the subdomain Part A in favor of the patients in the revascularisation group versus non-revascularisation but not reaching the clinically meaningful difference of eight points: 68.90 vs. 61.84 ($\Delta = -7.05$; $p = 0.0026$). For all other subdomains neither statistically significant nor clinically relevant differences in the SAQ scoring were observed: Part B: 65.46 vs. 65.52 ($\Delta = +0.06$; $p = 0.7916$); Part C: 79.54 vs. 78.63 ($\Delta = -0.90$; $p = 0.7695$); Part D: 86.07 vs. 87.22 ($\Delta = +1.15$; $p = 0.8066$); Part E: 64.14 vs. 62.25 ($\Delta = -1.89$; $p = 0.4182$).

Conclusion: Assessing Austrian real-world sAP patients stratified by prior revascularisation procedure or not with the CAD specific SAQ revealed statistically significant difference on how patients present with physical limitations, but not with angina stability and frequency, treatment satisfaction and disease perception. No clinically meaningful difference was observed for all 5 subdomains. This suggests, that besides revascularisation procedures, pharmacological treatment and

other patient management steps add together for sAP patient's wellbeing.

PS 10/10-6

Treatment of anemic patients with chronic heart failure and chronic kidney disease: criteria of effectiveness

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Introduction: Anemia and renal dysfunction are common comorbid conditions associated with poor prognosis in patients with chronic heart failure (CHF). Purpose: To analyze the predictive value of clinicoanamnestic indicators due to therapy effectiveness of anemia with CHF and CKD using an oral form of Fe(III) hydroxide complex polymaltose for optimization and providing an individual approach to every patient.

Methods: 68 pts with CHF II-IV FC due to IHD and CKD II-III st. were exemined. Among the causes of CKD were: chronic pyelonephritis in 50 pts, diabetic nephropathy in 18 pts. All pts with CHF and CKD had anemia. Hb level was within 78–91 g/l. Diagnosis of anemia was determined by criteria of the Medical Committee of Standards of Hematology (ICST, 1989). CHF FC was established by NYHA. Availability and stage of CKD was determined according to the National Kidney Foundation USA (NKF) K/DOQ classification. Pts with CHF and CKD were treated according to the standards. Pts with anemic syndrome received Fe(III)hydroxide polymaltose complex 100 mg orally 1–2 times a day. Hb target level was within 110–120 g/l. The observation period was 3 months. Evaluation of prognostic properties was performed using non-uniform procedures Wald-Genkina. All signs were distributed by gradient with subsequent calculation of prognostic factors (PF) and the general informative features (I).

Results: To assess the prognostic value of clinicoanamnestic parameters, pts ($n = 68$) that received Fe(III) hydroxide polymaltose complex, at the end of treatment were divided into 2 groups: a) with good antianemic effect ($n = 50$)-achieved the target level of Hb;b) a satisfactory effect ($n = 18$)-Hb levels approach to the target one. Very high informational content (I ≥ 6.0) is given to the duration of CHF(I $= 9.55$), CHF FC (I $= 8.03$),cardiac cachexia syndrome (I $= 7.16$). High predictive value ($6.0 > $I ≥ 1.0) to the severity of anemia (I $= 5.88$),lower extremities edema and dyspnea (I $= 5.60$), acute myocardial infarction (I $= 1.94$),post-infarction left ventricular aneurysm (I $= 2.82$),patient age (I $= 2.50$),severity of CKD (I $= 3.28$) and the presence of type 2 diabetes mellitus (I $= 1.16$). Moderate predictor properties ($1.0 > $I ≥ 0.50) identified in relation to BMI (I $= 0.82$),history of stroke (I $= 0.76$) and the presence of permanent atrial fibrillation (I $= 0.50$).

Conclusion: Clinicoanamnestic indicators revealed a high predictive informational content about the effectiveness of therapeutic correction of anemia with CHF and CKD using an oral form of Fe(III)hydroxide polymaltose complex that allows to include them into predictive algorithms. Most informative criteria: the duration and severity of CHF, cardiac cachexia formation on a background of biventricular cardiac decompensation, progression of renal dysfunction, severity of anemia, which leads to the desirability and feasibility of application of these criteria at all levels of preventive and curative care with the aim of stratification effectiveness of treatment strategies.

PS 10/10-7

High-sensitive CRP as a biomarker for cardiovascular risk prediction

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Introduction: C-reactive protein (CRP) is a commonly used biomarker to assess inflammation. Chronic systemic inflammation is linked with greater development of atherosclerosis, with the possibility of plaque rupture contributing to a common cause for acute MI. Increased levels of high-sensitive CRP (hs-CRP) of above 0.3 mg/dL indicate a high risk for cardiovascular (CV) events in asymptomatic individuals, as well as patients who have recovered from a previous MI. The purpose of this study was to assess the differences in mortality between patients in low, average and high risk hs-CRP groups, to gain a better hs-CRP's predictive value in the clinical setting.

Methods: 590 consecutive adult patients from November 2017 to October 2018 of our cardiology outpatient clinic with coronary artery disease were included in this single-center retrospective cohort study. Laboratory values, medical charts and electronic data were assessed. Patients were divided into hs-CRP risk groups: low <0.1 mg/dL, average 0.1–0.3 mg/dL, and high risk >0.3 mg/dL for CV events. To estimate the effect of hs-CRP on survival, Cox proportional hazards regression was applied, adjusting for the established CV risk factors (age, sex, hypertension, diabetes mellitus, hyperlipidemia, smoking, positive family history) as confounding factors.

Results: Thirty-eight percent of patients ($n = 222$) were classified to low risk, while 34 % to average risk ($n = 200$) and 28 % to high risk groups ($n = 168$) (Table 1). Cumulative survival corrected for covariates showed no significant difference between the low or high risk groups.

Conclusion: Hs-CRP does not appear to discriminate high or low CV risk in a clinical setting in a large patient cohort.

	Low (n=222)	Average (n=200)	High (n=168))	P value
Age; mean (range)	61 (18-86)	66 (23-93)	67 (20-90)	-
Gender male; n (%)	138 (62%)	106 (53%)	91 (54%)	0.09
Hypertension; n (%)	168 (76%)	156 (81%)	141 (86%)	0.03
Diabetes; n (%)	28 (13%)	38 (20%)	38 (23%)	<0.01
Hyperlipidemia; n (%)	158 (72%)	139 (70%)	112 (68%)	0.36
Smoking; n (%)	44 (20%)	45 (23%)	47 (29%)	0.05
Positive family history; n (%)	49 (22%)	55 (29%)	44 (27%)	0.29
Mortality; n (%)	5 (3.2%)	2 (1.5%)	6 (4.5%)	0.57

Abb. 1 | PS 10/10-7 Survival and baseline characteristics of low, average and high hs-CRP risk groups. *P* value was calculated by using ANOVA

POSTERSITZUNG 11 – HERZINSUFFIZIENZ 2

PS 11/11-1

Neuromuscular comorbidity, atrial fibrillation and left bundle branch block predict the prognosis of left ventricular hypertrabeculation/noncompaction

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Introduction: The prognosis of patients with left ventricular hypertrabeculation/noncompaction (LVHT) is controversially assessed. LVHT is frequently associated with neuromuscular disorders (NMDs). Aim of the study was to assess cardiac and neurological findings as predictors of mortality in LVHT-patients.

Methods: Included were patients with LVHT diagnosed between June 1995 and December 2019 in one echocardiographic laboratory. They underwent a baseline cardiologic examination and were invited for a neurological investigation. In January 2020, their survival status was assessed.

Results: LVHT was diagnosed in 310 patients (93 female, aged 53 ± 18 years) with a prevalence of 0.4 %/year. A neurologic investigation was performed in 205 patients (67 %). A specific NMD was found in 33 of the investigated patients (16 %), NMDs of unknown etiology in 123 (60 %) and the neurological investigation was normal in 49 (24 %) patients. During 86 months of follow-up, 59 patients received implanted electronic devices (cardioverter/defibrillator $n = 21$, antibradycardic pacemakers $n = 11$, cardiac resynchronization device/defibrillator $n = 22$, cardiac resynchronization device $n = 4$). During follow-up 105 patients died and 6 patients underwent heart transplantation. The mortality was 4.7 %/year. By multivariate analysis, the following baseline parameters were identified as predictors of mortality: increased age ($p = 0.0005$), inpatient-status when LVHT was diagnosed ($p = 0.0050$), presence of a specific NMD ($p = 0.0187$)

Conclusion: LVHT patients should be systematically investigated neurologically since neurological comorbidity has a prognostic impact. Electrocardiographic abnormalities like
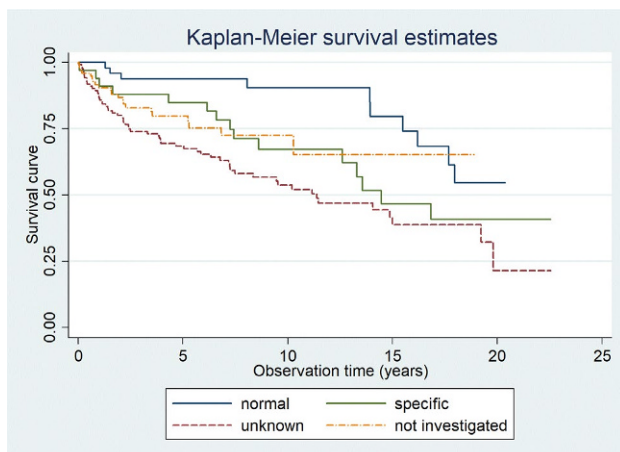
Abb. 1 | PS 11/11-1 Survival curves according to the neurologic findings

atrial fibrillation and left bundle branch block should be considered when planning pharmacotherapy and device-therapy. It has to be assessed by prospective studies, which measures improve the prognosis of LVHT.

PS 11/11-2

Prevalence and determinants of bone disease in patients with chronic heart failure–a prospective cohort study

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Introduction: Chronic heart failure is associated with an increased risk of bone disease, but due to a lack of prospective epidemiological data this comorbidity remains neglected in international guidelines and in clinical practice. We therefore aimed to assess the prevalence and determinants of bone disease in chronic heart failure patients.

Methods: We prospectively enrolled 205 patients with a previous diagnosis of heart failure and left ventricular ejection fraction <50 % at present into a single-center cohort study (ClinicalTrials.gov Identifier: NCT02922478). Further inclusion criteria were stable medication during the last 4 weeks and optimal medical heart failure therapy. Following a pre-specified protocol all patients underwent cardiovascular assessment and (1) dual-energy X-ray absorptiometry (DXA) of distal radius, hip and lumbar spine; (2) X-ray of both the thoracic and the lumbar spine to determine vertebral fracture status defined as Genant Score ≥1; and (3) comprehensive assessment of blood-derived parameters of bone turnover and mineral metabolism.

Results: Patients had a mean ($\pm$ SD) age of 65 ± 10 years and 45 subjects (22 %) were females. Median (IQR) NT-proBNP was 964 (336–2151) pg/ml and LVEF was 35 ± 9 %. Osteoporosis at any site was diagnosed in 31 subjects (15 %), and 36 patients (17 %) had at least one vertebral fracture. Overall, 56 patients (27 %) had manifest bone disease (i.e. osteoporosis or vertebral fracture) requiring specific treatment initiation. Circulating concentrations of parathyroid hormone (PTH) and the bone resorption marker beta-crosslaps (CTX) were elevated in 94 (45 %) and 66 (32 %) of patients, respectively. There was a linear increase of osteoporosis prevalence by increasing NT-proBNP quartiles (P for linear-by-linear = 0.012). Moreover, NT-proBNP was independently related both with PTH (adjusted beta-coefficient = 0.268, $P < 0.001$) and CTX (beta 0.163, $P = 0.039$) in multivariate linear regression analyses after adjustment for age, sex, creatinine, 25-hydroxyvitamin D and plasma calcium.

Conclusion: Bone disease affects approximately one in four patients with chronic heart failure. Moreover, the extent of cardiac congestion is associated with PTH levels, bone turnover and the prevalence of osteoporosis suggesting direct mechanistic links between both disease entities. Screening for bone

disease in routine heart failure care should be advised in heart failure patients.

PS 11/11-3

Etiologies underlying left ventricular hypertrophy – insights from a prospective, single-center screening program

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Introduction: Left ventricular (LV) hypertrophy is a common, often neglected finding in trans-thoracic echocardiography (TTE) and mainly attributed to pressure overload conditions. However, causes of LV hypertrophy, especially in younger patients, are versatile necessitating further clarification.

Methods: Consecutive patients with unexplained interventricular septum thickness (IVS) >15 mm assessed by TTE were screened. Patients with significant, untreated hypertension as well as significant left-sided valvular heart disease were excluded. Eligible patients underwent magnetic resonance imaging, 99mTc-3,3-Diphosphono-1,2-Propanodicarboxylic Acid Scintigraphy, complete serum and urine analysis including electrophoresis and genetic testing for Morbus Fabry.

Results: Between August 2018 and December 2019, 187 patients were included. Median age was 59 years (Interquartile range (IQR) 47–68), 59 (32 %) were female and median IVS was 18 mm (IQR 16–22). After completion of diagnostic workup, a definite diagnosis was made in 116 patients: 70 patients had hypertrophic obstructive cardiomyopathy (HOCM), 32 hypertrophic non-obstructive cardiomyopathy, 5 patients had heart failure with preserved ejection fraction (HFpEF), 4 transthyretin amyloidosis, 2 light-chain amyloidosis and 3 patients were diagnosed with Morbus Fabry. In 71 patients there are outstanding examinations or yet inconclusive results. Patients were treated according to recent guidelines. During follow-up, one male patient with HOCM (40 years) died awaiting heart transplantation and one female HFpEF patient (65 years) with concomitant chronic obstructive pulmonary disease died due to respiratory failure.

Conclusion: Systematic screening of patients with unexplained LV hypertrophy might enable diagnosis of rare entities in early stages facilitating timely administration of appropriate therapies.

PS 11/11-4

NT-pro-BNP in patients with left ventricular hypertrabeculation/noncompaction

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Introduction: Left ventricular hypertrabeculation/noncompaction (LVHT) is a cardiac abnormality of unknown pathogenesis and frequently associated with neuromuscular disorders (NMD). The *N*-terminal fragment of the pro brain natriuretic peptide (NT-pro-BNP) is a marker in heart failure whose relevance in LVHT-patients is unknown. Aim of the study was to assess, if NT-pro-BNP levels are related to clinical parameters and if they are prognostic markers.

Methods: Baseline and follow-up data were collected in a LVHT database, existing since 1996. The levels of NT-pro-BNP of the LVHT patients were collected from a hospital information system and analyzed retrospectively. The endpoints were death and heart transplantation.

Results: In 113 patients (median age 57 years, 76 % male), NT-pro-BNP measurements were found, ranging from 8.0 to 121,152.0 (median 2,029.0) ng/L. Higher NT-pro-BNP levels were associated with heart failure, valve abnormalities, hypertension, angina pectoris, dyspnea, edema, more LVHT-affected ventricular segments (1 vs. 3; $p = 0.034$), larger left ventricular end-diastolic diameter and lower systolic function. NT-pro-BNP levels were not associated with presence or absence of NMD. During a mean follow-up of 6 (range: 0–20) years, 35 % reached an endpoint. A correlation between higher NT-pro-BNP levels and the occurrence of an endpoint was found ($p = 0.000$). By multivariate analysis, predictors for endpoints were increased age ($p < 0.001$), atrial fibrillation ($p < 0.001$) and NT-pro-BNP levels ($p < 0.005$).

Conclusion: High NT-pro-BNP levels are associated with heart failure and systolic dysfunction in LVHT patients. Also in patients with LVHT, NT-pro-BNP levels are prognostic indicators.

PS 11/11-5

Stellenwert der Myokardbiopsie als Puzzlestein der personalisierten Herzinsuffizienztherapie: eine retrospektive Kohortenanalyse an der Kardiologie des Universitätsklinikum Krems

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Einleitung: Die Myokardbiopsie kann, in einem selektierten Patientengut, die zugrundeliegende Pathologie der Herzinsuffizienz (HI) identifizieren und eine therapeutische Konsequenz ergeben. Den Leitlinien der European Society of Cardiology folgend, stellt diese eine Klasse IIa Empfehlung in der Abklärung schnell progredienter Verläufe der Herzinsuffizienz unter laufender Standardtherapie dar. An der Kardiologie des Universitätsklinikum Krems werden Myokardbiopsien nach präziser Aufarbeitung der Krankengeschichte und strikter Indikationsstellung durch die Herzinsuffizienzambulanz durchgeführt. Die vorliegende Datenanalyse betrachtet Biopsieergebnisse, daraus resultierende therapeutische Konsequenzen und Komplikationen an der Kardiologie des Universitätsklinikum Krems.

Methoden: In dieser retrospektiven Kohortenanalyse werden alle Myokardbiopsien an der Kardiologie des Universitätsklinikum Krems ab dem Jahren 2011 dargestellt und analysiert. Demographische Daten der Patienten, Biopsieergebnisse und sich daraus ergebende therapeutische Konsequenzen sowie Komplikationen werden im Folgenden präsentiert. Die Ergebnisse werden in absoluten Zahlen, Mittelwert und Standardabweichung dargestellt.

Resultate: Seit der Implementierung der Herzinsuffizienzambulanz an der Kardiologie des UK Krems im Jahr 2011 wurden 2200 Patienten betreut. In Summe wurden 221 (10 %) dieser

Patienten im Laufe ihrer Erkrankung linksventrikulär myokardbioptiert. Bei der Fragestellung Sarkoidose wurde in 19 Fällen (8,6 %) auch rechtsventrikulär eine Probe entnommen. Das mittlere Alter der Patienten lag bei 60 ± 14 Jahren. 65 (29,4 %) der Patienten waren weiblichen Geschlechts. Die Ergebnisse der Biopsien sind in Tab. 1 dargestellt. Bei 103 (46,6 %) der Patienten ergab sich aus dem Biopsieergebnis eine konkrete therapeutische Konsequenz. In der Subgruppe der inflammatorischen Kardiomyopathie wurde bei 73,1 % eine Therapiemodifikation eingeleitet. In 9 Jahren der Myokardbiopsie am Universitätsklinikum Krems kam es zu insgesamt 8 (3,6 %) direkt biopsieassoziierten Komplikationen. In 5 (62,5 %) Fällen handelte es sich um eine passagere, transitorische Attacke (TIA), in 2 (25 %) Fällen um eine Blutung an der Einstichstelle und in 1 (12,5 %) Fall um Kammerflimmern im Rahmen einer Akut-Biopsie im kardiogenen Schock. Nach erfolgter Biopsie kam es weder zu einer, durch den Herzchirurgen zu sanierenden Komplikationen, noch zu einem Todesfall.

Schlussfolgerungen: Am Beispiel der Daten der Kardiologie am Universitätsklinikum Krems zeigt sich, dass bei präziser Indikationsstellung durch HI-Spezialisten die Myokardbiopsie in mehr als 46 % der Fälle eine konkrete therapeutische Konsequenz ergibt. Es kann überdies gezeigt werden, dass die Komplikationsrate bei regelmäßig durchgeführten Biopsien sehr niedrig gehalten werden kann.
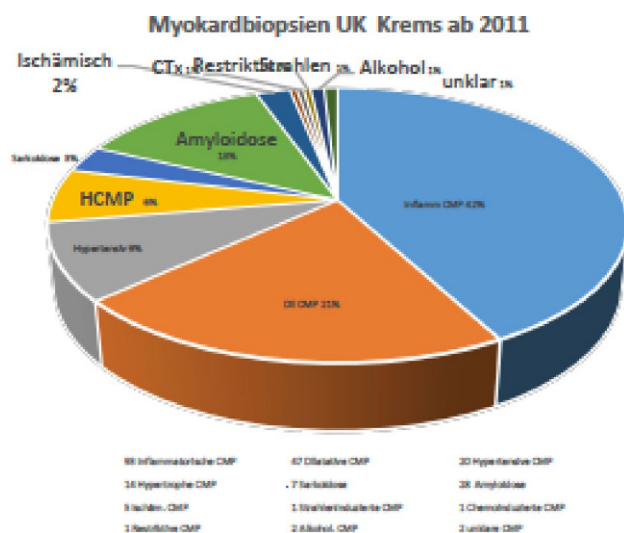
Abb. 1 | PS 11/11-5 Überblick über die Myokardbiopsien

Kardiomyopathie	n (%)	Therapeut. Konsequenz n/n/(%)
inflammat. CMP	93 (42,1)	68 (73)
dilatative CMP	47 (21,3)	
Amyloidose	28 (12,7)	28 (100)
hyperten. CMP	20 (9,0)	
Hypertroph. CMP	14 (6,3)	
Sarkoidose	7 (3,2)	7 (100)
ischäm. CMP	5 (2,3)	
alkohol. CMP	2 (0,9)	
unklare CMP	2 (0,9)	
Chemoinduz. CMP	1 (0,4)	
Restrikt. CMP	1 (0,4)	
Strahleninduz. CMP	1 (0,4)	

Abb. 2 | PS 11/11-5 Überblick über therapeutische Konsequenz der Myokardbiopsie

PS 11/11-6

Psoas muscle area as prognostic marker in patients receiving left ventricular assist device implantation

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Introduction: Several risk scores and classifications are available to predict peri- and post-operative mortality of patients with end stage heart failure receiving Left Ventricular Assist Device (LVAD) implantation. The INTERMACS classification is most commonly used but recently assessment of sarcopenia has been suggested as additional predictor for post-operative outcome. Therefore, we evaluated whether or not psoas muscle area can serve as additional prognostic marker.

Methods: A retrospective analysis on the patient cohort at the General Hospital of Vienna receiving LVAD implantation was performed. Exclusion criteria were age below 18 years and permanent biventricular support. In the time span from January 2014 to April 2019 a total of 178 adult LVAD implantations were performed. Of those, 107 patients received an abdominal CT scan prior to surgery. Psoas mean area at level of the superior endplate of the third lumbar vertebra was measured axially corrected and correlated with patients body surface area, giving the indexed psoas mean area (PMAi). Patients then were divided in high or moderate muscle mass and low muscle mass in accordance to patient sex. The primary end point was death in the first 30 days after LVAD implantation. Other outcomes of interest were length of ICU stay, necessity of temporary RVAD and overall survival. Data were analyzed for a Gaussian distribution and consequently subjected either to parametric tests (t-test or one way ANOVA) or non-parametric tests (Mann-Whitney U Test or Kruskal-Wallis Test) using SPSS software (IBM Version 22). Survival curves were estimated and depicted using the Kaplan–Meier method and tested by the log-rank test.

Results: Estimated survival calculated using the Kaplan–Meier-curve found a significant higher 30-day mortality in patients with low PMAi (log rank: 0.009). Whereas no differences were observed concerning in-hospital, 90-day and 1-year mortality. Lower muscle mass also had no impact on the length of ICU stay or necessity of temporary RVAD. Comparison of the distribution on the INTERMACS classification, revealed that patients with lower muscle mass had a higher probability to classify as INTERMACS level 1 or 2 (Level 1: 21 % vs. 38.1 %; Level 2: 14.8 % vs. 23.8 %).

Conclusion: Patients with low PMAi in our cohort represented the sicker patients and had statistically significant higher short-term mortality. These findings suggest that PMAi can be used as an additional tool for risk stratification in patients undergoing LVAD implantation.

PS 11/11-7

Massive cardiac AL amyloidosis in the absence of monoclonal gammopathy

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Introduction: A 69-year-old female patient was referred to our outpatient clinic for hypertrophic cardiomyopathies due to heart failure of unknown origin. Symptoms included progressive dyspnea with exertion (NYHA III), angina pectoris (CCS I), palpitations, and fatigue. In the clinical examination, congested jugular veins, mild leg edema, and a third heart sound were present. Cardiovascular risk factors comprised smoking (50 PY) and high blood pressure. Medication at the time of admission consisted of a beta blocker, ACE inhibitor, and spironolactone. Furthermore, the patient required thyroid hormone substitution due to a previously performed thyroidectomy.

Methods: Laboratory testing detected elevated levels of both NT-proBNP (1199 pg/mL) and high sensitivity cardiac troponin T (26 pg/mL). Transthoracic echocardiography showed a pronounced concentric bi-ventricular hypertrophy, a left ventricular (LV) ejection fraction of 47 %, diffusely reduced LV global longitudinal strain of 9.3 %, a restrictive transmitral filling pattern with an E/A ratio of 2.18, and a mild pericardial effusion. These echocardiographic findings were indicative of heart failure due to an infiltrative cardiomyopathy.
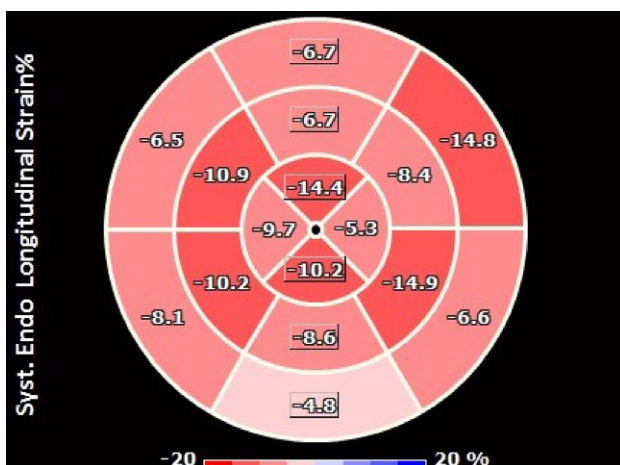
Abb. 1 | PS 11/11-7 LV Longitudinal Strain Bull's Eye
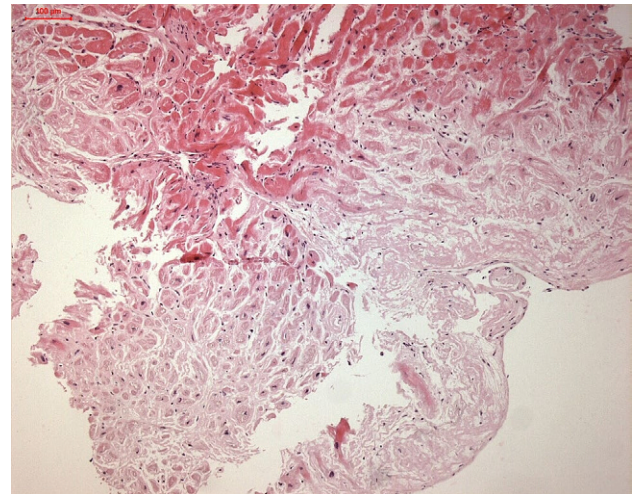
Fig. 2 | PS 11/11-7 EMB-Histology

Results: Native myocardial T1 and extracellular volume mapping by cardiac magnetic resonance imaging revealed increased values for both parameters (1411 ± 83 ms; z-Score = 7 and 69 ± 7 %; z-Score = 15, respectively). Following the growing suspicion of an underlying infiltrative cardiomyopathy, serum and urine immunofixation electrophoresis (IFE) as well as a serum free light chain (sFLC) assay were performed. Despite increased levels of lambda immunoglobulin light chains (30.20 mg/L), the ratio of free kappa to free lambda light chains was not altered (0.55) and IFE could not provide any evidence of present monoclonal paraproteins. 99mTc-DPD bone scintigraphy revealed a faint myocardial radiotracer uptake (Perugini Score = 1). Amyloid was not detectable in abdominal fat pad aspirations and an endomyocardial biopsy was performed. The latter diagnostic method revealed a myocardial amyloid infiltration that was histologically confirmed via congo-red stained tissue demonstrating pathognomonic green birefringence under polarized light on 40 % of the cutting surface. Immunohistochemical typing of amyloid depositions identified lambda immunoglobulin light chains. Furthermore, bone marrow aspiration unveiled normocellular bone marrow without evidence of an underlying multiple myeloma. Subsequent assessment revealed the presence of perivascular amyloid depositions and established the diagnosis of systemic AL amyloidosis with massive myocardial infiltration.

Conclusion: This case illustrates the rare condition of a female patient with AL amyloidosis and massive cardiac infiltration despite an inconclusive search for monoclonal gammopathy in laboratory testing comprising serum and urine IFE as well as a sFLC assay. In contrast to these laboratory analyses, cardiovascular imaging pointed the way towards an infiltrative cardiomyopathy early on. Given the rapid disease progression and its specific treatment options targeting the underlying plasma cell dyscrasia, correct and early diagnosis is crucial. Finally, this case demonstrates the need for a close interdisciplinary cooperation in the diagnosis and management of patients suffering from AL amyloidosis.

POSTERSITZUNG 12 – INTERVENTIONELLE KARDIOLOGIE 1

PS 12/12-1

Vergleich der intraprozeduralen Dynamik der kardialen Reizleitung zwischen zwei selbstexpandierbaren Klappenprothesen bei transfemoralem Aortenklappenersatz

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Einleitung: Bei älteren PatientInnen mit hochgradiger symptomatischer Aortenklappenstenose ist der transfemorale Aortenklappenersatz (TAVI) insbesondere bei erhöhtem Operationsrisiko eine etablierte Therapieoption. Trotz der seit Jahren vorangetriebenen technischen Evolution der am Markt befindlichen Klappenprothesen ist die Rate an postinterventionellen Reizleitungsstörungen, die eine Herzschrittmacherimplantation erfordern können, weiterhin beträchtlich.

Methoden: In einer prospektiven klinischen Studie an unserer Abteilung wurde bei PatientInnen, die sich einer TAVI unterzogen, mittels Platzierung eines His-Katheters über eine Femoralvene die Dynamik der AH-und HV-Zeit sowie der QRS-Dauer zu unterschiedlichen Prozedurzeitpunkten ermittelt. Im Rahmen der statistischen Aufarbeitung der erhobenen Daten wurden die intraprozeduralen Veränderungen der kardialen Reizleitung während TAVI zwischen den selbstexpandierbaren Klappenprothesen Abbott Portico und Medtronic Core Valve Evolut verglichen.

Resultate: Im Zuge der Studie wurden insgesamt 108 PatientInnen (66 Frauen, 42 Männer) mit einem mittleren Alter von 80,1 Jahren (80,5 ♀, 79,6 ♂) eingeschlossen. In der statistischen Analyse imponierte sowohl im Kollektiv jener 35 PatientInnen, die eine Abbott Portico Klappe implantiert bekamen, als auch in der Kohorte jener 73 PatientInnen mit Medtronic Core Valve Evolut Klappen ein signifikanter Anstieg der HV-Zeit und der QRS-Dauer einerseits nach Ballondilatation der nativen Klappe
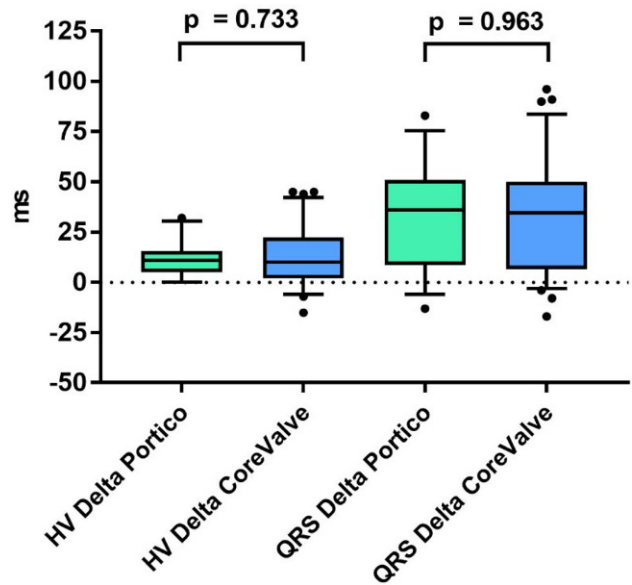
Abb. 1 | PS 12/12-1 Vergleich der Dynamik der HV-Zeit und QRS-Dauer zwischen Abbot Portico und Medtronic Core Valve Evolut Klappenprothesen während transfemoralem Aortenklappenersatz

($p < 0,001$), andererseits nach Implantation der Klappenprothese ($p < 0,001$). Zwischen den beiden Klappentypen zeigte sich jedoch kein signifikanter Unterschied (Delta HV-Zeit und Delta QRS-Dauer zwischen Abbott Portico und Medtronic Core Valve Evolut: $p = 0.733$ bzw. $p = 0,963$). Im Gegensatz zu HV-Zeit und QRS-Dauer zeigte die AH-Zeit keine signifikante intraprozedurale Dynamik ($p = 0.724$ (Portico); $p = 0.398$ (Core Valve)).

Schlussfolgerungen: Die Resultate unserer Studie demonstrieren sowohl bei Abbott Portico- als auch bei Medtronic Core Valve Evolut-Klappenprothesen eine signifikante Beeinträchtigung der infrahisären Reizleitung durch die mechanische Einwirkung am His-Bündel bzw. dem linken Tawara-Schenkel infolge der Ballondilatation und Klappenimplantation. Zwischen den beiden Klappenmodellen besteht hierbei allerdings kein signifikanter Unterschied.

Tab. 1 | PS 12/12-1 Intraprozedurale Veränderungen der kardialen Reizleitung bei Abbott Portico und Medtronic Core Valve Evolut R Klappenprothesen. Die Dauer der AH- und HV-Zeit und des QRS-Komplexes ist in Millisekunden angegeben. Sowohl bei Abbott Portico als auch Medtronic Core Valve Evolut Klappenprothesen zeigte sich verglichen zum Ausgangswert eine signifikante (*=$p < 0,001$) Zunahme der HV-Zeit und der QRS-Dauer sowohl nach Ballondilatation als auch Klappenimplantation

Intraprozedurale EP-Messungen	Abbott Portico	Medtronic Core Valve Evolut	P-Werte
AH Baseline	106,0 ± 31,1	101,2 ± 31,0	
AH Post Ballondilatation	103,0 ± 30,6	99,6 ± 27,2	
AH Post Klappenimplantation	103,9 ± 29,6	103,7 ± 28,8	
AH Delta	−1,6 ± 13,6	2,5 ± 17,8	$P = 0,502$
HV Baseline	56,6 ± 13,7	55,1 ± 12,2	
HV Post Ballondilatation	63,8 ± 15,1 *	62,8 ± 14,0 *	
HV Post Klappenimplantation	70,4 ± 18,0 *	67,1 ± 13,3 *	
HV Delta	12,5 ± 13,9	12,1 ± 9,3	$p = 0,733$
QRS Baseline	108,5 ± 26,5	100,7 ± 30,1	
QRS Post Ballondilatation	123,8 ± 32,8 *	112,6 ± 28,3 *	
QRS Post Klappenimplantation	140,8 ± 32,1 *	130,4 ± 31,2 *	
QRS Delta	32,7 ± 26,2	32,6 ± 23,6	$p = 0,963$

PS 12/12-2

Gender-spezifische Aspekte der Veränderung der kardialen Reizleitung während transfemoralem Aortenklappenersatz

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Einleitung: Der transfemorale Aortenklappersatz (TAVI) gilt seit Jahren als etablierte Behandlungsoption bei Patientinnen und Patienten mit hochgradiger Aortenklappenstenose. Trotz der seit Jahren vorangetriebenen technischen Evolution der am Markt befindlichen Klappenprothesen ist die Rate an postinterventionellen Reizleitungsstörungen, die eine Herzschrittmacherimplantation erfordern, weiterhin beträchtlich.

Methoden: In einer prospektiven klinischen Studie an unserer Abteilung wurde bei PatientInnen, die sich einer TAVI unterzogen, mittels Platzierung eines His-Katheters über eine Femoralvene die Dynamik der AH- und HV-Zeit sowie der QRS-Dauer zu unterschiedlichen Prozedurzeitpunkten ermittelt. Im Rahmen der statistischen Aufarbeitung der erhobenen Daten wurden die Gender-spezifischen Unterschiede in der Veränderung der kardialen Reizleitung während TAVI analysiert.

Resultate: Insgesamt wurden 108 PatientInnen (66 Frauen, 42 Männer) mit einem mittleren Alter von 80,1 Jahren (80,5 ♀, 79,6 ♂) in die Studie eingeschlossen. Die statistische Aufarbeitung der erhobenen Parameter zeigte sowohl bei Frauen als auch bei Männern einen signifikanten Anstieg der HV-Zeit sowie der QRS-Dauer unmittelbar nach Ballondilatation ($p < 0,001$) sowie nach Implantation der Klappenprothese ($p < 0,001$), wobei die AH-Zeit weitgehend konstant blieb ($p = 0.789$ ♀ bzw. $p = 0.734$ ♂). Während sich im weiblichen Kollektiv durchwegs kürzere HV-Zeiten fanden, so zeigte sich bei beiden Geschlechtern ein vergleichbarer Anstieg der HV-Zeit und QRS-Dauer (Abb. 1, Tab. 1). Auch in der Rate an postinterventionell vorliegenden kompletten AV-Blöcken und Linksschenkelblöcken gab es keinen signifikanten Unterschied zwischen den Geschlechtern.

Schlussfolgerungen: Die Analyse unserer erhobenen Daten zeigt eine signifikante Beeinträchtigung der infrahisären
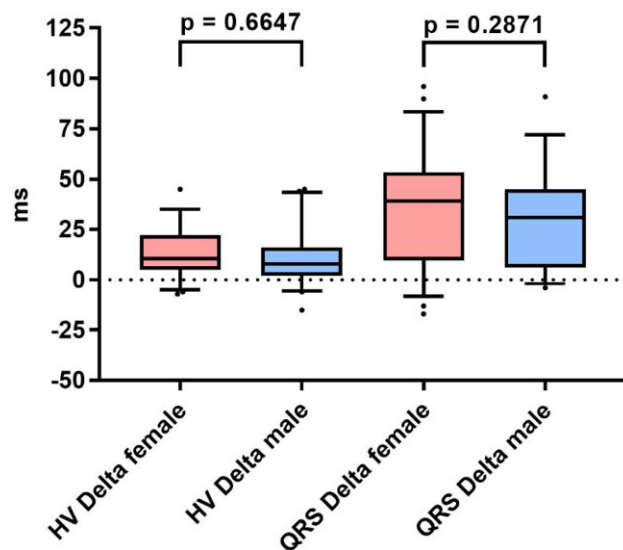
Abb. 1 | PS 12/12-2 Vergleich der Dynamik der HV-Zeit und QRS-Dauer zwischen Frauen und Männern während transfemoralem Aortenklappenersatz.

Reizleitung durch die mechanische Einwirkung am His-Bündel bzw. dem linken Tawara-Schenkel infolge der Ballondilatation und Klappenimplantation. Diese tritt bei Frauen und Männern in einem vergleichbaren Ausmaß auf. Ob es klinisch relevante Prädiktoren für das Auftreten signifikanter Reizleitungsstörungen gibt, wird im Zuge weiterer Analysen evaluiert.

PS 12/12-3

Feasibility of percutaneous transcatheter edge-to-edge repair of severe tricuspid regurgitation with off-label use of the MitraClip-system

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Tab. 1 | PS 12/12-2 Intraprozedurale Veränderungen der kardialen Reizleitung bei Männern und Frauen. Die Dauer der AH- und HV-Zeit und des QRS-Komplexes ist in Millisekunden angegeben. Sowohl bei Frauen als auch bei Männern zeigte sich verglichen zum Ausgangswert eine signifikante (*=$p < 0,001$) Zunahme der HV-Zeit und der QRS-Dauer sowohl nach Ballondilatation als auch Klappenimplantation

Intraprozedurale EP-Messungen	Frauen	Männer	P-Werte
AH Baseline	100,5 ± 29,2	110,6 ± 33,1	
AH Post Ballondilatation	98,0 ± 24,4	108,0 ± 35,6	
AH Post Klappenimplantation	100,4 ± 25,7	108,8 ± 33,4	
AH Delta	0,9 ± 16,0	−1,9 ± 14,0	$P = 0,9713$
HV Baseline	53,7 ± 13,2	60,0 ± 12,3	
HV Post Ballondilatation	61,1 ± 15,1 *	67,3 ± 13,4 *	
HV Post Klappenimplantation	66,1 ± 15,2 *	73,7 ± 17,5 *	
HV Delta	12,6 ± 11,7	12,0 ± 13,6	$p = 0,6647$
QRS Baseline	103,7 ± 29,4	109,5 ± 25,0	
QRS Post Ballondilatation	119,4 ± 34,2 *	121,2 ± 27,6 *	
QRS Post Klappenimplantation	137,7 ± 32,5 *	139,3 ± 28,9 *	
QRS Delta	34,7 ± 25,9	29,8 ± 24,3	$p = 0,2871$

Introduction: Percutaneous transcatheter edge-to-edge repair of severe tricuspid regurgitation (TR) with off-label use of the MitraClip-system has shown promising results. We herewith report the early experience of applying this technique in our center in patients with severe functional TR.

Methods: Patients with symptomatic severe TR were screened for the eligibility for transcatheter treatment and discussed in the heart team over a period of one year. We identified three patients with isolated severe TR who were considered at increased surgical risk (mean EuroSCORE II 6.65 %) and whose anatomy was deemed suitable for transcatheter repair. All patients suffered from severe right-sided heart failure (NYHA class III). One of three had previously undergone transcatheter MitraClip-treatment for severe mitral regurgitation, and two of three had a history of surgical mitral valve replacement. Applying a modified steering technique for the clip delivery system we followed the same strategy in all three patients respecting the pathomechanism of the TR jet with oval- to crescent-shaped EROAs reaching from the antero-septal commissure along the free side of the septal leaflet to the postero-septal commissure: placing a first MitraClip (XTR) between the septal and anterior leaflet, followed by a second clip (XTR) between the septal and posterior leaflet. A total of six clips (two per patient) were placed into the tricuspid valve apparatus.

Results: Procedural success was achieved in all patients. The TR grade was reduced from severe to mild in all patients. No major adverse events occurred. Short- and medium-term follow-up showed persisting good outcomes in terms of reduction of TR grade (mild insufficiencies in all three patients) and improvement in symptoms (NYHA class I in one patient, class II in two patients).

Conclusion: Percutaneous transcatheter edge-to-edge repair of severe TR with off-label use of the MitraClip-system is feasible in highly selected patients with a suitable anatomy and a strategy to first clip the septal-anterior commissure followed by the septal-posterior commissure.

PS 12/12-4

Intensivaufenthalt nach erfolgtem transfemoralen Aortenklappenersatz – eine monozentrische Analyse

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Tab. 1 | PS 12/12-4

	Dauer Intensivaufenthalt (Tage)	Body Mass Index (kg/m2)	Alter	Glomeruläre Filtrationsrate (ml/min)
Mittelwert	6,7	26,8	80,4	54,5
Maximum	24	46	94	179,6
Minimum	1	17	42	4,18
Median	7	26	81	50,6

Tab. 2 | PS 12/12-4

	Korrelation mit Aufenthaltsdauer Intensivstation (Tage)
Body Mass Index (kg/m2)	−0,015
Alter (Jahre)	0,127
Glomeruläre Filtrationsrate (ml/min)	−0,069

Einleitung: Die transfemorale Aortenklappenimplantation (TAVI) stellt eine schonende und nachhaltige Therapie für ältere, gebrechliche und multimorbide Patienten, die an einer symptomatischen Aortenklappenstenose leiden, dar. Nach erfolgter Implantation werden die PatientInnen standardmäßig auf der Intensivstation hämodynamisch und rhythmologisch monitiert. Derzeit besteht am Standort keine Möglichkeit einer telemetrischen Überwachung der Patienten auf der Normalstation, weswegen ein Aufenthalt auf der Intensivstation bis zur Entscheidung einer etwaigen Schrittmacherimplantation stattfindet. Bis dato gibt es keine klinischen Marker, welche die Dauer des individuell notwendigen Intensivaufenthaltes prognostizieren können. Wir untersuchten PatientInnen, welche von Jänner 2016 bis November 2019 eine TAVI an unserer Klinik erhielten, auf etwaige Korrelationen bezüglich klinischer und laborchemischer Einflussgrößen auf die Dauer des Intensivaufenthaltes

Methoden: Folgende Parameter wurden mit der Dauer des Intensivstationsaufenthaltes nach transfemoralen Aortenklappenersatz korreliert: (siehe auch Tab. 1) • Alter • Body Mass Index • Glomeruläre Filtrationsrate

Resultate: Insgesamt 233 Patientin konnten untersucht werden (123 Frauen, mittleres Alter 80,4 Jahre). Die mittlere Aufenthaltsdauer auf der Intensivstation betrug 6,7 Tage, bei einem medianen Body Mass Index von 26 kg/m2, und einer mittleren glomerulären Filtrationsrate von 54,5 ml/min. In den Analysen konnte keine wesentliche Korrelation bezüglich der untersuchten Parameter gefunden werden. In unserer Analyse hatte das Alter die noch höchste positive Korrelation.

Schlussfolgerungen: Prognostische Faktoren für die Dauer des Intensivaufenthaltes nach transfemoralen Aortenklappenersatz wären aus logistischen aber auch ökonomischen Gründen sehr wertvoll. Oftmals spielen vor allem organisatorische Gründe eine bedeutende Rolle bei der Entscheidung ob einer Verlegung eines Patienten auf die Normalstation. Eine telemetrische Überwachung auf der Normalstation für eine frühere Verlegung auf die Normalstation wäre in diesen Fällen wünschenswert. Weitere Untersuchungen bezüglich diesen Aspekts sind wünschenswert und notwendig.

PS 12/12-5

A routine nursing questionnaire adds predictive value to conventional risk scores for TAVI (Transcatheter Aortic Valve Implantation) outcome

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Introduction: Transcatheter aortic valve implantation (TAVI) is an established treatment method for patients with severe aortic valve stenosis (AS) and high surgical risk. Frailty is defined by the increased vulnerability to stress and decrease in
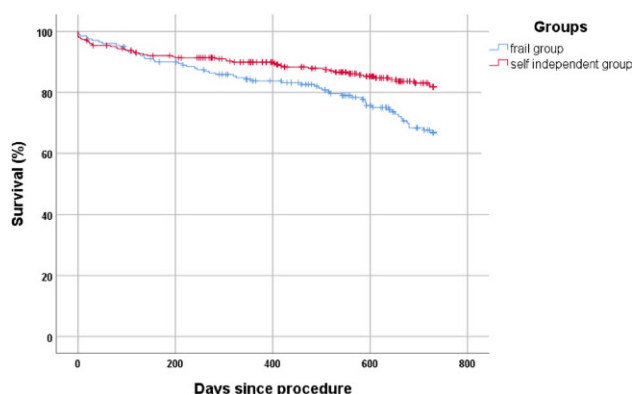
Fig. 1 | PS 12/12-5 Kaplan Meier

Table 1 *Multivariate analysis with forced consideration of age and left ventricle function, remaining values forward stepwise with $p_{in} = 0.05$; $p_{out} = 0.10$*

Parameter	HR (95% CI)	p
Alter (pro Jahr)	0.970 (0.938-1,002)	0.069
EF 30-50%	0.648 (0.381-1.102)	0,277
EF 20-30%	1.511 (0.594-3.846)	0.126
EF < 20%	1.349 (0.625-2.910)	0.127
BMI (per kg/m²)	0.941 (0.896-0.988)	0.015
AF	1.658 (1.104-2.490)	0.015
Frailty	1.705 (1.128-2,578)	0.011
STS Score	1.038 (1.053-1.230)	0.001
gender	1,663 (1,095 − 2,524)	0.017
neurological disease	0,453 (0,215 − 0,953)	0,037
TIA	2,338 (1,113 − 4,911)	0,025
Euroscore II	0,945 (0,897 − 0,997)	0,038

Table 2 *Multivariate analysis with forced consideration of risk scores*

Parameter	HR (95% CI)	p
STS Score	1.098 (1,021-1.181)	0.012
AF	2.016 (1.316-3.089)	0.001
Frailty	1.550 (1.010-2.380)	0.045
gender	1,653 (1,100 − 2,484)	0,016
liver cirrhosis	4,822 (1,104 − 21,059)	0,036
neurological disease	0,380 (0,178 -0,811)	0,012
TIA	2,355 (1,125 − 4,925)	0,023

Abb. 2 | PS 12/12-5 Multivariate analysis

physiological body functions and reserves and can be assessed by an Frailty assessment. Previous studies indicate that frailty is associated with poor outcome following TAVI. Purpose: This study aimed to investigate whether a questionnaire routinely administered by nurses might serve as a surrogate for frailty and predict outcome in TAVI patients in addition to conventional risk scores.

Methods: This is a retrospective single-centre study including 567 consecutive patients (age 82 ±6 years, 59.3 % female) scheduled for TAVI between 2012 and 2017. Based on seven questions addressing levels of patients' self-dependence assessed by nurses on admission, TAVI patients were divided into a "frail" group (at least one answer indicating limited self-dependence) and a self-dependent group (all other patients). We sought to assess (1) prevalence of frail TAVI patients, and (2) impact of frailty on two-year mortality assessed by Cox regression in addition to established risk scores.

Results: The prevalence of frail TAVI patients was 40.2 %. Frail patients had significantly higher two-year mortality than self-dependent patients (28.4 % vs 15.5 %%, $p = 0.002$, Figure). The procedural complication rate was not affected by self-dependency (45.6 % vs 42.8 %, $p = 0.422$). In multivariate analysis stratified for age and left ventricular function, frailty, body mass index (BMI), atrial fibrillation (AF), gender, STS Score, Euroscore II, neurological diseases and transient ischemic attack (TIA) were significant predictors of increased two-year mortality (frailty: HR 1.71 [1.13–2.58], $p = 0.011$). Stratified for the Society of Thoracic Surgeons (STS) risk of mortality score, frailty, AF, gender, neurological diseases, liver cirrhosis and TIA significantly predicted increased two-year mortality (frailty: HR 1.55 [1.01–2.38], $p = 0.045$).

Conclusion: This study shows that a routine nurses' questionnaire covering levels of self-dependence serves as risk indicator for long-term mortality after TAVI. This geriatric assessment adds predictive power for two-year mortality to conventional risk scores (such as STS) and might be used to stratify patients for greatest benefit from TAVI.

PS 12/12-6

Which case of left atrial appendage closure (LAAC) will become difficult? Predictors for complications in the Austrian LAAC Registry

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Introduction: Left atrial appendage closure (LAAC) is an established treatment option for patients with atrial fibrillation and contraindication of oral anticoagulation. However, a considerable proportion of patients experience major complications during implantation.

Methods: This is a retrospective analysis of the national multicentre Austrian LAAC Registry, including consecutive patients of all 9 centres which currently perform LAAC interventions in Austria. Primary endpoint was the occurrence of any severe periprocedural complication requiring intervention, failure to implant LAAC or death. Bivariable and multivariable analyses were performed to find predictors for complications, using logistic regression analysis ($p < 0.1$ for entry).

Results: Between 2010 and 2019, 244 patients received LAAC in Austria. Most patients were male (64.3 %) and median age was 70 (interquartile range, 70–79) years. The primary endpoint occurred in 9.0 % of cases, including pericardial tamponade (4.5 %), failure to implant the LAAC device (2.9 %), pseudoaneurysma (2.0 %), stroke (0.8 %), thrombus (0.8 %), embolization (0.4 %) and death (0.4 %). Abnormal liver function, chronic obstructive pulmonary disease, low haemoglobin and absence of any history of bleeding were associated with higher complication rate in bivariable analysis ($p < 0.1$ for all).

Predictors for periprocedual complications
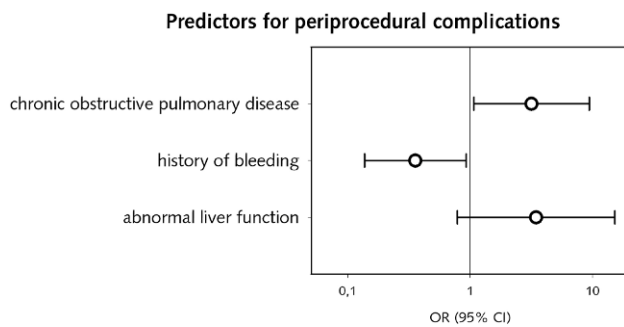
Fig. 1 | PS 12/12-6 Predictors for periprocedual complication

In multivariable analysis, absence of bleeding (OR 0.36 [95 % CI 0.14–0.93], $p = 0.03$) and chronic obstructive pulmonary disease (OR 3.17 [1.07–9.37], $p = 0.04$) remained significant predictors (figure). Age ($p = 0.30$), gender ($p = 0.36$), CHA2DS2-VASc ($p = 0.96$) and HAS-BLED ($p = 0.81$) scores, as well as centre experience ($p = 0.73$), were not associated with higher complication rates.

Conclusion: Among patients undergoing LAAC, the occurrence of peri-interventional complications may not be determined by classical risk scores. The absence of history of bleeding and chronic obstructive pulmonary disease may be associated with higher complication rate.

PS 12/12-7

Einfluss des SYNTAX-Score und des SYNTAX II-Scores auf das Langzeit-Outcome nach interventioneller ungeschützter Hauptstammintervention – ein retrospektives Langzeit Follow-up

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Einleitung: Die Festlegung der optimalen Revaskularisationsstrategie bei Patienten mit koronarer Herzkrankheit, insbesondere mit Hauptstammbeteiligung, bleibt nach wie vor eine Herausforderung in der täglichen klinischen Routine. Die aktuellen Leitlinien empfehlen die Miteinbeziehung des SYNTAX-Scores und anderer Scores zur Entscheidungsfindung. Gleichzeitig sehen die Guidelines Evidenzlücken und fordern mehr Daten bezüglich der Verwendung von Scores vor allem in der Subgruppe von Patienten mit Hauptstammstenosen.

Methoden: Wir haben Patienten aus dem UNPROLEMA (UNPROtected LEft MAin disease) Register an unserer Abteilung untersucht, bei denen eine ungeschützte Hauptstammintervention durchgeführt wurde, und dabei den rein koronarmorphologischen SYNTAX Score mit dem SYNTAX II Score verglichen, der auch klinische Variablen mit einbezieht. Dabei wurden die Patientendaten durch Informationen aus Krankengeschichten sowie aus strukturierten Telefon-Interviews und Meldeamtsanfragen bzgl. Follow-up komplettiert. Die Patienten wurden gemäß Syntax-Score in drei Gruppe mit niedrigem (0–22), mittlerem (23–32) und hohem (>32) unterteilt. Des Weiteren erfolgte eine Gruppierung nach den SYNTAX II Score Terzilen: untere Terzile Score 0–31,7, intermediäre Ter-

zile 31,8–43,2, sowie obere Terzile >43,2. Die Gesamtmortalität und das Auftreten von major adverse cardiac and cerebrovascular events (MACCE: definiert als Myokardinfarkt, Zielgefäßrevaskularisation [interventionell oder operativ], Insult/TIA oder Tod jedweder Genese) wurden mittels Kaplan-Meier Kurven für beide Unterteilungen analysiert. Ein Log-rank Test wurde zur Prüfung der statistischen Signifikanz durchgeführt. Zudem wurde mittels Cox Proportional Hazards Modell für potentielle Confounder adjustiert. Die prädiktive Güte der beiden Modelle (SYNTAX Score bzw. SYNTAX II Score) wurde anhand des Akaike Information Ciriterion (AIC) weiter untersucht. Kleinere Werte des AIC im Vergleich sprechen dabei für eine bessere Güte des Vorhersagemodells.

Resultate: Im Untersuchungszeitraum wurde bei 293 Patienten eine ungeschützte Hauptstammintervention durchgeführt. Davon wurden 136 Patienten der Gruppe mit niedrigem (46 %), 109 der Gruppe mit mittlerem (37 %) und 48 Patienten der Gruppe mit hohem SYNTAX Score (16 %) zugeordnet. Die Terzilen des SYNTAX II Scores umfassten 92 (untere), 100 (intermediäre) und 96 Patienten (obere). Zwischen den anhand des SYNTAX-Scores unterteilten Gruppen zeigte sich bei einer medianen Follow-up-Dauer von 3,4 Jahren (IQR 1,4–6,3; Spannweite 0–12,1 Jahre) in der Kaplan-Meier-Analyse der Todesfälle jedweder Ursache kein signifikanter Unterschied (Log-rank Test $p = 0,982$; HR 0,98 (0,73–1,31), AIC 855). Nach Adjustierung für potenzielle Confounder betrug die HR 0,78 (0,57–1,08, $p = 0,141$). Ebenso zeigte die Analyse der MACCE keine statistische Signifikanz (Log-rank $p = 0,883$; HR 0,97 (0,76–1,24), AIC 1257). Nach Adjustierung blieb die HR 0,93 (0,71–1,23, $p = 0,621$) im Wesentlichen unverändert. Im Gegensatz dazu, zeigte sich beim Vergleich der Terzilen des SYNTAX II-Scores ein signifikanter Unterschied bezüglich der Mortalität (Log-rank $p < 0,001$; HR 2,49 (1,85–3,37), AIC 811). Nach Adjustierung für Confounder, die im SYNTAX II Score nicht inkludiert sind, veränderte sich die HR mit 2,44 (1,79–3,33, $p < 0,001$) kaum. In der Analyse der MACCE konnte auch eine statistische Signifikanz beobachtet werden: Log-rank $p = 0,011$; HR 1,34 (1,07–1,66), AIC 1250, bzw. HR nach Adjustierung 1,30 (1,03–1,63), $p = 0.024$.

Schlussfolgerungen: Der SYNTAX II Score konnte die Mortalität und das Auftreten von MACCE während des Langzeit-Follow-ups in unserer single-center Kohorte besser vorhersagen als der rein anatomische SYNTAX Score. Die Mitbetrachtung klinischer Variablen neben der rein anatomischen Koronarmorphologie bei der Entscheidung PCI vs. CABG bei ungeschützter Hauptstamm-Stenose ist daher aus unserer Sicht essentiell.

PS 12/12-8

Echocardiographic changes after percutaneous edge-to-edge mitral valve repair

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Introduction: Previous studies examining echocardiographic changes in patients undergoing percutaneous edge-to-edge mitral valve repair (pMVR) show discrepant results regarding the efficacy of the intervention. We aimed to investigate

changes in echocardiographic parameters, routine biomarkers, and clinical presentation after pMVR.

Methods: We prospectively enrolled consecutive patients with severe functional (FMR) or degenerative mitral regurgitation (DMR) scheduled for pMVR. Transthoracic echocardiography and assessment of clinical and laboratory parameters were performed prior to and 4.0 ± 2.6 months following intervention.

Results: In total, 97 patients (75.6 ± 8.8 years; 59% female) were included, 64 (66%) presented with FMR. At baseline, FMR was associated with worse left (LVEF: 39.2 vs. 49.5%, $p = <0.001$) and right ventricular ejection fraction (RVEF: 50.2 vs. 51.9%; $p = <0.001$), and higher NT-proBNP serum levels (7229 vs. 5097 pg/mL; $p = 0.03$), when compared to DMR. Following pMVR, LVEF significantly improved (LVEF: 41.1 to 43.7%; $p = 0.012$), however changes were only significant in the FMR subgroup (ΔLVEF: 3.4 vs. 0.9%). Moreover, tricuspid regurgitation (TR) severity declined after pMVR (TR $\geq$II: 52 to 41%; $p = 0.003$). Furthermore, right atrial size (RA: 63.3 to 61.3 mm; $p = 0.016$) declined, while RVEF and RV size remained unchanged ($p = 0.377$ and $p = 0.964$). Finally, NT-proBNP serum levels tended to decrease (6661 to 5539 pg/mL; $p = 0.161$), and NYHA functional status improved (NYHA $\geq$III: 83 to 21%; $p = 0.100$)–however, not significantly.

Conclusion: LVEF significantly improves after pMVR, with more pronounced changes in the FMR subgroup. In addition, a significant decrease in TR severity was observed at 4 months follow-up.

PS 12/12-9

Anhaltende Angina pectoris nach Stent-Unterexpansion im Bereich eines chronischen Koronarverschlusses

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Einleitung: Die Folgen einer perkutanen Koronarintervention können vielseitig sein. Neben symptomatischer Besserung kann es auch zu anhaltender Angina pectoris kommen. Eine der möglichen Ursachen für diese ist eine Unterexpansion des implantierten Stents, wie unser Fall zeigt.

Methoden: Eine 72-jährige Frau mit einer Vorgeschichte von gut eingestellter arterieller Hypertonie, Hyperlipidämie und einer positiven Familienanamnese präsentierte sich mit einer anhaltenden typischen Angina pectoris nach Eröffnung einer chronisch verschlossenen LAD vor 6 Monaten. Einige Monate zuvor wurde die Patientin stationär aufgenommen, nachdem sie in einem Belastungs-EKG negative T-Wellen im Bereich der Vorderwand entwickelt hatte und eine Episode instabiler Angina pectoris erlitt. In der Akut-Koronarangiographie fand sich eine Drei-Gefäßerkrankung, wobei die LAD chronisch verschlossen war und durch Kollateralen ausgehend vom Ramus circumflexus versorgt wurde. In einer Lävokardiographie zeigte sich eine apikale Akinesie, während in einer transthorakalen Echokardiographie keine regionalen Wandbewegungsstörungen festgestellt werden konnten. Die chronisch verschlossene LAD wurde als culprit lesion interpretiert. Nachdem eine Myokardszintigraphie eine erhaltene Vitalität der Vorderwand zeigte und eine optimale medikamentöse Therapie keine Beschwerdefreiheit erbrachte, wurde auf die Eröffnung der chronischen Läsion mittels perkutaner Koronarintervention entschieden.

Resultate: Die Drahtpassage mit einem weichen Führungsdraht war problemlos möglich. Nach Vordilatation und intrakoronarer Gabe von Nitroglycerin wurden zwei Drug-eluting Stents mit einem Durchmesser von 2,5 mm und 2,75 mm, sowie einer Gesamtlänge von 48 mm implantiert. Weiters wurde die signifikante Läsion des Ramus circumflexus erfolgreich mittels Implantation zweier Drug-eluting Stents interveniert. Die abschliessende Angiographie zeigte ein sehr gutes Ergebnis mit TIMI 3 Fluss, sodass auf eine Kontrolle mittels optischer Kohärenztomographie (OCT) verzichtet wurde. Nur wenige Monate nach diesem Eingriff wurde die Patientin wegen auftretender typischer Angina pectoris wieder vorstellig und deshalb eine Re-Koronarangiographie durchgeführt. Auf den ersten Blick zeigte sich in der LAD ein guter Blutfluss (TIMI III) und keine sichtbaren Stenosen. Aufgrund der anhaltenden Angina pectoris wurde eine OCT durchgeführt, welche eine Malapposition, sowie Unterexpansion der Stent-Struts zeigte. Nach Optimierung der Stents betrug die minimal lumen area (MLA) statt anfangs 4,40 mm2, nun 6,42 mm2 und war an den Gefäßdurchmesser angepasst, wie eine Kontroll-OCT abschliessend zeigte (Abb. 1 und 2). Die Patientin beschrieb einen deutlichen Rückgang der Angina mit Verbesserung ihrer Lebensqualität.

Schlussfolgerungen: Im vorliegenden Fall wurde nach strenger Indikationsstellung die PCI des chronischen Verschlusses für eine perkutane Intervention zur Wiedereröffnung entschieden. Diese gelang problemlos und führte zu einem visuell guten Ergebnis mit TIMI III Fluss. Bei der Intervention kam es zu einer unentdeckten geringfügigen Malapposition bzw. Unterexpansion des Stents. Diese kann durch rein angiographische Beurteilung leicht übersehen werden, wie in unserem Fall gezeigt wurde. Intravaskuläre Bildgebung wie OCT oder intra-

Abb. 1 | PS 12/12-9 Die Re-Koronarangiographie zeigt (A) eine insignifikante Stenose der LAD im Bereich der bereits inserierten Stents. Eine OCT-Aufnahme (Abb. 2) zeigte eine Unterexpansion, sowie Malapposition, welche mittels Dilatation interveniert wurde. (B) zeigt eine Reduktion der Stenose
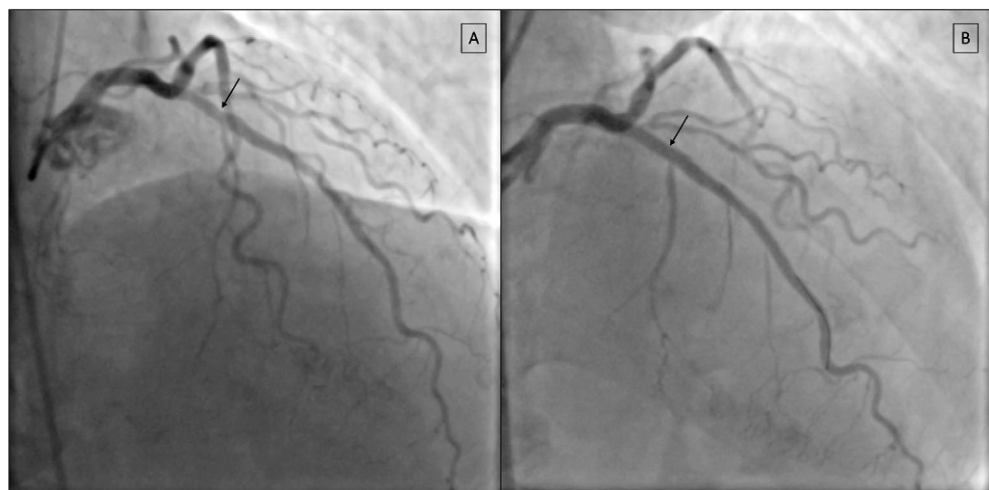
Abb. 2 | PS 12/12-9
OCT-Aufnahmen während der Re-Koronarangiographie zeigen (A) den unterexpandierten Stent mit einer minimal Lumen Area (MLA) von 4,40 mm2 (entsprechend der C-Area in Rosa), während (B) den nachdilatierten Stent mit einer MLA von 6,41 mm2 zeigt. Ebenso zu sehen ist eine Malapposition des Stents in den Längen B (blau) und E (gelb) mit einem Abstand von 0,37 mm und 0,38 mm von der Gefäßintima
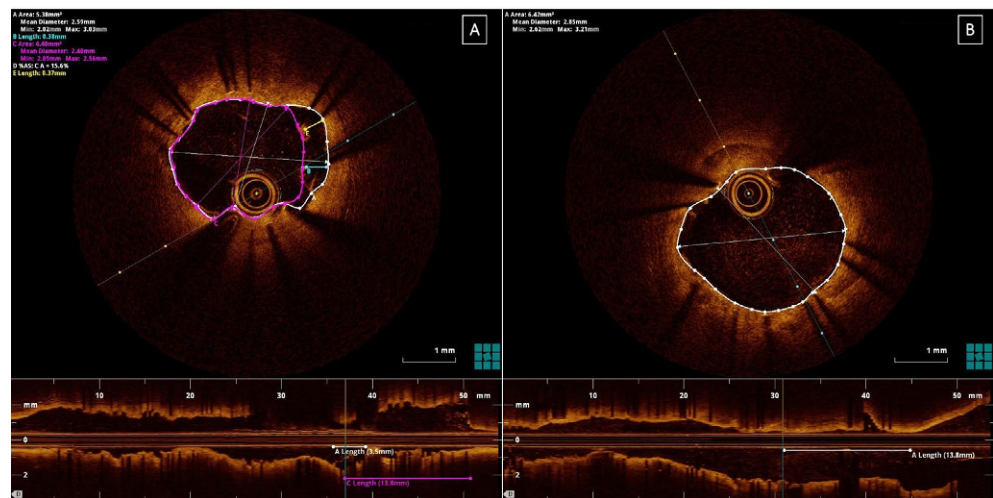
vaskulärer Ultraschall (IVUS) können dabei eine große Unterstützung sein. In den letzten Guidelines [1] wurde die Anwendung dieser beiden Methoden bereits auf eine Klasse IIa, Level B Empfehlung angehoben, um ein optimales Stent Ergebnis zu erzielen. In diesem Fall konnte mittels Behebung der Malapposition und Unterexpansion der Stents die Lebensqualität einer Frau deutlich verbessert werden, was für den breiteren Einsatz von OCT und IVUS in diesen speziellen Indikationen, wie durch die ESC Guidelines empfohlen, spricht.

POSTERSITZUNG 13 – PULMONALE HYPERTENSION 1

PS 13/13-1

Oral Anticoagulation in patients with CTEPH undergoing balloon pulmonary angioplasty

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Introduction: Chronic thromboembolic pulmonary hypertension (CTEPH) is a pulmonary vascular disease caused by chronic obstruction of major pulmonary arteries. While the mainstay of treatment is Pulmonary Endarterectomy (PEA), Balloon Pulmonary Angioplasty BPA is an emerging treatment option that improves survival for inoperable patients and for those with recurrent pulmonary hypertension after PEA. No data exist regarding the effect and the safety of direct oral anticoagulants (DOACs) in CTEPH patients undergoing BPA. Aim: To assess the impact of the type of oral anticoagulation (OAC) on the effectiveness of BPA and survival.

Methods: Data of 112 patients who underwent BPA in our institution between 2014–2019 were analysed. 93 patients received VKA, while 15 patients were treated with DOACs. BPA success was defined by a final mPAP <31 mm Hg. Survival was analysed with Kaplan-Mayer curves and COX regression analysis.

Results: mPAP response was greater in VKA patients. Median single in-between-change of mPAP was minus 2 mm Hg compared with plus 0.5 mm Hg in the DOAC group. A significant difference in both all-cause mortality and PH-related mortality was found. 1 year survival rate was 91 % in the VKA group compared to 76 % in the DOAC group, mean survival in the VKA group was 49.215 months from first BPA (95 % CI 44.19–54.24) compared with 29.685 months (95 % CI 18.965–40.404) in the DOAC group ($p=0.0016$). COX regression analysis for known survival related factors including age and CI showed baseline mRAP as a predictor of outcome. 22.9 % of VKA patients had a procedure-related complication compared with 20 % of DOAC patients ($p=0.777$). Periprocedural hemoptysis occurred in 11 patients, all treated with VKAs. Other complications such as various stages of lung injury (7), pulmonary artery perforation (1), groin hematoma (2), and pseudoaneurysm of the femoral vein (1) occurred in both groups equally.

Conclusion: In our series, type of OAC appears to impact outcome in patients undergoing BPA. A larger scale study is needed to understand DOACs in CTEPH patients undergoing BPA.

PS 13/13-2

The role of asymmetric dimethylarginine (ADMA) in the follow-up of patients with precapillary pulmonary hypertension (PH)

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Introduction: Asymmetric dimethylarginine (ADMA) interferes with L-arginine in the production of nitric oxide, a key mediator of endothelial cell function. ADMA is elevated in pulmonary arterial hypertension (PAH) and chronic thromboembolic pulmonary hypertension (CTEPH) and is associated with unfavorable outcomes. Aim: To assess the role of ADMA to monitor disease progression of PH patients treated with PAH-specific therapy.

Methods: ADMA was measured by competitive ELISA at baseline (BL) and follow-up (FU). Risk assessment including a clinical assessment, echocardiography, 6-minute walking test, NT-pro-BNP and hemodynamic assessment by right heart catheterization was performed accordingly. Risk was calculated according to the ESC/ERS 2015 guidelines by the SPHAR method.

Results: ADMA samples were collected from 113 patients treated at our institution between 2012–2019. 89 (79 %) patients had PAH, 15 (13 %) were diagnosed with CTEPH and 9 (8 %) with group 3–PH associated with lung disease. 69 % were females. 15 (13.3 %) patients had a low risk at baseline, 96 (85 %) intermedi-

Fig. 1 | PS 13/13-1 Kaplan-Mayer survival curves. Survival analysis of BPA patients on VKA (blue) vs. DOACs (red). A: All cause mortality B: PH-Related mortality

a

b
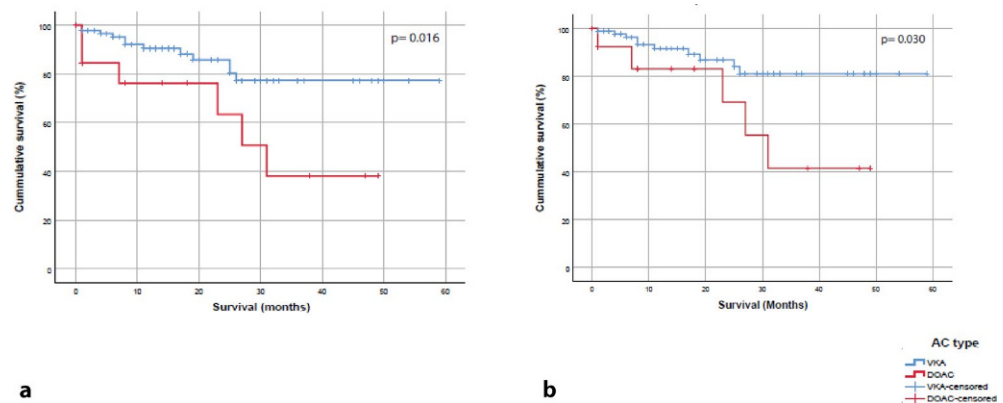
Fig. 2 | PS 13/13-1 Multi-variate COX survival model showing hazard ratio (95 % CI) of persistent PH, baseline PVR, baseline mRAP, baseline mPAP and AC type for BPA patients. Significance was shown only for AC type (HR 3.253, $p = 0.03$) and mRAP (HR 1.121, $p = 0.029$)
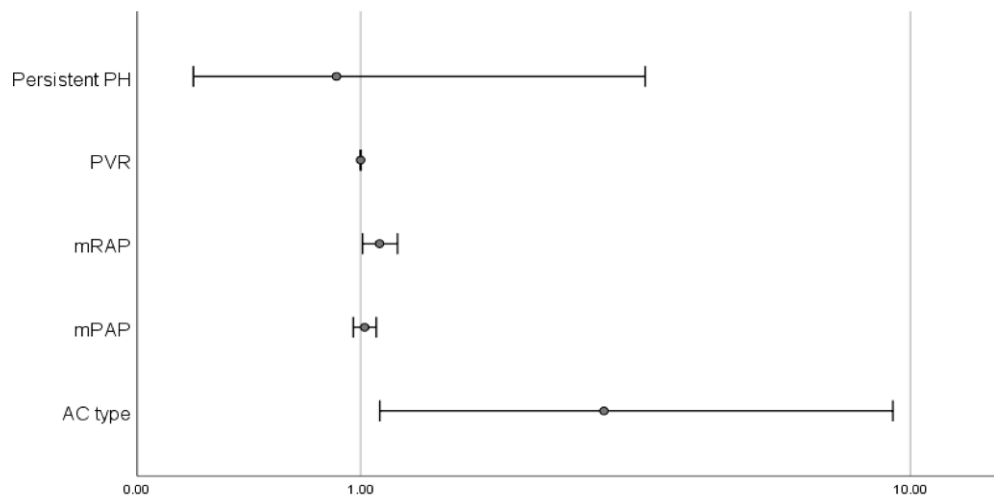
ate risk and 2 (1.8 %) were high risk patients. 75 % received oral medications, 31 % received subcutaneous treprostinil. Median baseline ADMA was 0.738umol/l. At BL no significant difference of ADMA plasma levels was found among the different PH types ($p = 0.063$), or between different risk categories ($p = 0.531$). Change in ADMA plasma levels correlated with change in risk ($p = 0.002$, rs 0.291) and with change in mixed venous saturation ($p = 0.034$, rs −0.205). Change in ADMA plasma levels also correlated with risk at FU ($p = 0.011$, rs 0.240). Patients categorized as low risk at FU had a median ADMA plasma level decrease of 22 %, compared with −3 to 0 % ADMA plasma level change in patients with moderate to high risk at FU ($p = 0.04$). Patients who improved their risk category had a median decrease of ADMA plasma level of 23 % vs. 2.3 % in patients who did not improve ($p = 0.011$). Decrease of ADMA plasma levels was a weak but significant discriminator for improvement of risk in ROC analysis ($p = 0.032$, AUC 0.374).

Conclusion: ADMA plasma levels paralleled the hemodynamic and clinical benefit of PAH-specific treatments in patients with precapillary PH. ADMA could be used as a biomarker for monitoring treatment effects in precapillary PH.

PS 13/13-3

Switch to generic Treprostinil in PAH–single center experience

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Introduction: Background For its pharmacological properties Treprostinil is the prostanoid of choice at our center since more than a decade. We both administer subcutaneous (sc) and intravenous (iv) Treprostinil using implantable pump systems and overlook one of the largest patient cohorts in Europe. A generic Treprostinil formulation was licensed in Austria in 2018. As we had used this formulation in a clinical trial before we decided to transition patients to the more cost-effective drug. Purpose We describe our experience in switching all patients from originator to generic Treprostinil over a period of 15 months.

Methods: A retrospective analysis of Treprostinil patients between October 2018 and December 2019 was performed.

Results: After commercial availability of generic Treprostinil we gradually started to transition patients from originator

to generic drug. In a second step new patients were initiated on the generic as well. Over 15 months 59 patients were transitioned and 21 patients were newly initiated on generic Treprostinil. Besides administrative issues like initial confusion of the more convenient vial size of 10 ml and some additional need for patient information no unexpected events were observed. This affects both subcutaneous use and iv administration in implantable pumps. Clinical benefits achieved with originator could be maintained, no additional safety signals were detected. In patients newly initiated on generic Treprostinil we did not observe any difference to our experience over the last 15 years.

Conclusion: In a fragile patient population like PAH patients switch to generic drugs is a critical decision. Based on our experience in a clinical trial we successfully switched all our PAH patients to the more cost-effective generic over 15 months.

PS 13/13-4

Long-term safety of implantable pumps for pulmonary arterial hypertension–a retrospective analysis

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Introduction: Parenteral prostacyclins are a mainstay in the management of pulmonary arterial hypertension (PAH); however administration is technically challenging and limited by frequent local reactions for subcutaneous (sc) or rare but possibly life-threatening infections for intravenous (iv) administration using external pumps. Implantable pumps might help to overcome these challenges but require surgery and surgical intervention in case of system malfunctions. Purpose We describe our long-term experience with implanted pumps for intravenous Treprostinil for PAH with a focus on system-related interventions.

Methods: A retrospective analysis of all implantations and related interventions between September 2010 and January 2020 was performed.

Results: Following a standardized surgical procedure 93 pumps were implanted during the observation period, 15 of those within a clinical trial. All patients had been uptitrated subcutaneously and were independently assessed by the PAH expert the surgeon and the anesthesiologist prior to implantation. Intraoperatively one case of ventricular tachycardia was observed, the patient recovered without sequelae. During postoperative stay two cases of pneumothorax, one case of hematothorax in a patient with concomitant hematological malignancy and one case of pleural effusion were successfully managed. In 7 cases mild seroma were observed postoperatively, none of them requiring invasive treatment. During more than 1900 patient-months 10 pump or catheter related issues led to surgical intervention: in 4 cases fixation failure and twisting of the pump required refixation and 6 catheter related problems had to be corrected. Over time the fixed flow rate of several pumps increased from 1.3 ml/day to a maximum of 2.3 ml/day in one case. As flow rates are monitored at every refill, Treprostinil treatment was adapted accordingly leading to a shortened refill interval in meanwhile 9 patients to 3 weeks and to two weeks in one patient. 7 pumps were changed as the shorter refill cycle was not considered acceptable. In 3 patients the infusion pump

was changed to a model with larger volume to facilitate dose increase. No case of line infection was observed.

Conclusion: Given a strict interdisciplinary approach infusion pumps for intravenous Treprostinil can safely be implanted in patients with PAH. The risk for catheter related infections seems neglectable. In our cohort unplanned surgical interventions related to the pump system were rare with less than one procedure per 15 patient-years. Increase of the flow rate over time mandates careful monitoring and may require change of the device.

PS 13/13-5

2000 refill procedures of implanted pumps for intravenous Treprostinil in patients with pulmonary hypertension

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Introduction: Background Since 2010 we have acquired vast experience with intravenous Treprostinil in the treatment of pulmonary hypertension administered by fully implantable pumps. We were the first center in Europe to implant an infusion pump with a refill interval of 28 days for Treprostinil. Purpose We describe our long-term experience with implanted pumps for intravenous Treprostinil for PAH with a focus on pump refills.

Methods: A retrospective analysis of all refills between October 2010 and December 2019 was performed.

Results: 89 pumps were implanted at our center between September 2010 and November 2019, 15 of those in a clinical trial. 7 patients who were referred from other centers for pump implantation were refilled at the referring hospitals, the other refills were performed at our outpatient clinic exclusively. All refills followed a strict protocol and were performed by dedicated physicians and staff only. No catheter or device related infections were observed. In more than 2000 refills only one serious complication occurred. Briefly after refill the patient reported dizziness, significant hypotension led to the suspicion of some paravasate. According to our standards the pump was emptied immediately, reflow confirmed the suspicion of a paravasate of around 4 ml. Using cathecholamines the patient was stabilized at our intensive care unit and was dismissed from hospital three days later. Notably this event occurred in late 2019 after more than 9 years of refill experience. Another important finding was the increasing flowrate of the implanted pump. Reduced reflow volumes at refill occurred in some pumps after 15 to 18 months. Treprostinil dose and/or refill intervals were adapted accordingly. From originally 1.3 ml/day flow rate increased to a maximum of 2.3 ml in one case. Meanwhile we have changed 7 pumps for increased flowrates.

Conclusion: Over more than 9 years we observed one serious complication with more than 2000 refill procedures. We are convinced that strict adherence to our standards contributed to these results. Nevertheless, despite all routine serious events like in our case may occur. It is essential to have defined standards also for worst case scenarios in place when managing this treatment option. Careful documentation of reflow volumes is mandatory to detect flow rate increases.

PS 13/13-6

Bilateral lung transplantation in a patient with HIV-associated PAH resulting in complete recovery of right ventricular function–Case presentation

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Introduction: HIV associated PAH is a rare complication of HIV infection. It occurs in approximately 0.5 % of infected patients. HIV-PAH belongs to group 1 PAH in the clinical classification of pulmonary hypertension [1]. While the pathogenesis of HIV-PAH is not fully established, specific viral and host factors seem to play an important role. After diagnosis of HIV-PAH, PAH directed medication should be offered to all patients in addition to antiretroviral therapy. HIV is still a relative contraindication for lung transplantation, with the exception of patients with undetectable HIV-RNA, no current diagnosis of AIDS and adherence to antiretroviral therapy. Concerning the right ventricle, even in severe reduced function, after lapse of pressure load, a complete recovery is possible.

Methods: Our patient is a 55-year-old male with HIV-PAH resulting in severe right ventricular dysfunction and significant functional tricuspid regurgitation. The disease was diagnosed in 2005. He is under specific medication with Sildenafil, Macitentan and subcutaneous Treprostinil. Under antiretroviral therapy HIV-RNA is below detection limit. Despite specific triple therapy, the patient suffers from recurrent right heart decompensation and pericardial effusion. Two times catecholamines were required to provide intensive diuretic treatment in acute renal failure of cardiorenal origin. Ascites paracentesis was also performed several times. The patient was evaluated for lung transplantation at the University Hospital of Vienna. In December 2018 bilateral lung transplantation was performed. In the follow up visits, right ventricular function recovered well and the PAP values decreased to normal ranges. Specific PAH medication was stopped. The patient receives immunosuppressive therapy with Tacrolimus, Mycophenolatmofetil and Prednisolone. Antiretroviral therapy was continued and HIV-RNA levels are still not measurable.

Results: After lung transplantation, right ventricular function recovered and the severity of tricuspid regurgitation improved from severe to mild.

Conclusion: Bilateral lung transplantation is an option in patients with progressive right heart failure due to HIV associated PAH with negative HIV-RNA and compliance in taking antiretroviral medication. It is very important that cardiologists, HIV specialists and transplant surgeons work together in an interdisciplinary team to provide the best treatment for the patient.

eGFR= estimated glomerular filtration rate
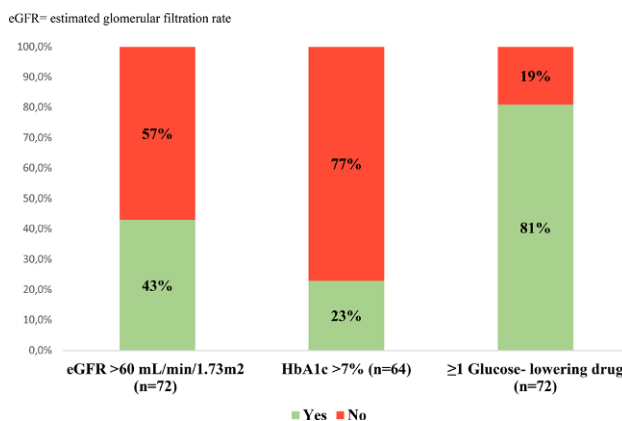
Fig. 1 | PS 14/14-1 Proportion of patients (without SGL-T2i) with eGFR >60 ml/min/1.73 m2, HbA1c >7 % and ≥1 glucose-lowering drug

Table 1 | PS 14/14-1 Baseline characteristics

Baseline	All (*N*=89)	SGLT2i (*N*=17)	NoSGLT2i (*N*=72)
Age(years)	70	66.7	71
BMI(kg/m2)	29.3	31.3	28.8
LVEF<50 %(%)	42	41	42
Metformin(%)	61	65	58
DPP4i(%)	26	41	22
GLP1-RA(%)	9	12	8
Sulfonylureas(%)	14	6	15
Insulin(%)	21	18	22
eGFR(ml/min/1.73 m2)	62	68	60
HbA1c(%)	6.8	7.5	6.7

BMI=Bodymassindex,DPP4i=Dipeptidylpeptidase-4inhibitor,eGFR=estimatedglomerularfiltrationrate,
GLP1-RA=Glucagon-likepeptide1-receptoragonist,LVEF=Leftventricularejectionfraction,SGLT2i=Sodium-glucosecotransporter2inhibitor

POSTERSITZUNG 14 – RISIKOFAKTOREN/STOFFWECHSEL/LIPIDE 2

PS 14/14-1

Prescription of SGLT2 inhibitors in hospitalized cardiovascular patients

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Introduction: Randomized controlled trials have proved that sodium-glucose cotransporter 2 inhibitors (SGLT2i) reduce cardiovascular (CV) events in patients with type 2 diabetes (T2 DM). Criteria for renumeration of SGLT2i by social health insurance and therefore potential hurdles for the use in clinical practice are an eGFR >60 ml/min/1.73 m2, an HbA1c >7 % and a pre-existing antidiabetic therapy. Real world evidence assessing the implementation in clinical practice is limited.

Table 1 | PS 14/14-2 Baseline characteristics

Baseline	All (N = 89)	GLP-1RA (N = 8)	NoGLP-1RA (N = 81)
Age(years)	70	64	71
BMI(kg/m2)	29.3	33.3	28.8
LVEF<50 %(%)	42	38	42
Metformin(%)	61	75	59
DPP4i(%)	26	0	28
SGLT2i(%)	19	25	19
Sulfonylureas(%)	14	13	14
Insulin(%)	21	63	17
eGFR(ml/min/1,73 m2)	62	78	60
HbA1c(%)	6.8	9.4	6.6

BMI = Bodymassindex, DPP4i = Dipeptidylpeptidase-4inhibitor, eGFR = estimatedglomerularfiltrationrate,
GLP1-RA = Glucagon-likepeptide1-receptoragonist, LVEF = Leftventricularejectionfraction, SGLT2i = Sodium-glucosecotransporter2inhibitor

Methods: Patients with coronary artery disease, cerebrovascular disease, or heart failure who were admitted to a cardiological ward of a tertiary referral hospital between 07/2019–10/2019 were screened for presence of T2 DM.

Results: Among 418 patients with CV disease, T2 DM was present in 89 (21 %) individuals. Of those patients with T2 DM, 89 % suffered from atherosclerotic disease and 69 % from heart failure. SGLT2i were prescribed only in 19 % of patients with T2 DM. Among patients without an SGLT2i, 77 % presented with an HbA1c < 7.0 %, 57 % had an eGFR <60 mL/min/1.73 m2 and 19 % did not have any antidiabetic therapy. Thus, 9.2 % of patients without an SGLT2i met the criteria for the use of an SGLT2i. Lowering the eGFR cut-off to 30 ml/min/1.73 m2, would allow additional 15.2 % of patients to qualify for an SGLT2i. Left ventricular ejection fraction did not differ significantly between those who received an SGLT2i vs those who did not (41.2 % vs. 41.7 %, $p = 0.971$) but patients with an SGLT2i tended to be younger (67 vs 71 yrs; $p = 0.095$).

Conclusion: SGLT2i use appears to be limited due to renumeration criteria of a required HbA1c level <7 % followed by an eGFR <60 ml/min/1.73 m2. Reducing the threshold of renal function based on the inclusion criteria of clinical trials (and in accordance with the U.S. Food and Drugs Administration) as well as accepting prescriptions of SGLT2i irrespective of HbA1c levels as recently recommended by the American Diabetes Association would allow a substantially larger proportion of high-risk patients with T2 DM and CV disease to be eligible for these cardioprotective drugs.

PS 14/14-2

Prescription of GLP-1 receptor agonists in hospitalized cardiovascular patients

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Introduction: Randomized controlled trials have proved that glucagon-like peptide 1 receptor agonists (GLP-1RA) reduce cardiovascular (CV) events in patients with type 2 diabetes (T2 DM). Criteria for renumeration of GLP-1RAs by social health insurance and therefore potential hurdles for the use in

eGFR= estimated glomerular filtration rate, BMI= Body mass index
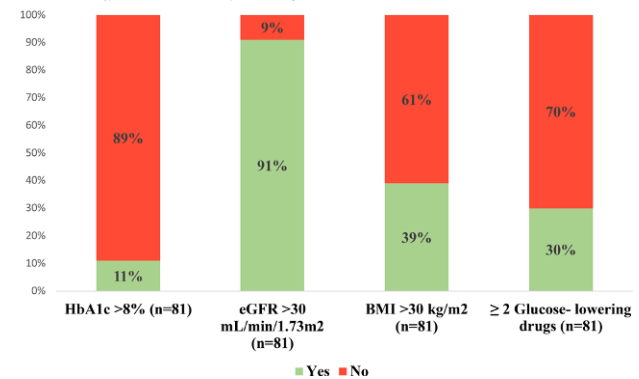
Fig. 1 | PS 14/14-2 Proportion of patients (without GLP-1RAs) with HbA1c >8 %, eGFR >30 ml/min/1.73 m2, BMI >30 kg/m2, and ≥2 glucose-lowering drugs

clinical practice are an eGFR >30 mL/min/1.73 m2, an HbA1c >8 %, a body mass index (BMI) >30 kg/m2 and at least two pre-existing glucose-lowering drugs. Real world evidence assessing the implementation in clinical practice is limited.

Methods: Patients with coronary artery disease, cerebrovascular disease, or heart failure who were admitted to a cardiological ward of a tertiary referral hospital between 07/2019–10/2019 were screened for presence of T2 DM.

Results: Among 418 patients with CV disease, T2 DM was present in 89 (21 %) individuals. Of those patients with T2 DM, 89 % suffered from atherosclerotic disease and 69 % from heart failure. GLP-1RAs were prescribed only in 9 % of patients with T2 DM. Among patients without a GLP-1RA, 89.2 % presented with an HbA1c <8.0 %, 70.4 % did not have at least two pre-existing antidiabetic therapies, 60.8 % had a BMI <30 kg/m2 and 8.6 % had an eGFR <30 mL/min/1.73 m2. Thus, 2.7 % of patients without a GLP-1RA met the criteria for the use of a GLP-1RA. As compared with individuals without, patients treated with a GLP-1RA were more likely to have higher HbA1c levels (9.4 % vs 6.6 %, $p < 0.001$), be younger (64 vs 71 yrs, $p = 0.024$) and tended to have a higher body mass index (33.3 vs 28.8, $p = 0.075$).

Conclusion: Despite large outcome trials showing efficacy and safety, GLP-1RAs are infrequently prescribed to patients with T2 DM and CV disease in clinical practice. Remuneration regulations that better reflect the inclusion criteria of the CV outcomes trials would allow more patients at high risk to receive these CV protective drugs.

PS 14/14-3

Visceral adipose tissue and waist circumference for prediction of cardiometabolic risk factors: Sex-specific insights from the Framingham Heart Study

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Introduction: Simple anthropomorphic measures of obesity, including body mass index (BMI) and waist circumference (WC) are easy to obtain. However, it remains unknown to what degree they reflect the association of visceral adipose tissue (VAT), a harbinger of inflammation, and incident cardiometabolic risk factors.

Methods: We included participants from the Framingham Heart Study (Offspring and 3rd generation), who underwent abdominal multidetector computed tomography (CT) scanning between 2002 and 2005. We stratified participants by metabolic health at baseline, defined as <2 Adult Treatment Panel-III criteria, and obesity (BMI ≥30 kg/m2). Thus, four distinct metabolic phenotypes were delineated: metabolically healthy non-obese/obese (MHN/MHO), and metabolically unhealthy non-obese/obese (MUN/MUO). We assessed VAT as CT-based measure of fat, and WC as anthropomorphic measure of abdominal fat. We further assessed incident cardiometabolic risk factors (diabetes, hypertension, low HDL, hypertriglyceridemia) as measured during clinical follow-up visits between 2005 and 2011. We used logistic regression models to test the association of VAT and WC with incident cardiometabolic risk factors. Sex-specific analyses were performed in all participants and across the four metabolic phenotypes, adjusted for age and smoking.

Results: Out of 3,482 participants (50.8 ±10.3 y/o, 48.1% female), 1,999 (57.4%) were metabolically healthy, among them 321 (16.1%) were obese. Out of 1,483 (42.6%) metabolically unhealthy participants, 643 (43.4%) were obese at baseline. Overall, both VAT and WC were associated with all inci-dent cardiometabolic risk factors (diabetes, hypertension, low HDL, hypertriglyceridemia; OR range 1.62 [1.39–1.89] to 3.77 [2.91–4.87] per 1-SD increase) during a median follow-up of 6.34 years. We observed a significant interaction between sex and VAT volume for all outcomes ($p < 0.0001$ for diabetes, low HDL and hypertriglyceridemia; $p = 0.031$ for hypertension). In contrast, there was only an interaction between sex and WC for hypertriglyceridemia ($p = 0.036$), but not for diabetes, hypertension, and low HDL ($p > 0.239$ for all). In sex-specific analyses, WC had similar strength of associations as compared to VAT in men. In contrast, the association of VAT with all cardiometabolic risk factors was notably stronger than that of WC in women (upper panel of the figure; adjusted odds ratios are given per 1-SD increase of VAT and WC). These results were consistent across the four metabolic phenotypes (lower panels of the figure).

Conclusion: In men, WC accurately reflects the association of VAT with incident cardiometabolic risk. In contrast, VAT is more closely associated with incident cardiometabolic risk as compared to WC in women. Sex-specific approaches for risk assessment should be further examined. WC may be considered as an appropriate initial anthropometric measure of cardiometabolic risk assessment in men but not in women.
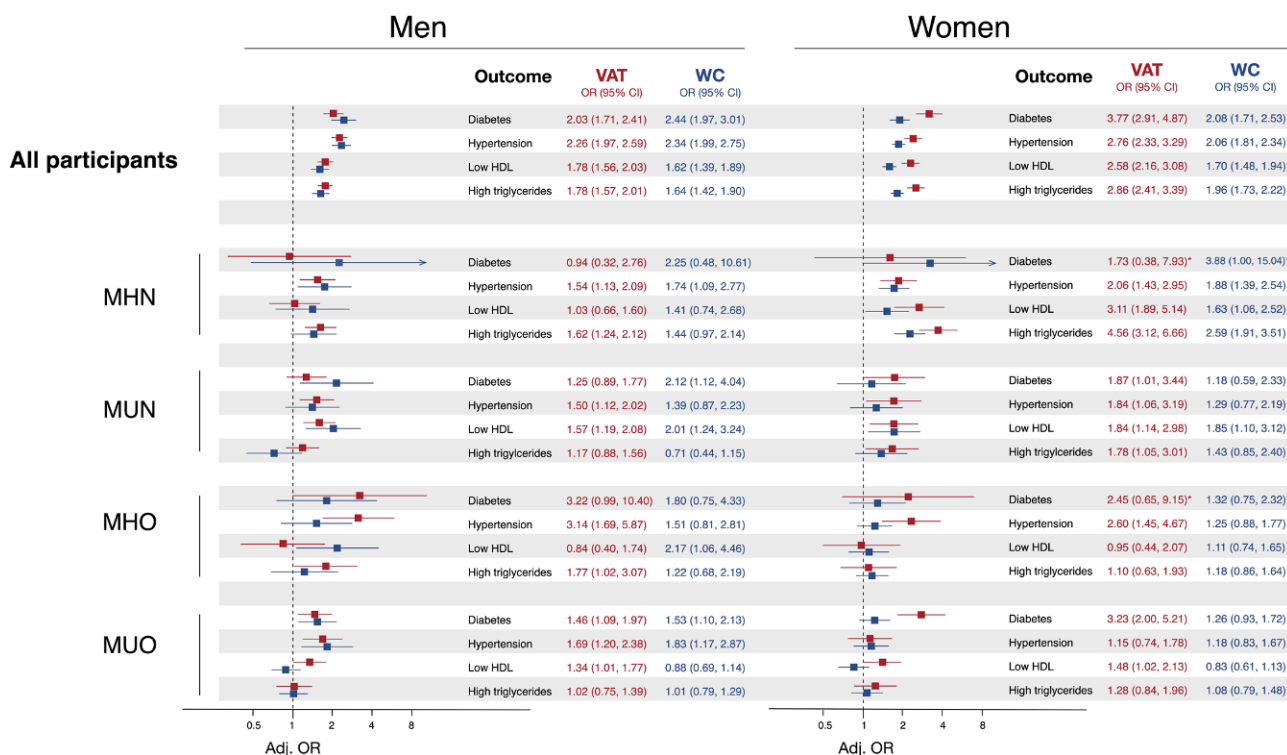
Abb. 1 | PS 14/14-3

PS 14/14-4

Assessing the impact of switching to the Tobacco Heating System on cardiovascular disease: Translating basic science into clinical benefit

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Introduction: Cigarette smoke (CS) is causally linked to the development of cardiovascular diseases (CVD). Tobacco harm reduction, by virtue of substituting cigarettes with less harmful products, is a complementary approach to current strategies for smokers who would otherwise continue to smoke. The Tobacco Heating System (THS) 2.2 is a novel tobacco product that heats tobacco instead of burning it, never allowing the temperature to exceed 350℃, thereby preventing the combustion process from occurring and producing substantially lower levels of toxicants than CS.

Methods: Our assessment program aims to demonstrate that switching to THS has the potential to reduce the risk of smoking-related diseases compared with continued smoking. The program includes in vitro/in vivo toxicology testing methods that follow OECD guidelines and Good Laboratory Practice, a systems toxicology approach, and randomized, controlled clinical studies that follow the principles of Good Clinical Practice.

Results: The results of the THS assessment program demonstrated that cardiovascular toxicants are reduced by an average of >92 % in THS aerosol relative to CS and that THS aerosol contains no solid carbon-based nanoparticles. The effects of THS aerosol on the adhesion of monocytic cells to human coronary endothelial cells in vitro are significantly reduced. Switching to THS halted the progression of CS-induced atherosclerotic changes in ApoE–/– mice in vivo. Biomarkers linked to the development of smoking-related diseases were analyzed following a 6-month randomized, controlled clinical study with THS, which demonstrated a consistent improvement of biomarkers in different pathophysiologic pathways leading to atherosclerosis.

Conclusion: The evidence available to date indicates that switching to THS has the potential to reduce the risk of smoking-related diseases such as CVD. As a next step, we will complement our THS assessment program with cardiovascular outcome studies intended to further support the clinical benefits of switching to THS over continuous smoking.

PS 14/14-5

Primärprävention im Frisiersalon-Design und Rationale der Friseurinitiative Favoriten 2020

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Einleitung: Bluthochdruck ist weltweit die häufigste Todesursache. Die Folgeerkrankungen der Hypertonie sind in Form
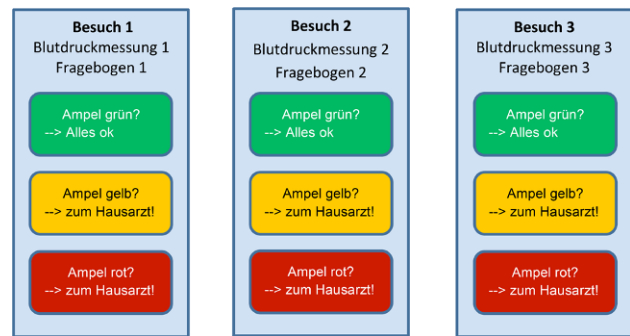
Abb. 1 | PS 14/14-5

von Schlaganfall, Herzinfarkt, sowie Herz- und Niereninsuffizienz sowohl individuell schwerwiegende Ereignisse, als auch gesundheitsökonomisch höchst bedeutsam. Österreich schneidet beim Erreichen des Therapiezielwertes bei PatientInnen mit Hypertonie im internationalen Vergleich sehr schlecht ab. Rezente Studien zeigten, dass nur ca. 40 % der PatientInnen das Blutdruckziel (office Messung <140/90 mm Hg) erreichten. Darüber hinaus ist in Ostösterreich das Bewusstsein für das Vorhandensein von Risikofaktoren in Bezug auf die eigene Person gering. Gerade die Awareness für kardiovaskuläre Risikofaktoren ist eine conditio sine qua non, um ärztliche Hilfe in Anspruch zu nehmen und auch ein Prädiktor für eine höhere Lebenserwartung.

Methoden: Die Studie wird als longitudinale Kohortenstudie (Pre-Post Intervention Quasi-Experimental Design und repetitive Messungen) angelegt. (Abb. 1) Epidemiologisches Setting: Alle Betreiber eines Frisiersalons in Favoriten werden eingeladen an der Studie teilzunehmen, insgesamt sollen 30 Salons teilnehmen. Eine Repräsentanz aller soziokulturellen Gruppen des Bezirks wird angestrebt. Jeder teilnehmende Salon soll 20 Stammkunden in die Studie einschließen. Es wird jeweils ein Mitarbeiter der teilnehmenden Salons in der Durchführung einer Blutdruckmessung geschult und das Studienprotokoll erklärt. Blutdruckmessung: Nach mindestens 5 min in sitzender Position erfolgen zwei Blutdruckmessungen in üblicherweise mit einer Minute Abstand voneinander auf dem linken Arm. Die Blutdruckmessung erfolgt mit einem automatischen Messgerät mit Ampelfunktion (s. u.). Es wird der Mittelwert aus beiden Messungen zur weiteren Analyse herangezogen. Zuweisung zum Hausarzt: Liegt der Durchschnitt der Blutdruckmessungen 1, 2 oder 3 über dem Wert von 160/100 mm Hg (Ampel rot) oder über 140/90 mm Hg (Ampel gelb), so werden die ProbandInnen aufgefordert ihren Hausarzt/Hausärztin mit dem ausgehändigten Informationsblatt aufzusuchen.

Resultate: Primärer Endpunkt ist die Erfassung des Anteils an Probanden mit Bluthochdruck und die Ermittlung des Anteils an hypertensiven Probanden, die der Empfehlung einen Arzt aufzusuchen nachkommt. Sekundäre Endpunkte sind Unterschiede bei Patienten mit und ohne Migrationshintergrund, Unterschiede zwischen Männern und Frauen, sowie eine mögliche Blutdruckreduktion bei Patienten zu messen, die in der ersten oder zweiten Messung hypertensive Werte hatten.

Schlussfolgerungen: Wir hoffen mit diesem neuartigen Zugang ein effizientes Modell schaffen, um einen Beitrag zur Verbesserung der Blutdruckeinstellung in Wien zu ermöglichen.

PS 14/14-6

Bleeding events in patients with cardiac amyloidosis

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Introduction: Amyloid cardiomyopathy (CA) is a life-threatening disease that arises from the accumulation of insoluble fibrous deposits of misfolded proteins in the myocardium [1]. Infiltration of the conduction system results in a high prevalence of atrial fibrillation (AF) [2]. Due to a notable thrombo-embolic risk, long-term anticoagulation is preferred once AF is detected in amyloidosis [2]. However, anticoagulation may exacerbate the hemorrhagic tendency, previously described in amyloid light-chain (AL) amyloidosis [3]. Therefore, the potential benefits of anticoagulation must be carefully weighed against hemorrhagic complications.

Methods: We recorded bleeding events in a prospective cohort of CA patients.

Results: Out of 140 patients (median age of 73 years (IQR 62–77) and 77.1 % of male gender) 65 (46.4 %) were diagnosed with AL amyloidosis and 69 (49.3 %) had transthyretin amyloid deposits (ATTR). Median NYHA functional class was 3 (IQR 2–3) and NT-proBNP 2574 pg/mL (1070–7173). 42.1 % of patients ($n = 59$) were treated with OAC with 13 (22.0 %) receiving vitamin-K-antagonists (VKA) and 46 (78.0 %) novel oral anticoagulants (NOACS). During a median follow up time of 23 months (IQR 9–35) 22 bleeding events occurred. In total, we recorded 1 cerebral bleeding, 7 gastrointestinal bleedings, 8 urogenital bleedings, 4 hematomas and 2 nasal bleedings. Of those, fifteen (68.2 %) were observed in orally anticoagulated patients ($p = 0.198$). 4 (30.8 %) bleeding events occurred in the VKA treated group and 11 (23.9 %) under NOAC treatment ($p = 0.335$). Bleeding events were more prevalent in patients with AL compared to TTR amyloidosis (14, 63.6 % vs 8, 36.4 %, $p = 0.011$). No difference was observed in terms of CHA2DS2-VASc score: 4 (IQR 2–5) in the bleeding group versus 3 (IQR 1–4) in the control group ($p = 0.579$). On the contrary, HASBLED score was higher in patients with bleeding events: 4 (IQR 4–5) versus 2 (IQR 1–3) ($p < 0.001$). Patients with bleeding events had a higher prevalence of arterial hypertension ($p = 0.010$) and chronic kidney disease ($p = 0.002$). Overall survival was comparable between the groups (Fig. 1).

Conclusion: Although CA is associated with potentially life-threatening hemorrhage, our cohort did not reveal a higher prevalence of fatal bleeding in patients treated with oral anticoagulation. Patients who experienced bleeding events did not show worse outcomes.

COVID-19 UND HERZ 2 POSTERSITZUNG

CO-2-1

Cardiac complications in a COVID-19 patient population from a medical non-university hospital intensive care unit in Styria

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Introduction: Between 6th of march and 29th of april 2020 16 patients with a Covid-19 infection were admitted to the medical intensive care unit of the reporting hospital. The following abstract places emphasis on the cardiovascular complications of this special patient population.

Methods: Retrospective Analysis.

Results: The population consisted of more male patients ($n = 10$) and the mean age was 68.7 years (50–84a range). Pre-existing conditions related to the cardiovascular system were present in the majority of patients: arterial hypertension (75 %), obesity (63 %), diabetes (19 %), coronary artery disease (12 %), atrial fibrillation (12 %) and 56 % of patients were on preexisting therapy with either an ace-inhibitor or an angiotensin-II-receptor blocker and 15 % of patients were already on oral anticoagulation (vitamin k antagonist or noac). Cardiac complications were a very frequent adverse event in the Covid-19 patient population, with 69 % ($n = 11$) of patients experiencing one or more events related to the cardiovascular system: 8 patients had a significant high sensitive troponin elevation and 4 patients showed an abnormal left ventricular ejection fraction. Further relevant cardiac complications were new onset of atrial fibrillation ($n = 2$), sustained ventricular arrhythmia ($n = 1$), pericardial tamponade with the need of pericardiocentesis ($n = 1$) and pulmonary artery embolism with the need for cardiopulmonary resuscitation and thrombolytic therapy ($n = 1$). Of 11 patients with one or more cardiac complications 45 % died ($n = 5$).

Conclusion: Cardiac complications were a frequent finding in critically ill patients with Covid-19 infection in the reporting intensive care unit. Treating physicians have to be alert to recognize this complications early and must be well trained to interpret elevated cardiac markers in a correct manner and to perform diagnostic (e.g. echocardiography) and advanced life saving procedures (e.g. pericardiocentesis) bedside despite suboptimal circumstances.
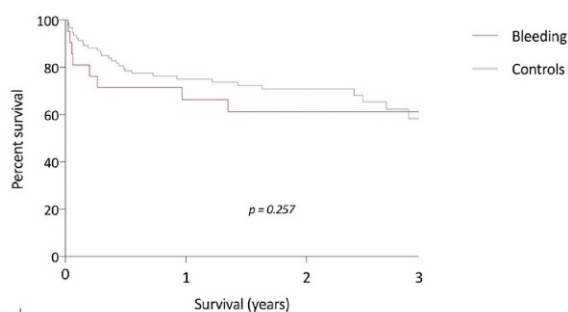
Fig. 1 | PS 14/14-6 Kaplan Meier curves

CO-2-2

Comprehensive assessment of symptoms and clinical status during and after SARS-CoV-2 infection

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Introduction: The SARS-CoV-2 pandemic of 2020 has an influence on people's lives world-wide, impacting global health and putting pressure on health care systems, politics and economies more than any other pandemic so far. This virus continues to cause a considerable number of deaths, and there is no proven solution to prevent it from spreading. While there have been many studies describing the epidemiology, pathophysiology and possible therapies of COVID-19, little is known about the long-term symptoms and physical performance after recovery. So far it remains unclear, which symptoms sum up to be the most relevant and characterizing ones and which symptoms might predict a more severe course and how patients recover from this disease in the long term. The aim of this study was to assess all symptoms experienced by the patients, distinguishing between the initial symptoms during the hospital admission and the long-lasting ones. These results should help characterize SARS CoV-2 more precisely, single out the early warning signs and show the long- term effects especially on cardiovascular function and performance.

Methods: We included patients after verified SARS-CoV-2 infection, who had been treated at our hospital and recovered. Three months after discharge patients were interviewed and examined. Information about symptoms at admission, during hospital stay and 3 months after discharge were collected. Blood was analysed 3 months after discharge. In addition, antibodies and a comprehensive list of blood parameters were assessed including NTpro BNP, and several other tests were performed. In all patients further testing is scheduled 6 months, one and two years post discharge.

Results: In this ongoing trial, we present data of the first 6 patients (2 males, 4 females; median age: 39.5 years (IQR 29.4–53.4)). In case of acceptance, data on 200 patients in total will be presented.
All patients included so far only had a mild course of disease and none of them had to be admitted to an intensive care unit at any time. The median time from hospital discharge to the clinical assessment was 120.0 ± 9.6 days. Main symptoms described by 67 % of the patients were fever, 50 % suffered from cough and fatigue followed by 30 % from nausea, cephalea and loss of taste and smell. The average duration of symptoms before admission was 1–3 days. During the hospital stay, with an average length of 5.8 days, additionally pneumonia, acute kidney failure and blood pressure crisis occurred. The length of symptoms were between 3–21 days. After 3 months, 4 patients still complain about fatigue and weakness, 1 about palpitations, and 1 about loss of taste and smell. All the patients were scored NYHA I, one of them CCS I, the others 0. The median NT proBNP was 45.4 (IQR 16.5–88.7) and thus not suggestive for heart failure.

Conclusion: Early symptoms included fever, cough, cephalea and fatigue. Even 3 months after discharge, the majority of patients still suffer from symptoms like fatigue, weakness, palpitations, or loss of taste and smell. By the end of the study we shall present comprehensive information about the course of symptoms in a large study population.

CO-2-3

Massive Pulmonalembolie mit Rechtsherzbelastung und akuter Psychose bei einem ambulanten Patienten mit mildem Verlauf von COVID-19

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Einleitung: Das erhöhte thromboembolische Risiko von Patienten mit Corona Virus Erkrankung 2019 (COVID-19) ist bereits bekannt und eine prophylaktische Antikoagulation von stationär aufgenommenen Patienten wird empfohlen. Eine massive Pulmonalembolie mit Rechtsherzbelastung, gefolgt von einer akuten Psychose, von einem nicht hospitalisierten Patienten und mit mildem Verlauf einer COVID-19-Infektion wurde bisher nicht beschrieben.

Methoden: Ein 46-jähriger, bisher gesunder, Patient ohne familiäre Vorgeschichte von venösen Thromboembolien suchte im April 2020 wegen akuter Dyspnoe und linksseitigen Thoraxschmerzen die Notaufnahme auf. Seit 18 Tagen litt der Patient unter trockenem Husten, Hämoptysen, Schwindel, Kopf-
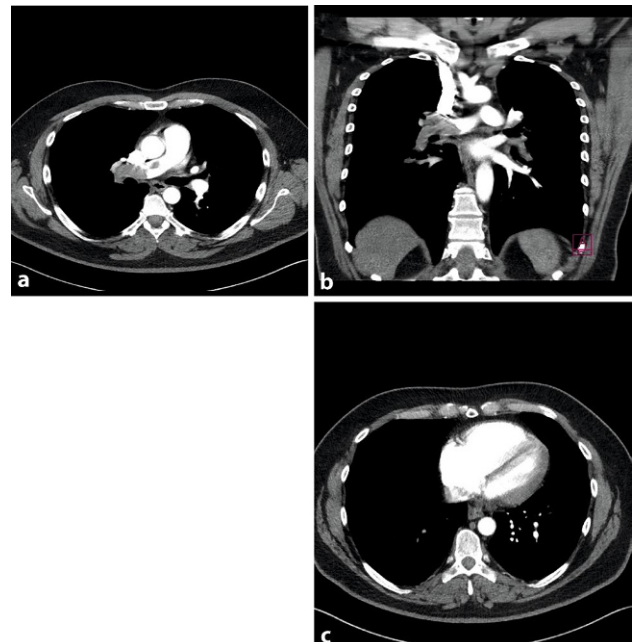
Abb. 1 | CO-2-3 Computertomographische (CT) pulmonale Angiographie. A (axial) and B (koronal) Bilder eines CT Scans mit Aufnahme der akuten pulmonalen Emboli in der rechten Pulmonalarterie, sowie der linken Pulmonalarterie, wo sie Füllungsdefekte erzeugen. Die Thromboembolie reicht in mehrere segmentalen und subsegmentalen Äste beider Pulmonalarterien. C Das axiale CT Bild zeigt eine Rechtsherzbelastung mit Dilatation des rechten Ventrikels

Besserung der Symptome

Akute Dyspnoe
Krankenhausaufnahme
Diagnose: Pulmonarem-
bolie und COVID-19
-> Transfer auf eine
COVID-19 Station
-> Start mit Heparin

Ceftriaxon

Entlassung vom Spital
in Heimquarantäne
bis 3. Mai

Patient verhält
sich zunehmend
psychotisch

Besserung der
psychiatrischen
Symptomatik
COVID-19 Test
ist negativ

Niedergelassener
Psychiater setzt Valproat
und Risperidon ab

20.03.2020   05.04.2020   10.04.2020   14.04.2020   20.04.2020   22.04.2020   25.04.2020   15.05.2020
30.03.2020   07.04.2020   13.04.2020   19.04.2020   21.04.2020   24.04.2020

Beginn der Symptome:
Husten, Kopfschmerz,
Schwindel, Diarrhö,
Erbrechen
-> Immobilisation

Schwellung im
linken Unterschekel

Erste Zeichen
einer Psychose
Start mit Risperdal
Heparin zu Edoxaban

Wechsel von
Heparin zu Edoxaban

Anfallsgeschehen
-> stationäre
Wiederaufnahme
Diagnose: psychogener
nicht-epileptischer
Anfall

Transfer an eine
psychiatrische
Abteilung
COVID-19 Test ist
negativ

Entlassung mit
Valproat und
Risperidon

Zuhause des Patienten

verschiedene Spitäler

Niedergelas-sener Arzt

Abb. 2 | CO-2-3

schmerz, Dysgeusie mit einem metallischen Geschmack, sowie Erbrechen und hatte die meiste Zeit im Bett verbracht. Seit 2 Tagen spürte er eine Schwellung und Schmerzen im rechten Unterschenkel. In der körperlichen Untersuchung waren ein BMI von 30,5 kg/m2, eine Atemfrequenz von 25/min, sowie eine Herzfrequenz von 120/min auffällig. Im EKG fanden sich Zeichen einer Rechtsherzbelastung. Das D-Dimer lag bei 17 mg/l (normal <0,5 mg/l). Mittels Computertomographie wurde eine massive beidseitige Pulmonalembolie diagnostiziert (Abb. 1) und eine Behandlung mit niedermolekularem Heparin begonnen. Wegen der Symptome, die schon vor der Aufnahme aufgetreten waren, erfolgte ein nasopharyngealer Abstrich, der eine positive PCR auf Corona Virus 2 (SARS-CoV-2) ergab. Der Patient wurde an eine COVID-19 Station transferiert. Wegen eines erhöhten C-reaktiven Proteins erhielt der Patient 2 g/d Ceftriaxon für fünf Tage. Die Antikoagulation wurde nach fünf Tagen von niedermolekularem Heparin auf Edoxaban 60 mg/d umgestellt (Abb. 2).

Resultate: Drei Tage nach Beginn der antibiotischen Therapie begann der Patient zu halluzinieren. Nach wenigen Gaben Risperidon (je 2 mg/d für 10 Tage), schien sich die psychische Situation zu stabilisieren und er wurde in Heimquarantäne entlassen. Nach wenigen Stunden wurde der Patient wegen eines Krampfanfalls neuerlich aufgenommen. Klinisch-neurologische und bildgebende Untersuchungen waren unauffällig, sodass ein psychogener nicht-epileptischer Anfall diagnostiziert wurde. Der psychische Zustand des Patienten verschlechterte sich und es erfolgte eine Überstellung an eine psychiatrische Abteilung, wo der Patient schließlich Wahngedanken entwickelte. Unter einer Therapie mittels Risperidon und Valproat besserten sich die Symptome zusehends, sodass der Patient, nach zwei negativen SARS-CoV2 Abstrichen, 12 Tage nach der ersten Krankenhausaufnahme nach Hause entlassen werden konnte. Ein telefonisches Follow-Up erfolgte 32 Tage nach Entlassung und ergab, dass die Therapie mit Valproat und Risperidon von einem niedergelassenen Psychiater beendet wurde. Der Patient ist nicht mehr dyspnoisch und bezeichnet sich selbst als mental geheilt.

Schlussfolgerungen: Bei unserem Patienten handelt es sich, um die Entwicklung einer Pulmonalembolie außerhalb eines stationären Aufenthaltes. Der genaue Grund, warum COVID-19 mit einem erhöhten thromboembolischen Risiko einhergeht, und ob die Immobilisation dabei eine Rolle spielt, ist bisher ungeklärt. Die Inzidenz von Thromboembolien, sowie Pulmonalembolien erscheinen in einer Obduktionsstudie gehäuft als Todesursache. [1] Dass das Auftreten von thromboembolischen Ereignissen nicht nur auf gravierende Krankheitsverläufe begrenzt ist, sondern ebenfalls in milden Fällen vorkommen kann, zeigt unser Fallbericht. Die Ursache der Entwicklung seiner Psychose ist bisher ungeklärt. Die Pandemie [2], Thromboembolien [3], sowie die virale Infektion könnten Ursachen der paranoiden Adaptionsstörung des Patienten sein. Wir wollen mit diesem Fall die Auswirkungen der COVID-19 Pandemie aufzeigen, welche nicht nur stationäre Patienten betrifft, sondern ebenso jene, die zu Hause in Quarantäne sind. Die psychiatrische Entwicklung des Patienten sollte Anstoß zur weiteren Forschung im Hinblick auf die neuro-psychiatrischen Auswirkungen von Pandemien, Quarantäne-Maßnahmen, sowie Viruserkrankungen geben.

CO-2-4

Effects of SARS-CoV-2 infection on cardiac function–preliminary data on echocardiographic parameters after COVID-19

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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. While
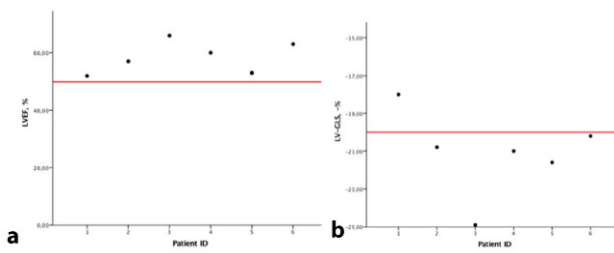
Red lines indicate cut-off values for normal LVEF (50%) and LV-GLS (-20%) respectively.

Abb. 1 | CO-2-4 Left ventricular function parameters measured by echocardiography. Panel A shows left ventricular ejection fraction (LVEF), and panel B left ventricular global longitudinal strain (LV-GLS)

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Age, years	46.7	42.3	36.6	27.1	30.2	73.2
Sex	Male	Male	Male	Male	Female	Female
Height, cm	172	185	190	203	166	160
Weight, kg	60	77	95	105	54	75
SBP, mmHg	140	114	130	125	118	150
DBP, mmHg	70	70	80	83	82	80
HR, mmHg	62	80	70	70	77	85
SpO2, %	98	98	99	97	99	97
In-hospital time, days	13.0	4.0	1.0	7.0	1.0	14.0
Respiratory support	None	None	None	NC	None	NC
Comorbidities	MDS, Bone marrow transplant, chronic renal failure	Asthma	Hypothyroidism	Allergies (Pollen)	None	Arterial hypertension,

SPB indicates systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate; SpO2, peripheral oxygen saturation; NC, nasal cannula and MDS, myelodysplastic syndrome.

Abb. 2 | CO-2-4 Baseline characteristics of the total study population

there have been a multitude of studies describing the epidemiology, pathophysiology and possible therapies of COVID-19, little is known about possible long-term effects in patients who have overcome an infection with SARS-CoV-2. Even though the virus is said to mostly affect the lungs, it has been shown, that some patients show signs of cardiac involvement, which can even develop into severe myocarditis. However, we do not know, if the virus has more subtle effects on the heart even in patients without evident cardiac involvement. The aim of this study was to assess subclinical myocardial dysfunction by measuring left and right ventricular strain by transthoracic echocardiography in patients after COVID-19.

Methods: We included patients after a verified infection with the SARS-CoV-2 virus, who had been discharged from the hospital. Baseline parameters 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) strain was measured in an RV-optimized four-chamber view. In addition, standard 2-D and Doppler measurements were performed in each patient to describe cardiac dimensions as well as systolic, diastolic and valvular function.

Results: In total, 6 patients were included in this study. Of these, 4 patients were male and 2 were female. Median age was 39.5 years (IQR 29.4–53.4) and the mean time from hospital discharge to echocardiography assessment was 120.0 ± 9.6 days. All patients included in this study had only mild course of disease and none of them had to be admitted to an intensive care unit at any point. Baseline characteristics of all patients are shown in table 1. LV-ejection fraction and RV function were normal in all patients. LV-GLS was borderline impaired in only 1 patient (–18.0 %), however this individual had a medical history of bone marrow transplant due to myelodysplastic syndrome and

chronic renal failure, which may have impacted these results (figure 1 and figure 2).

Conclusion: Our preliminary data show that there is no definite sign of subclinical cardiac dysfunction, in patients after an infection with COVID-19 when applying 2D speckle tracking echocardiography to measure left-and right ventricular strain.

CO-2-5

Sinoatrial arrest in patient with coronavirus disease (COVID-19)

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Introduction: Pulmonary problems are the most frequent manifestations of COVID-19 infections, but cardiac involvement may occur. These complications may occur as myocarditis, acute coronary syndrome, cardiogenic shock, heart failure and tachyarrhythmia. Sinoatrial arrest has, to our knowledge, so far not been reported in COVID-19 infection. A 47-year old Caucasian male patient was admitted on March 23rd, 2020 because of a syncope at home on the way to the toilet, lasting about two minutes. Thirty minutes after admission in the emergency room, he again lost consciousness. The ECG monitor showed a sinoatrial arrest of 10 s. After two chest compressions, normal sinus rhythm returned. The patient had a history of hypothyroidism since 9 years, arterial hypertension since 6 years and diabetes mellitus type 2 since one year. He had suffered from fever and cough for 7 days and diarrhea and emesis for one day. He was on a medication with metformin 850 mg twice a day, ramipril 10 mg, hydrochlorothiazide 25 mg and levothyroxine sodium 75 microgram.

Methods: His blood pressure was 124/84 mm Hg. The clinical investigation showed, except a body mass index 38, no further abnormalities. Blood tests showed a mildly elevated C-reactive protein (24.3 mg/l, normal <5 mg/l), hypokalemia (3.2 mmol/l, normal 3.4–4.5 mmol/l) and elevated transaminases (ASAT 69 U/l, ALAT 82 U/l, normal <50 U/l). Creatinkinase and high-sensitive troponin T were within the normal range. The chest X-ray showed a slightly enlarged heart and a left-sided pulmonary consolidation. A SARS-CoV-2 PCR was carried out from a nasal swab. While waiting for the result, 25 h after the first syncope, a new episode of bradycardia, followed by junctional rhythm and sinoatrial arrest of 13.5 s occurred, which necessitated 15 chest compressions. Bedside transthoracic echocardiography showed slightly thickened left ventricular walls and a normal systolic function.

Results: A positive PCR test for COVID-19 was obtained 34 h after admission. Sputum and blood cultures, borrelia antibodies, legionella antigen, as well as examination for influenza A and B viral nucleic acid were negative. The patient was treated with cefuroxime for 6 days. No further episodes of sinoatrial arrest were detected, nor did patient feel any dizziness or syncope. No further ECG changes were detected. The patient recovered and was discharged from the hospital after 8 days. At telephone follow-up, three months after admission, he reported that he did not suffer from further dizziness or syncope.

Conclusion: A causal relationship of the sinoatrial arrest with the COVID-19 infection cannot be excluded. Potential mechanisms are cytokine-mediated inflammation/injury of the cardiomyocytes of the conduction system or a vaso-vagal reaction due to the viral infection.

Clinical history & status
Laboratory analyses
Echocardiography +/- Cardiac magnetic resonance imaging
Spiroergometry
Pulmonary function test
Six minute walk test
Thoracic imaging

Fig. 1 | CO-2-6 Examinations and assessments at follow up visits, 3, 6, 12 and 24 months after hospital discharge

CO-2-6

Evaluation of long-term outcomes after COVID-19-infection–rationale and design of an explorative prospective study

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Background: 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. While there have been a multitude of studies describing the epidemiology, pathophysiology and possible therapies of COVID-19, little is known about possible long-term effects in patients who have overcome an infection with SARS-CoV-2.

Methods: The aim of this study is to assess long-term effects on lung and heart in patients after COVID-19 and to evaluate possible prognostic factors.

Results and Conclusion: We plan to include 200 patients after a verified infection with the SARS-CoV-2 virus. Four follow-up visits (figure 1) will be scheduled (3, 6, 12 and 24 months after discharge from hospital). Baseline parameters will be assessed, including clinical history, quality of life questionnaires (EQ-5D, Kansas-City Questionnaire), vital signs and symptoms. Standard laboratory parameters will be measured and extended laboratory testing will include SARS-CoV-2 antibody tests, renin-anigotensinaldosterone (RAS) fingerprint, T-cells analyses. Transthoracic echocardiography (TTE) will be performed in every patient. Cardiac magnetic resonance imaging will only performed in cases with abnormal findings on TTE. Pulmonary imaging will be performed using pulmonary ultrasound and low-dose-computed tomography to evaluate the presence of pathological long-term findings. Functional tests will evaluate the cardiopulmonary performance of affected patients and will include pulmonary function tests, diffusion capacity of carbon monoxide, spiroergometry and a six-minute walk test.

POSTERSITZUNG 15 – BASIC SCIENCE 2

PS 15/15-1

Overexpression of sirtuin 4 impairs systemic glucose homeostasis and promotes cardiac hypertrophy during high-fat diet

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Introduction: Type 2 diabetes is often accompanied by increased serum lipid levels and ectopic lipid storage which may contribute to diabetic cardiomyopathy with cardiac hypertrophy and diastolic dysfunction, among other diabetic complications. Sirtuin 4 (SIRT4) is a NAD+-dependent lysine deacylase which regulates the activity of energy metabolic enzymes by modulation of protein lysine modifications and is induced in diabetes mellitus. While SIRT4 deficiency has been shown to attenuate diet-induced obesity and fat storage, overexpression of SIRT4 accelerates cardiac hypertrophy following treatment with angiotensin II. Thus, we hypothesized that induction of SIRT4 in diabetes may link impaired systemic lipid homeostasis with development of diabetic cardiomyopathy.

Methods: Mice with global SIRT4 overexpression (SIRT4tg) and respective controls were fed a high fat (HFD) or low fat (LFD) diet for 12 weeks. Glucose tolerance was investigated in glucose tolerance tests (GTT) or using hyperglycemic clamp experiments. Insulin secretion was evaluated in pancreatic islet perifusion assays. Cardiac function and mass were evaluated by echocardiography or in isolated working heart experiments.

Results: HFD-fed SIRT4tg showed impaired glucose clearance in GTTs and needed lower glucose infusion rates to maintain blood glucose levels in hyperglycemic clamp experiments compared to HFD-fed controls, indicating impaired systemic glucose tolerance in HFD-fed SIRT4tg. In islet perifusion assays, islet cells from HFD-fed SIRT4tg showed an attenuated secretion of insulin in response to glucose stimulation compared to HFD-fed controls. Akt phosphorylation was unaltered in liver and skeletal muscle among groups. Bodyweight and tissue triglyceride levels (liver, muscle, heart, kidney) did not differ between SIRT4tg and controls following HFD. Compared to HFD-fed controls, heart weight-to-tibia length ratios as well as left ventricular mass calculated by echocardiography were increased in HFD-fed SIRT4tg. In contrast, left ventricular ejection fraction was preserved in all groups, and cardiac output and efficiency showed no difference in ex vivo isolated working heart experiments.

Conclusion: Overexpression of SIRT4 impairs systemic glucose homeostasis in HFD-fed mice and may promote cardiac hypertrophy in response to HFD, suggesting a potential role of SIRT4 in the development of diabetes mellitus and diabetic cardiomyopathy.

PS 15/15-2

The new myokine myonectin is significantly associated with type 2 diabetes in patients with peripheral artery disease

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Introduction: The novel myokine myonectin is predominantly expressed in skeletal muscle and is involved in the regulation of metabolic homeostasis. A putative association between myonectin and type 2 diabetes mellitus (T2 DM) has been discussed controversially in current literature. The association between myonectin and T2 DM in subjects with peripheral artery disease (PAD) has not been investigated at all and is addressed in the present study.

Methods: We measured myonectin in 224 patients with sonographically proven PAD, of whom 93 had T2 DM (41.5 %).

Results: Myonectin concentrations were significantly decreased in PAD patients with T2 DM compared to non-diabetic PAD patients (median 2.00, interquartile range [1.19–3.35] versus 2.63 [1.58–3.88] ng/ml; $p = 0.030$). Analysis of covariance revealed that the association between myonectin and T2 DM remained significant after adjusting for age, sex, body mass index, LDL-cholesterol, smoking, as well as systolic and diastolic blood pressure ($F = 5.42$; $p = 0.021$). Further, plasma myonectin significantly correlated with HbA1c (spearman's rho $= -0.164$; $p = 0.015$), but not with levels of fasting glucose or 2-hour glucose in oral glucose tolerance tests (rho $= -0.059$; $p = 0.387$ and rho $= -0.148$; $p = 0.053$, respectively).

Conclusion: We conclude that plasma myonectin levels are significantly associated with T2 DM in patients with PAD.

PS 15/15-3

Modulation of inflammation in patients with Fabry disease

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Introduction: Fabry disease (FD) is a lysosomal storage disorder and is caused by a mutation of the GLA gene, leading to a deficient production of the enzyme alpha-galactosidase A (αGAL A). As a consequence accumulation of globotriaosylceramide (Gb3) and globotriaosylsphingosine (lysoGb3) occurs and is responsible for the clinical manifestations in FD. [1] The deacylated Gb3, called lysoGb3, accumulates to a lesser extent compared to the primary substrate Gb3 and serves as a promising biomarker during monitoring the efficiency of therapy. [2]

Methods: Human endothelial cells were isolated as described previously [3]. Cells were treated with 25 μM and 50 μM Gb3 or lysoGb3 for 2 h. In addition preincubation for 1 h with an αGAL A inhibitor and subsequent treatment with Gb3 was conducted. To observe proinflammatory effects in vitro, qPCR for ICAM, VCAM, E-selectin was performed. To screen for a potential involvement of circulating miRNAs, samples of three Fabry patients before and after enzyme replacement therapy (ERT) were analyzed.

Results: Incubation of human endothelial cells with Gb3 or lysoGb3 led to mild proinflammatory effect. Results obtained from the qPCR showed a 1.5- to 2 fold upregulation of adhesion molecules, especially ICAM. Preincubation with the αGAL A inhibitor demonstrates no significant difference compared to cells which were only treated with Gb3 on the expression profile of VCAM, ICAM and E-selectin. Identification of the miRNA network of Fabry patients revealed that two members of the let-7 family were significantly upregulated after enzyme replacement therapy. Namely, the miRNAs let-7d-3p and let-7a-3p (p-value: 00.006498, 00.016466).

Conclusion: In inflammatory activated endothelial cells a wide variety of adhesion molecules are up-regulated. [4] A mild inflammatory effect on adhesion molecules was observed upon stimulation with Gb3/lysoGb3 fitting to previously published data. Upregulation of adhesion molecules in endothelial cells due to the accumulation of Gb3 and lysoGb3 might represent the basic mechanism behind the progression of the disease. It is widely known that the let-7 family is able to induce anti-inflammatory effects, as well as pro-inflammatory effects and is closely connected with endothelial function [4]. Hence, investigating the mechanism behind the let-7 miRNA in FD might allow to understand the role of miRNAs in progression disease in vitro.

PS 15/15-4

Cathepsin G bypasses the classic renin angiotensin system, leading to enhanced neutrophil extracellular trap formation

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Introduction: Previous studies associated elevated neutrophil extracellular trap (NET) formation with disease severity in atherosclerosis. We recently observed that Angiotensin-II (Ang-II) boosts NET formation, while angiotensin receptor blockers (ARB) decreased NETs in comparison to angiotensin converting enzyme inhibitors (ACE-I). We hypothesized that this difference is explained by Cathepsin G, a serine protease produced by neutrophil granulocytes and highly expressed by cells in atherosclerotic lesions, which has been shown to convert Ang-I into Ang-II even in the absence of ACE. We thus aimed to characterize the effect of Cathepsin G on NET formation and its natural counter mechanism DNase.

Methods: Blood samples were collected from the antecubital vein of healthy donors and patients. Neutrophils were isolated, stimulated to form NETs and made visible using Sytox or Pico Green. NETs were treated with DNase and Cathepsin G to measure degradation. CLS and femoral blood of STEMI patients ($n = 12$) was drawn during percutaneous coronary intervention. Cathepsin G concentration was measured by ELISA.

Results: Cathepsin G levels in CLS were significantly higher than in the peripheral site. Neutrophils treated with Cathepsin G and ionomycin did not show a difference in NET formation than neutrophils treated with only ionomycin. NETs treated with Cathepsin G and DNase had a significant decline in DNase activity in comparison to NETs only treated with DNase.

Conclusion: Cathepsin G seems to stabilize NETs via a decline in DNase activity. Cathepsin G levels appear to be elevated in CLS, which might influence atherosclerosis and progression of disease.

PS 15/15-5

Effects of Tenascin-C on left ventricular hemodynamics and high-energy phosphates levels in isolated rat hearts

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Introduction: Upregulation of Tenascin C (TN-C) upon myocardial infarction and heart failure is associated with cardiac dysfunction. More recently, we demonstrated that TN-C applied in H9c2 cardiomyoblasts resulted in a reduction of high-energy phosphate (HEP) concentration, such as ATP and phosphocreatine (PCr). However, the direct impact of TN-C on cardiac hemodynamics has not been investigated yet. This study aims to investigate the direct hemodynamic effects of TN-C on LV function in isolated perfused rat hearts.

Methods: Healthy male Sprague-Dawley rats (age 12–14 weeks) were used. The hearts were rapidly excised from the thorax and mounted on an erythrocyte-perfused, isolated working heart (WH) system. Then, either the recombinant TN-C (cumulative dosage: (80 ng/ml; $n = 3$)) or vehicle solution (0.9 % saline; $n = 3$) was injected into the coronary vasculature via a side arm. To evaluate cardiac function the following parameters were used: cardiac output (CO), left ventricular (LV) systolic pressure (LVSP), heart rate (HR), coronary flow (CF) and external heart work (EHW). In addition, HEP measurement was performed by liquid chromatography (HPLC) in LV myocardial biopsy taken at the end of experiment. Additionally, inflammatory cytokine expression in LV tissue was assessed by RT-qPCR.

Results: After 30–40 min of the 80 ng/ml cumulative dosage of TN-C the cardiac function was severely depressed. Correspondingly, the TN-C treated hearts showed a significant decrease in CO ($p < 0.001$), LVSP ($p < 0.05$) and EHW ($p < 0.01$) compared to their baseline and as well as in comparison to vehicle treated group ($p < 0.01$, respectively). These hemodynamic changes were accompanied by the reduction in ATP ($p < 0.05$) and PCr ($p < 0.05$) levels in TN-C treated hearts and a massive upregulation of TNF-alpha ($p < 0.001$) and IL1-beta ($p < 0.001$) compared to the vehicle treated group.

Conclusion: In summary, TN-C exerts direct hemodynamic effect on isolated rat hearts and is a potent myocardial depressant. These changes are accompanied by reduction in HEP and the upregulation of cytokine expression in the myocardium. Thus, the upregulation of TN-C after MI and heart failure promotes cardiac hemodynamic dysfunction and represents a therapeutic target.
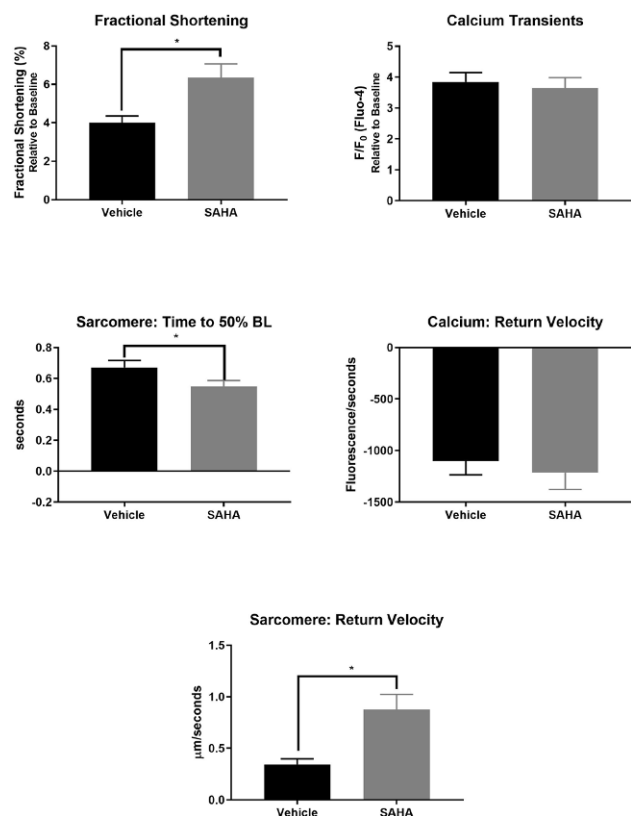
Fig. 1 | PS 15/15-6 Kinetics and Calcium transients in feline CM

PS 15/15-6

Effects of pan-HDAC inhibition on Ca2+-cycling and sarcomere-kinetics

M. Kasa[1], D. Eaton[2], S. Ljubojevic-Holzer[1], C. Koyani[1], D. von Lewinski[1], A. Zirlik[1], P. Angleitner[3], A. Zuckermann[3], A. Aliabadi-Zuckermann[3], S. R. Houser[4], P. Rainer[1], M. Wallner[1]

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Introduction: Heart failure (HF) with preserved ejection fraction (HFpEF) is an increasing worldwide health problem with no proven effective therapies. It was previously shown that inhibition of histone deacytelase catalytic activity improves cardiac systolic and diastolic function in rodent and feline models of HFpEF via hyperacetylation of a variety of proteins. The specific mechanisms underlying enhanced cardiac function are not fully understood. The aim of this study was to assess the effects of suberanilohydroxamic acid (SAHA), a pan-HDAC inhibitor, on calcium cycling and sarcomere kinetics in murine and feline ventricular cardiomyocytes.
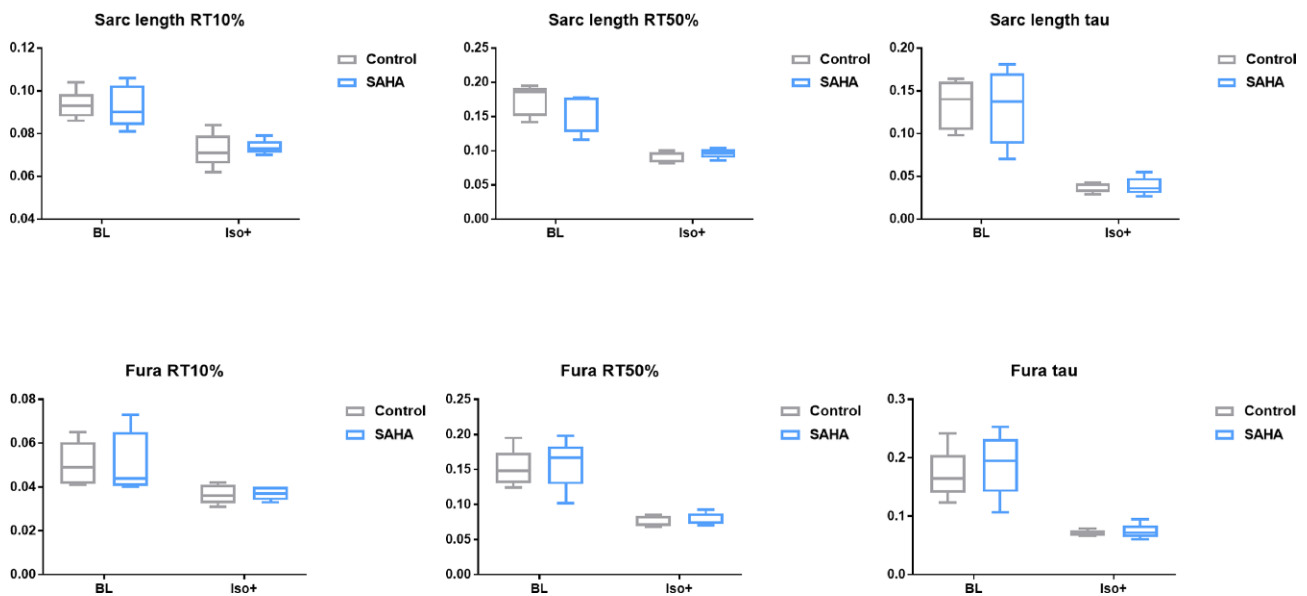
Fig. 2 | PS 15/15-6 Kinetics and Calcium transients in feline CM

Methods: Experiments were performed using ventricular cardiomyocytes (CM) isolated from healthy C57BL/6J mice ($n = 5$) and male domestic short hair cats ($n = 4$). Freshly isolated murine and feline CM were incubated with 5 µM or 2.5 µM SAHA respectively for 90 min and compared to untreated CM. Sarcomere shortening and cytosolic calcium were measured using an epifilureszenz microscope and calcium sensitive dyes while electrically stimulated.

Results: SAHA did not induce any changes in diastolic sarcomere length, amplitude or parameters of relaxation in murine CM, both at baseline and following exposure to isoprenaline. Furthermore, diastolic calcium levels, amplitude as well as RT50 and RT90 were unaffected by SAHA. In contrast, feline CM treated with SAHA had significant increases in sarcomere shortening and maximum sarcomere return velocity and decreased relaxation time RT50. However, calcium transients and kinetics were unchanged.

Conclusion: SAHA treatment resulted in a significant improvement in both contraction and relaxation characteristics of feline CM, without substantially altering calcium cycling. These findings may be explained by an increase in myofilament calcium sensitivity. The dichotomy between murine and feline CMs may be explained by differences in the myosin heavy chain (MHC) isoforms. The predominant MHC isoform in cats and human are beta-MHC, thus human CM could respond in a similar way to HDAC inhibition vs. mice which are predominantly alpha-MHC.

PS 15/15-7

Preventing CD62P-mediated neutrophil infiltration enhances thrombus resolution

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Introduction: Venous thromboembolism encompasses deep vein thrombosis and its complication pulmonary embolism and is a major health. Deep vein, fibrin-rich thrombi often occur in large veins of the legs and thrombus resolution is important to prevent embolic events. In early stages predominantly neutrophils and later monocytes and macrophages infiltrate the thrombus regulating its persistence via fibrinolytic responses, coagulation factors clearance and neutrophil extracellular trap formation. This immune cell accumulation is mainly mediated by selectins and adhesion molecules such as *P*-selectin (CD62P). CD62P is expressed on platelets and endothelial cells and interacts predominately with *P*-selectin glycoprotein ligand of leukocytes, which is crucial for leukocyte extravasation. Thus, we aim to define effect of CD62P-mediated leukocyte extravasation and activation on thrombus resolution.

Methods: By ligating the inferior vena cava stenosis was induced in mice and after 1 day, when the thrombus was formed a CD62P blocking antibody was applied. On day 3 the formed thrombus and the surrounding vessel were extracted and cryo-embedded or homogenized for flow cytometry to analyse leukocyte accumulation and activation. In addition thrombus resolution was monitored over 14 days by ultrasound measurements.

Results: Blocking CD62P significantly reduced the interaction of platelets with neutrophils as well as with Ly6Chigh monocytes. We found that this treatment led to a reduced neutrophil accumulation in the cranial part of the surrounding vessel wall as well as in the cranial part of the thrombus. Moreover, the total numbers of inflammatory Ly6Chigh macrophages were diminished in the total thrombus. This led to an enhanced thrombus resolution after 3 and 14 days, indicated by decreased thrombus length and weight in the treatment group.

Conclusion: Taken together, our data show that reducing neutrophils in the cranial part and inflammatory macrophages by targeting CD62P promotes thrombus resolution.

PS 15/15-8

Negative correlation of circular RNA CDR1as with miR-7 in a porcine model of myocardial infarction

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Introduction: Circular RNAs (circRNAs) are long non-coding RNAs characterised by the formation of a covalently closed loop. They lack a 3'- and a 5'end making them more stable than linear RNA because of their resistance to exonucleases. One of the various functions of circRNAs is the capability of regulating translation by binding micro RNAs (miRNAs). Recently, a rodent study showed that CDR1as aggravates myocardial infarction (MI) in mice by binding miR-7. Until now, this crucial information of CDR1as and its role in MI was only shown in small animal models. Our aim was to examine the miRNA sponge capacity of the circRNA CDR1as (repressing the miRNA function) in the translational large animal model of reperfused myocardial infarction.

Methods: 15 domestic female pigs underwent reperfused myocardial infarction by percutaneous balloon occlusion of the mid-LAD for 90 min, followed by reperfusion. After a 2 month follow-up, the animals were euthanized and myocardial tissue samples from the infarcted region were collected in RNAlater. RNase R was added to the RNA isolation to enrich circRNAs. RNA was isolated using a column-based chloroform extraction and reverse transcribed into cDNA to perform qPCR analyses. Divergent primers were designed allowing only the amplification of a circular transcript. The characteristic backsplice junction of circRNAs was confirmed via Sanger sequencing. Correlation of CDR1as expression with miR-7 expression was then calculated.

Results: RNase R treatment degraded linear transcripts and enriched circRNAs, indicating the high stability of circRNAs. We successfully detected CDR1as in pig hearts using qPCR (normalized gene expression: 1.14 ± 0.81) and confirmed the circularity of the qPCR product via Sanger sequencing. Gene expression of CDR1as and miR-7 shows a moderate negative correlation (–0.5,
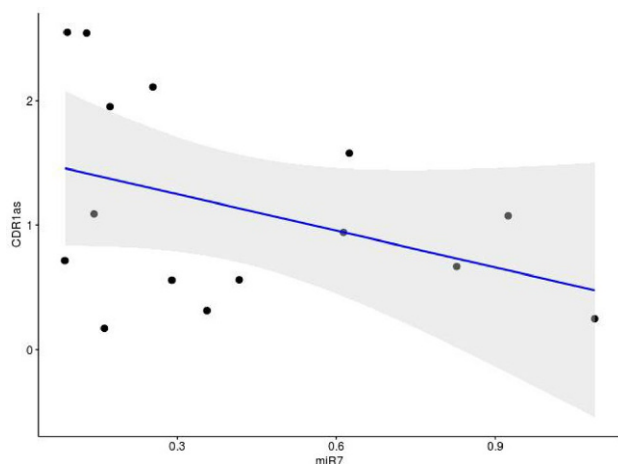
Spearman's rank correlation coefficient, $p = 0.062$), indicating a sponge function of CDR1as to miR-7 in pig hearts (Figure).

Conclusion: Our results confirm the sponging function of CD1as to miR-7 in a translational animal model of reperfused MI, suggesting a therapeutic target to reduce ischemia-reperfusion-induced myocardial injury.

POSTERSITZUNG 16 – CHIRURGIE 1

PS 16/16-1

Cardiac surgery in Jehovah's Witnesses: a propensity score analysis

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Introduction: The refusal of allogeneic blood transfusions in members of the Jehovah's Witness (JW) community represents a major challenge in case of complex surgical procedures. In the present retrospective study we reviewed our experience with JW patients undergoing cardiac surgery.

Methods: Between 2008 and 2017 a total of 35 adult JW patients (mean age 68.0 ± 9.5 years; median logistic EuroSCORE 5.8 %) underwent cardiac surgery at our department providing optimal patient blood management. Outcomes were compared with 35 non-JW patients who accepted blood transfusions applying propensity score matching.

Results: There were no significant differences in clinical and operative data between the groups. Twelve JW patients (34.3 %) received erythropoietin and iron preoperatively, resulting in an increase in mean hemoglobin of 2.0 g/dL. On admission, hemoglobin was 14.1 ± 1.1 g/dL in JW patients compared to 13.2 ± 2.0 g/dL in non-JW patients ($p = 0.02$). The calculated perioperative red blood cell loss was lower in JW patients than in non-JW patients (619 ± 420 mL vs 929 ± 520 mL; $p = 0.01$). Throughout the hospital stay, the hematocrit values in JW patients were significantly higher compared to the non-JW patients, although 51.4 % of non-JW patients received allogeneic packed red blood cells. At discharge hemoglobin was 11.5 ± 1.5 g/dL in JW patients and 10.3 ± 1.3 g/dL in non-JW patients ($p < 0.001$). In-hospital mortality was 2.9 % in each group (one patient). There were no differences regarding major complication rates and long-term survival.

Conclusion: By implementing optimal patient blood management, cardiac surgery in JW patients can be performed with low morbidity and mortality. Preoperative optimization of hemoglobin and minimization of perioperative blood loss are cornerstones in the prevention of blood loss, anemia, and transfusions.

Fig. 1 | PS 15/15-8 Scatter plot of CDR1as versus miR-7 in infarcted pig hearts

PS 16/16-2

Conduction disorders and permanent pacemaker implantation following transcatheter aortic valve replacement–a systematic review

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Introduction: With the wider adaption of Transcatheter Aortic Valve Replacement (TAVR) and despite all improvements in adverse events, concerns about short and long term impact of conduction disorders, that are associated with the procedure at a higher range, are remaining. The predisposing factors and outcome on mortality and morbidity have been poorly characterized so far.

Methods: A systematic PubMed-search was performed to give an overview on current data in TAVR regarding the predisposing factors, incidence and timing of conduction disturbances and indications for subsequent permanent pacemaker implantation (PPI), as well as on short and long term on morbidity and mortality

Results: Impairment of intra-cardiac conduction frequently occurs in transcatheter treatment of aortic valve stenosis, with a wide range of prevalence among different devices being used. Patient and procedure-related factors have been identified, that put candidates at higher risk to develop relevant conduction disorders. Baseline right bundle branch block, LVOT calcium load, type of device and depth of implantation are amongst the most significant. Patients with new conduction abnormalities and new pacemaker implantation are at higher risk for hospitalization due to heart failure, data on the outcome in mortality still remains controversial.

Conclusion: TAVR is an evolving procedure with consistent advances in procedural methods, devices, patient selection and utilization of resources. Conduction disorders and subsequent demand for PPI and its associated potential adverse effects on long-term outcome remain to be a controversial issue. As TAVR is likely to become the predominant treatment for severe symptomatic aortic stenosis in lower surgical risk classes, potential adverse events like reduced long-term device durability due to structural valve deterioration and impaired quality of life following progressive left ventricular heart failure after PPI will have to be taken into considerations, when counselling patients in their choice of treatment.

Abb. 1 | PS 16/16-3

Abb. 2 | PS 16/16-3

PS 16/16-3

Spezielle Indikationen für sondenlose Herzschrittmacher (Leadless pacing)

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Einleitung: Leadless pacing stellt eine Alternative zur herkömmlichen sondenbasierten Schrittmachertherapie dar. Auf Grund der hohen Kosten des Systems wird dieses für spezielle Indikationen eingesetzt. Wir sehen auf Grund der im MICRA Registry nachgewiesenen verminderten Infektionsanfälligkeit des Systems 2 Hauptindikationen: 1 Vorbestehende Infektion v. a. nach Sondenextraktion oder Dialyse 2. Fehlender venöser Zugang über die Vena cava superior (SVC).
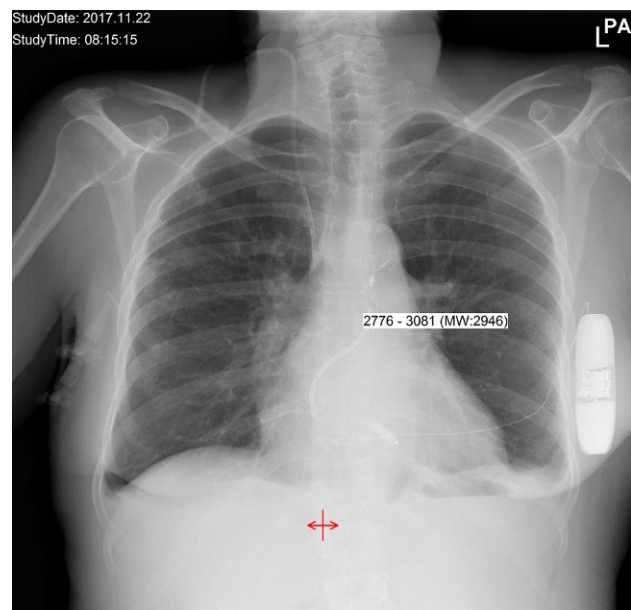
Methoden: An der klinischen Abteilung für Herzchirurgie in Graz wurden von 2017–2020 6 leadless Pacer MICRA VR der Firma Medtronic erfolgreich implantiert.. 4 Männer, 2 Frauen, mittleres Alter 70,5 Jahre. 4 Patienten nach Sondeninfektion 7–16 Tage nach Sondenextraktion (2 SM und 2 ICD). Die ICD Patienten erhielten dann noch zusätzlich einen subcutanen ICD ohne Probleme durch Interferenzen mit dem MICRA. 2 Patienten mit fehlendem Zugang bei Thrombose der V-subclavia und der SVC. Eine Implantation musste wegen einer Stenose der IVC abgebrochen werden.

Resultate: Intraoperativ waren bis zu 6 Positionierungen notwendig, bis eine stabile Lage mit guten Messwerten erreicht wurde. Postoperativ kam es zu keinen Komplikationen, die Schrittmacherkontrolle zeigte normale Werte und es kam zu

keinen Rezidivinfektionen des MICRA im Follow up bis zu drei Jahren. 1 Patient verstarb an einer progredienten Sepsis bei Vorfußgangrän.

Schlussfolgerungen: Der sondenlose Schrittmacher MICRA stellt eine vielversprechende Alternative zur konventionellen Schrittmachertherapie nach Infektionen dar und kann auch mit subcutanen ICD kombiniert werden. Als weitere Indikation sehen wir die Thrombose der Venen der oberen Körperhälfte. Ein Nachteil ist, dass das System derzeit nur als Einkammersystem zur Verfügung steht.

PS 16/16-4

Aortic dissection type A in the young: should we always treat the root?

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Introduction: The objective was to evaluate the outcome of young patients suffering from acute aortic dissection type A (AADA) and the risk of retained proximal aortic tissue following root-sparing techniques for aortic reoperation or death.

Methods: We retrospectively reviewed our hospital database for patients aged 45 or younger who suffered from AADA ($n = 70$; median age 40 years [IQR 36y; 43y]).

Results: Eleven patients (16 %) had an underlying genetic aortopathy, 14 patients (20 %) a bicuspid aortic valve, 17 patients (24 %) an arch anomaly. Moderate to high aortic regurgitation was present in 41 patients (59 %) preoperatively. In 47 % ($n = 33$) the aortic root was replaced. 8 (11 %) patients died during follow-up, 4 (6 %) within 30 days postop. In 12 patients (17 %) redo surgery or intervention was necessary. Median follow-up time was 6 years [IQR 3y; 10y]. Mean aortic growth rates in the untouched segments were 4.7 mm for the aortic root; 3.6 mm for the arch; 4.6 mm in the descending and 4.3 mm in the infrarenal aorta. Root treatment strategy had no impact on reoperation rate ($p = 0.97$), and long-term survival ($p = 0.67$).

Conclusion: In young patients growth rate of the untouched root is slow, secondary root events are low. Liberal root replacement is not indicated despite young age.

PS 16/16-5

Pulmonary valve replacement with decellularized homografts. A single-center experience

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Introduction: Decellularized homografts (DH) have been used since the 2000s and have already shown great outcome in younger patients. In addition, spontaneous recellularization of DH was observed. Therefore, the major advantages of the DH are not only freedom of lifelong anticoagulation, no immunologic rejection and good hemodynamic characteristics but also a possible regeneration of the valve on the homograft matrix. The aim of this work was to evaluate the performance of decellularized pulmonary homografts implanted in our center.

Methods: Vienna General Hospital took part in the multicentrer European Trial ESPOIR (European Clinical Study for the Application of Regenerative Heart Valves). The aim was to evaluate the safety and efficacy of decellularized pulmonary homografts (DPH) in terms of hemodynamic performance, valve related adverse events and survival. Each patient received a decellularized pulmonary homograft and and has been followed up in our center after the surgery.

Results: Since 2015, we enrolled 59 patients, mean age 32.2 ± 13.2 years (8 to 58 years). Fifteen patients (25 %) previously underwent pulmonary valve replacement. Mean diameter of the implanted DPH was 25.6 ± 6.7 mm, mean bypass and cross-clamp time was 159.7 ± 70.6 min and 135.4 ± 47.1 min respectively. Mean follow up is 9.9 months, up to 43.7 months. Concomitant procedures were performed in 47 (79.6 %) patients, of which 28 (47.5 %) were Ross-operations. Intraoperative, a 2nd cross-clamp time due to moderate aortic valve regurgitation of the implanted autograft, was necessary in one patient (1.7 %). The perioperative mortality was 1.7 % ($n = 1$); in this case, a pre-existing antiphospholipid antibody syndrome was associated with postoperative bleeding events, ECMO implantation and finally multiorgan failure. Two patients (3.4 %) underwent a reoperation with homograft explanation due to structural valve deterioration (stenosis 11 months after implantation of the homograft in both cases). Mean postoperative gradient of the pulmonary homograft was 8.8 mm Hg, pulmonary regurgitation occurred in 13 (22 %) cases: 12 trace, 1 mild. No other adverse events including late mortality, valve thrombosis or non-structural dysfunction have been observed.

Conclusion: Early results showed a low rate of perioperative complications and good hemodynamic characteristics of DPH at our center.

PS 16/16-6

Defining a therapeutic range for regeneration of ischemic myocardium via shock waves

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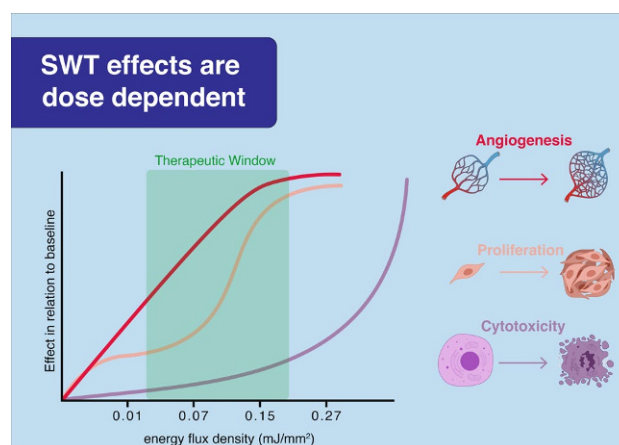
Fig. 1 | PS 16/16-6

Introduction: Shockwave therapy (SWT) represents a promising regenerative treatment option for patients with ischemic cardiomyopathy. Although no side-effects have been described upon SWT, potential cellular damage at therapeutic energies has not been addressed so far. In this work, we aimed to define a therapeutic range for shock wave application for myocardial regeneration.

Methods: Both in vitro and vivo experiments were performed to asess ideal shock wave intensities. First, human umbilical vein endothelial cells (HUVECs) were treated with 300 impulses with an an energy flux density of 0.01, 0.07, 0.15 and 0.27 mJ/mm^2 at a frequency of 5 Hz as described previously. Subsequently, cytotoxicity, proliferation and angiogenic gene expression were measured via necrosis assay, western blotting, proliferation assay and qPCR. In a next step, hind limb ischemia was induced in C57/BL6 mice as described previously. After application of shock waves with equal energy flux density as in vitro, limb perfusion was measured using laser doppler perfusion imaging at different time points. In addition, histological sections were obtained and stained for neovascularization.

Results: We could demonstrate that SWT does not induce cellular damage beneath energy levels of 0.27 mJ/mm^2 total flux density. Endothelial cell proliferation, angiogenic gene expression and phosphorylation of AKT and ERK are enhanced in a dose dependent manner until 0.15 mJ/mm^2 energy flux density. SWT induces regeneration of ischemic muscle in vivo via expression of angiogenic gene expression, enhanced neovascularization and improved limb perfusion in a dose-dependent manner.

Conclusion: Therefore, we provide evidence for a dose-dependent induction of angiogenesis after SWT, as well as the absence of cellular damage upon SWT within the therapeutic range. These data define for the first time a therapeutic range of SWT, a promising regenerative treatment option for ischemic cardiomyopathy.

PS 16/16-7

Chirurgische Therapie bei Endokarditis nach interventionellem oder chirurgischem Pulmonalklappenersatz

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Einleitung: Pulmonalklappenkonduits und Transkatheter-Pulmonalklappen stellen ein erhöhtes Risiko für eine Pulmonalklappenendokarditis dar. Bei Kindern und Erwachsenen mit angeborenem Herzfehler ist jedoch häufig eine Implantation eines Konduits zwischen rechtem Ventrikel und Pulmonalarterie (RVPAC) notwendig. Prothesenendokarditiden stellen eine konservativ kaum beherrschbare, lebensbedrohliche Situation dar. Die Ergebnisse der chirurgischen Behandlung sollen aufgezeigt werden.

Methoden: Dreizehn PatientInnen (7 Kinder, 6 Erwachsene) mit Pulmonalklappenendokarditis nach Pulmonalklappenersatz wurden zwischen Jänner 2009 und Februar 2020 einem RVPAC Wechsel zugeführt. Medianes Alter war 17 Jahre [3–32], 4 PatientInnen waren weiblich (30,8 %). Primäre Diagnose war bei acht PatientInnen eine Fallot'sche Tetralogie oder Double outlet right Ventricle, bei drei ein Truncus arteriosus communis und zwei Patienten erhielten aufgrund eines Aortenklappenvitiums primär eine Ross-Operation. Medianes Intervall zwischen RVPAC oder Transkatheter-Pulmonalklappen Implantation und chirurgischer Therapie der Endokarditis war 4,2 Jahre [0,25–16,7]. Die endokarditisch befallene Klappe war in fünf Fällen eine Transkatheter-Pulmonalklappe (Melody®) in weiteren vier ein boviner RVPAC (Contegra®), in drei ein Homograft und eine xenogene Pulmonalklappe (Matrix Plus ®). Das Studiendesign gestaltete sich retrospektiv in einem Single Center.

Resultate: Es fand keine Krankenhausmortalität statt. Das gesamte befallene prosthetische Material und Reste des Pulmonalarterienhauptstammes bis zur Bifurkation wurden aus dem rechtsventrikulären Ausflusstrakt entfernt. In allen Fällen wurde ein orthotoper pulmonaler Homograft implantiert. In einem Fall erfolgte zusätzlich eine Embolektomie septischer Vegetationen aus der Lungenarterie. Begleitende Eingriffe waren jeweils einmal ein Aortenwurzelersatz, ein VSD-Patch Wechsel, eine Trikuspidalklappenreparatur und ein PFO Verschluss. Mittlere Bypasszeit war 193 min [67–561]. Die mediane Aufenthaltsdauer auf der Intensivstation betrug 3 Tage [1–24]. Bei einem Patienten musste postoperativ aufgrund einer Nachblutung eine Rethorakotomie durchgeführt werden. Es zeigten sich keine neurologischen Komplikationen und eine postoperative ECMO Therapie war bei keinem der PatientInnen notwendig. Die mikrobiologische Auswertung ergab als häufigste Erreger verschiedene Streptokokken Spezies und in einem Fall eine invasive Aspergillose.

Schlussfolgerungen: Durch die stetige Zunahme von PatientInnen mit Pulmonalklappenersatz aufgrund angeborener Vitien ist eine steigende Anzahl von Pulmonalklappenendokarditiden zu erwarten. Die chirurgische Therapie bei Endokarditis nach RVPAC oder Transkatheter-Pulmonalklappen Implantation stellt eine sichere und effektive Therapie dar. Eine frühzeitige chirurgische Vorstellung bei Verdacht auf Pulmonalklappenendokarditis sollte daher angestrebt werden, um Folgekomplikationen wie Rechtsventrikelversagen, septische Lungenembolien, intrakardiale Ausbreitung der Endokarditis und Antibiotikaresistenzen zu vermeiden.

POSTERSITZUNG 17 – ASSISTENZPERSONAL

PS 17/17-1

Die Implementierung des Projektes „Kardiologisches Beratungsgespräch – Pflege für Patient*innen und Angehörige" – Eine Evaluationsstudie

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Einleitung: Gesundheitsinformationen können chronisch Erkrankten helfen, eigene Entscheidungen zu treffen und die Auswirkungen der Erkrankung im Alltag besser zu bewältigen

[1,2]. Vor diesem Hintergrund wurde das Projekt „Kardiologisches Beratungsgespräch – Pflege für Patient*innen und Angehörige" initiiert. Das Ziel des Projektes liegt darin, die Gesundheitsinformationsmaterialien für die Erkrankten und ihre Bezugspersonen bereitzustellen und ihnen ein persönliches Beratungsgespräch anzubieten. Ziel der vorliegenden Studie ist es, den Bedarf der Erkrankten und ihrer Bezugspersonen an dem Beratungsgespräch und die wesentlichen Themen des Beratungsgespräches darzustellen sowie auch die Durchführbarkeit des Projektes zu evaluieren.

Methoden: Um das Ziel der Studie zu erreichen, wurde ein Mixed-Methods Studiendesign gewählt. Die Datenerhebung erfolgt mittels Patientendokumentation und eines Evaluationsbogens. Die erhobenen Daten wurden mittels deskriptiver Statistik ausgewertet. Die Auswertung der schriftlichen Feedbacks der Betroffenen erfolgt inhaltsanalytisch nach Mayring [3].

Resultate: Die Ergebnisse belegen eine hohe Zufriedenheit der Erkrankten und ihrer Bezugspersonen mit diesem Beratungsgespräch. „Körperliche Aktivität nach dem Herzinfarkt", „Notwendigkeit der Rehabilitation", „Die Strategie bei der Notfallsituation", „Gesunde Ernährung", „Funktion des Herzens", „Raucherentwöhnung", „Einnahme der Medikation" und „Stressbewältigung" sind die im Vordergrund stehenden Themen des Beratungsgespräches. Die Ergebnisse machen auch deutlich, dass die vorgegebenen Ziele des Projektes mit zur Verfügung stehenden Personalressourcen erreicht wurden.

Schlussfolgerungen: Als Fazit ergibt sich, dass das Projekt – „Kardiologisches Beratungsgespräch – Pflege" eine wertvolle Einrichtung für die Erkrankten und ihre Bezugspersonen ist. Es

Abb. 1 | PS 17/17-1

Abb. 2 | PS 17/17-1

zeigt sich ein steigender Bedarf für die Weiterführung und den Ausbau des Beratungsgespräches in der klinischen Versorgung.

PS 17/17-2

Die pflegerische 360 Grad Betreuung der S-ICD Patienten – eine Erfolgsgeschichte

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Einleitung: Bei der Implantation eines subkutanen Defibrillators (S-ICD) sind im Vergleich zum konventionellen, transvenösen ICD System einige zusätzliche Dinge zu beachten. Speziell in der Pflege kommen hier wichtige Aufgaben hinzu, um eine erfolgreiche ganzheitliche Betreuung zu ermöglichen. Dies gilt vor allem für die pflegerische Begleitung von Patienten, die eine S-ICD Implantation unter Anwendung von Regionalanästhesie erhalten. Im KH Nord werden diese Patienten von Anfang an multidisziplinär betreut und aus pflegerischer Sicht auf ihrem Weg begleitet, beginnend beim Erstkontakt bis hin zu Nachkontrollen über die gesamte Lebensdauer.

Methoden: Der Weg des Patienten beginnt bei der Aufnahme und Vorbereitung auf seine S-ICD Implantation. Um schon vorab den Erfolg der Implantation sicherzustellen, erfolgt ein Screening, bei welchem über Elektroden am Patienten überprüft wird, ob die Signalqualität ausreichend ist. Nach erfolgreichem Screening folgt die Patienten-Aufklärung, welche auch aus pflegerischer Sicht viele Herausforderungen mit sich bringt. Die Implantation unter Anwendung von regionalanästhetischen Blöcken (Parasternalblöcke und Serratus P. Block) ist eine Methode, die in Österreich derzeit ausschließlich im KH Nord angewendet wird. Eine große Rolle dabei spielt das Schmerzmanagement des Patienten. Hierfür wird von der Pflege ein Pain Assessment Tool (Comparative Pain Scale Chart) angewandt, um aussagekräftige Langzeit-Daten über den Patientenkomfort vor, während und nach der Prozedur zu sammeln.

Resultate: Erste kurzfristige Ergebnisse zeigen die Tendenz, dass die Patienten geringen Schmerz empfinden, dieser allerdings im Großteil der Aktivitäten des täglichen Lebens nicht hinderlich ist und auch keine psychologischen Auswirkungen zur Folge hat. Die pflegerische Rundum-Betreuung schließt sich mit der Entlassungskontrolle einen Tag nach Implantation und den Nachkontrollen über die weitere Lebensdauer des Patienten. Eine optimale und anhaltende pflegerische Nachbetreuung in Hinblick auf Patienteninformationen, dem Sicherstellen der Funktionalität des S-ICDs und einem Wund- sowie Schmerzmanagement kann ein langfristiges Wohlbefinden des Patienten sicherstellen und zeigt die immer wieder kehrenden Aufgaben in der langfristigen 360 Grad Betreuung des Patienten.

Schlussfolgerungen: Im KH Nord werden die Patienten umfassend multidisziplinär betreut. Mit einem genaueren Blick auf die einzelnen Disziplinen lässt sich schlussfolgern, dass die Pflege hier sowohl bei der Betreuung des Patienten, als auch bei der Organisation der multidisziplinären Zusammenarbeit eine Schlüsselrolle spielt.
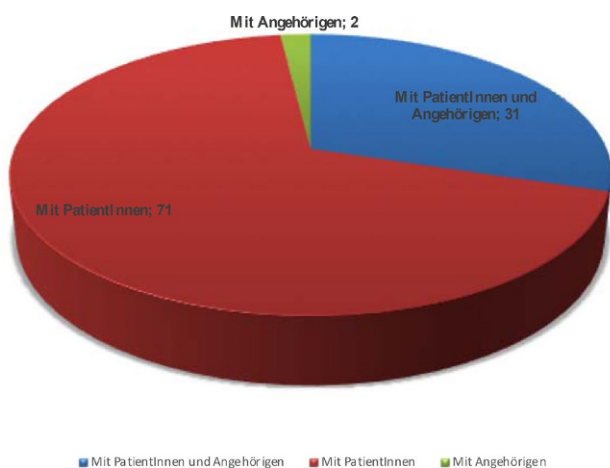
PS 17/17-3

Tagesklinische PatientInnenversorgung – Konzeptentwicklung und Umsetzung 2019

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Einleitung: Eingriffe im Herzkatheterlabor werden immer weiter optimiert. Durch die zahlreichen durchgeführten Studien der letzten Jahre werden best-practice Empfehlungen in die Praxis implementiert und die Sicherheit der Eingriffe für die PatientInnen verbessert sich stetig. Internationale Untersuchungen zeigen, dass daher auch die tagesklinische Versorgung der PatientInnen nach einem kardiologischen Eingriff zunimmt, daraus resultiert wiederum eine bedeutende Einsparung an Kosten. Die tagesklinische Versorgung und vor allem die Gewährleistung der Sicherheit der PatientInnen auch nach der Entlassung, erfordert ein strukturiertes Programm. Dazu wurde das „Ambulante Herzkatheter" Konzept erstellt. Ziele waren, die Aufnahmetermine übersichtlich zu dokumentieren, einen systematischen Aufnahmeprozess zu entwickeln, die Optimierung der Herzkatheter-Indikationsprüfung, und eine Verkürzung des stationären Aufenthaltes der PatientInnen.

Methoden: Das Konzept beinhaltet, dass für die tagesklinische Versorgung generell PatientInnen in Frage kommen, bei denen ein Zugang über die a. radialis, aber auch v. femoralis geplant ist. Zu den Eingriffen zählen daher Koronarangiographien, die präoperative Statusbestimmung der Koronargefäße vor Transplantationen, Rechtsherzuntersuchungen und Myocardbiopsien. Im ersten Schritt werden die externen Zuweisungen von InterventionistInnen bearbeitet und anschließend ein ambulanter Evaluierungstermin vergeben. Bei diesem Termin findet ein ausführliches Anamnesegespräch mit den PatientInnen statt und Vorbefunde werden gesammelt. Außerdem werden die notwendigen Untersuchungen durchgeführt. Danach wird entschieden, ob die PatientInnen für eine stationäre oder tagesklinische Aufnahme in Frage kommen, beziehungsweise, ob der Herzkatheter Eingriff überhaupt nötig ist. Zwei Wochen nach dem Evaluierungstermin erfolgt die Aufnahme, die Untersuchung im Herzkatheterlabor findet am selben Tag statt. Stationäre PatientInnen werden nach dem Eingriff zurück auf die Station transferiert und je nach Befund ein bis zwei Tage später entlassen. Tagesklinische PatientInnen kommen nach dem Eingriff zurück auf die Tagesklinik, dort erfolgt die fachgerechte Versorgung und Entfernung des TR-Bandes und am Nachmittag die Entlassung durch KardiologInnen.

Resultate: Im Jänner 2019 ist das Konzept in die Umsetzungsphase übergegangen. 1080 PatientInnen kamen zu Evaluierungsterminen in die Kardiologie Ambulanz. Insgesamt wurden 514 PatientInnen von der Tagesklinik in das Herzkatheterlabor zugewiesen.

Schlussfolgerungen: Das Konzept „Ambulante Herzkatheter" konnte 2019 erfolgreich implementiert werden. 2020 wurde es evaluiert und Verbesserungen im Ablauf und den Rahmenbedingungen ausgearbeitet. In Zukunft wird die Umsetzung weiter optimiert und die tagesklinische Versorgung der PatientInnen ausgebaut.

PS 17/17-4

Ermittlung des Herzzeitvolumens (HZV) im Rahmen einer Rechtherzkatheter-Untersuchung

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Einleitung: Problem: Im Rahmen einer HZV-Bestimmung entstehen viele Fehlerquellen, welche das Messergebnis beeinflussen (z. B. Menge oder Temperatur der Indikatorlösung). Durch den Einfluss dieser vielen Faktoren kann es zu großen Schwankungen der ermittelten Werte kommen. Fragestellungen: a) Welche Faktoren können bei der Ermittlung des Herzzeitvolumens Einfluss nehmen? b) Welche Aufgaben und Herausforderungen entstehen für diplomiertes Pflegepersonal und RadiologietechnologInnen?

Methoden: a) Durch eine qualitative Inhaltsanalyse von Fachliteratur wurden Einflussfaktoren in der Ermittlung des Herzzeitvolumens identifiziert und hinsichtlich ihrer Auswirkungen betrachtet. b) Durch Leitfadeninterviews mit erfahrenem Personal eines Herzkatheterlabors eines Landeskrankenhauses wurden die Aufgabenbereiche und entstehende Herausforderungen für RadiologietechnologInnen und diplomiertes Pflegepersonal ermittelt.

Resultate: Aus der qualitativen Inhaltsanalyse ging hervor, dass Einflussfaktoren wie der Atemzyklus des Patienten, Menge und Temperatur der Indikatorlösung, pathologische Gegebenheiten wie Trikuspidalinsuffizienz oder intrakardiale Shunts, sowie Veränderungen der Grundtemperatur durch Hypothermie oder hohe intravenöse Infusionsraten auftreten und Messungenauigkeiten verursachen können. Die Kurvenauswahl zur Mittelung der gemessenen Werte ist ebenfalls ein beeinflussender Faktor. Auch verschiedene Charakteristika von durchführenden MedizinerInnen wie Greifkraft oder BMI können eine Auswirkung auf die Messung haben, den größten Einfluss jedoch hat die praktische Erfahrung der durchführenden Person. Für DKGPs und RTs entstehen während einer HZV-Ermittlung viele Aufgaben (Vorbereitung der verwendeten Materialien, gutes Zeitmanagement, ...), welche allesamt mit entsprechender Achtsamkeit durchgeführt werden sollten, um eine bestmögliche Messgenauigkeit zu erreichen. Laut den Aussagen der Interview-Probanden hat das Assistenzpersonal den größten Einfluss auf das Ergebnis einer HZV-Messung. Der Einfluss des Patienten selbst ist vergleichsweise klein. Die wichtigste Grundlage bei der Arbeit im Herzkatheterlabor ist jedoch eine gute Kommunikation und Zusammenarbeit aller dort tätigen Berufsgruppen.

Schlussfolgerungen: Im Rahmen einer HZV-Ermittlung können sehr viele Einflussfaktoren entstehen, welche hauptsächlich durch das durchführende Personal verursacht werden. Darum ist eine entsprechende Schulung der mitwirkenden Personen besonders wichtig, um über die Auswirkungen der entsprechenden Fehlerquellen Bescheid zu wissen und diese so gut als möglich vermeiden zu können. Die wichtigsten Grundlagen für das Personal sind entsprechende Kenntnisse über Notfallmanagement, technische Kenntnisse sowie eine gute Kommunikation und Zusammenarbeit aller Arbeitsbereiche. Durch zunehmende praktische Erfahrung kann jedoch die beste Grundlage für eine effiziente Arbeitsweise gelegt werden.

PS 17/17-5

ECMO im Herzkatheter – eine interdisziplinäre Herausforderung

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Einleitung: Bis Dato gibt es drei große Studien, durchgeführt von Zapol et al., Morris et al. und Peek et al., mit insgesamt 310 eingeschlossenen PatientInnen, welche die extracorporale Membranoxygenierung (ECMO) untersuchten. In allen drei Studien konnte eine signifikante Verbesserung der Überlebensrate ohne schwere Behinderung nach 6 Monaten nachgewiesen werden In Graz wurden seit Einführung des Systems vor einem Jahr an der Universitätsklinik für Innere Medizin (UKIM) 7 PatientInnen mit venoarteriellen (va)-ECMOs im Herzkatheterlabor versorgt. Bei drei dieser PatientInnen konnte ein positiver Outcome bezüglich der Mortalität erlangt werden.

Methoden: Die ECMO wird dabei stets als Bridgingverfahren, nie als Therapieziel gesehen und kommt zur Überbrückung eines kardiogenen Schocks, bei St.p. CPR, sowie einer Pulmonalen Arteriellen Embolie (PAE). Das Assistsystem kann als bridge to recovery, to HTX, to LVAD oder to decision genutzt werden. Grundsätzlich besteht die ECMO aus drei Komponenten: Kanülen und Heparin beschichtete Schläuche für Blutentnahme und -rückgabe, (zentrifugal) Pumpe mit Steuereinheit und Oxygenator mit Wärmetauscher. Mit diesen Bestandteilen kann man nicht nur den Gasaustausch gewährleisten, sondern auch annähernd das gesamte Herz-Zeit-Volumen (HZV) des PatientInnen aufrechterhalten.

Resultate: Wird im Herzkatheter eine schockierte bzw. reanimierte PatientIn angekündigt, wird der interne Entscheidungs-Algorithmus vom Arzt zu Hilfe gezogen. Steht fest das die/der PatientIn eine ECMO bekommt müssen bestimmte vorbereitende Maßnahmen getroffen werden: Die Konsole und das zugehörige Material muss von der Intensivstation aufgerüstet und ins Herzkatheterlabor gebracht werden. Zusätzlich wird der Intensivturm (Beatmung, Monitoring und Perfusoren) und das Ultraschallgerät mit Linearschallkopf. Aus Gründen des erleichterten Workflows empfiehlt es sich die Gerätschaften vor dem Eintreffen der PatientIn richtig zu positionieren, da so die weiterführende Versorgung der PatientIn erleichtert wird. Das Assistenzpersonal übernimmt dabei sterile und unsterile Aufgaben. Am Tisch werden alle Materialien gespült, geprüft und vorbereitet. Der unsterile Beidienst behält die Vitalfunktionen der PatientIn im Auge (EKG, SpO2, Blutgase etc.) und bereitet alle geforderten Medikamente zu. Nach dem Eingriff werden die PatientInnen mit Hilfe eines Rollboards in das Intensivbett umgelagert. Dabei wird neben dem Tubus, ein besonderes Augenmerk auf die ECMO-Schläuche gelegt. Eine Ärztztin steht kopfseitig und fixiert den Tubus, eine zweite Ärztin ist nur für die Fixierung der Schläuche zuständig.

Schlussfolgerungen: Zusammenfassend kann gesagt werden, dass die PatientInen von der Therapie potenziell profitieren. Aufgrund des hohen Aufwandes der Implantation und auch der Nachbetreuung, ist ein gut geschultes Team unerlässlich. Zum optimalen und komplikationsarmen Arbeitsablauf wird das komplette interdisziplinäre Team regelmäßig auf die Gerätschaften und die Situationen geschult. Für uns wäre es optimal in Zukunft notfallsmäßige ECMO-Implantationen in unserem hauseigenen Simulationszentrum zu üben.

PS 17/17-6

Herzensbildung – Strukturierte PatientInnen-Information vor Spitalsentlassung

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Einleitung: Herzkranke PatienInnen werden im Krankenhaus multiprofesionell einer Diagnose und Therapie unterzogen. Aufgrund der immer kürzeren Liegedauer fehlt zunehmend Zeit für eine ausführliche Patientenaufklärung. Ziel ist es, Patientinnen aufzuklären bezüglich Risikofaktoren, Untersuchungs- und Behandlungsmöglichkeiten. Dabei sollen sie unterstützt werden, im Gespräch die richtigen Fragen zu stellen, auf den Umgang mit Risikofaktoren und der Krankheit selbst sowie möglichen Folgen im Alltag aufmerksam machen und auf Beratungs- und Hilfsangebote hinzuweisen.

Methoden: Seit Anfang 2018 werden an der 5. Medizinischen Abteilung/Kardiologie einmal wöchentlich Schulungen für Herzkranke (vorwiegend Herzkranke) abgehalten. An diesen Treffen nehmen PatientInnen sowie vereinzelt auch Angehörige teil. Vortragende sind DGKP, die neben Deutsch auch fließend BKS spricht, sowie ein Arzt, der zusätzlich türkisch beherrscht. Die Schulungen werden 1/Woche durchgeführt, dauern 60 min (plus 30 min für diverse persönliche Nachgespräche). Die Gruppengröße umfasst maximal 15 Personen. Zur Weiterführung des Projektes wurde das Institut für Frauen- und Männergesundheit/FEM Süd und MEN von der Magistratsabteilung 24/Wiener Gesundheitsfonds beauftragt und wird aus den Mitteln der Wiener Landeszielsteuerung finanziert.

Resultate: Im Jahr 2019 haben insgesamt 569 Teilnehmer in 52 Schulungstermine zu ihrem Nutzen besucht (444 PatientInnen und 125 Angehörige). Neben der Erklärung über Krankheitszustände sowie ihre Ursachen, die Ergebnisse von Untersuchungen wie Echokardiographie oder Computertomographie, steht das Interesse an Behandlungsmethoden im Vordergrund. Auf einzelne Laborbefunde wie z. B. das LDL-Cholesterin oder HbA1C, die Wichtigkeit einer guten Blutdruckeinstellung, des kompletten Nikotinverzicht, sowie einer Gewichtsreduktion durch Bewegung und Ernährungsumstellung wird hingewiesen. Kleine Broschüren mit der Möglichkeit, Messwerte im Verlauf zu dokumentieren, unterstützen das eigene Verantwortungsgefühl und die Kontrolle.

Schlussfolgerungen: Wir haben mit diesem Projekt bisher ausgezeichnete Erfahrungen gemacht, inzwischen weisen auch niedergelassene KardiologInnen zu unseren Donnerstag-Terminen in das KFJ zu.

PS 17/17-7

Delir an einer IMC – Auswirkung auf die Betreuungsqualität

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Einleitung: Hypothese: Patientinnen und Patienten mit dem Krankheitsbild Delir, einer akuten psychiatrischen Erkrankung, werden an IMC's durch das Fehlen von psychiatrisch ausgebildeten Pflegepersonal nicht bestmöglich versorgt.

Methoden: Einleitung: Einer der häufigsten akuten psychiatrischen Erkrankungen im intensivmedizinischen Bereich

stellt das Delir dar. Es kann aber auch auf anderen Abteilungen beobachtet werden. Das Auftreten dieser Erkrankung hat nicht nur negative Auswirkungen auf das Patientenoutcome sondern vermindert auch die Überlebenschance. Die Prävalenz bei beatmeten Patientinnen und Patienten liegt bei 60–82 % und bei nicht beatmeten Patienten bei 40–60 % auf einer Intensivstation. Zudem ist die Behandlung dieser Erkrankung und deren Folgen mit mehr Arbeitsaufwand von Pflegepersonen verbunden. Das Delir beeinflusst nicht nur den Gesundheitszustand und den Krankheitsverlauf, sondern auch die Lebensqualität und die Alltagsbewältigung der Patientinnen und Patienten.

Resultate: Methodik: Eine ausführliche systematische Literaturrecherche wurde betrieben, um relevante wissenschaftliche Literatur zu finden. Es wurden sowohl elektronische Datenbanken als auch Onlinebibliotheken in den Suchprozess einbezogen. Ebenfalls wurde eine Krankengeschichte eines Patienten XY mit einbezogen, um praktische Bezüge herzustellen.

Schlussfolgerungen: Ergebnisse: Die Ergebnisse zeigen, dass die GuKP durch verschiedene Maßnahmen das Krankheitsbild und dessen Symptomatik reduzieren kann. Ebenfalls konnte aufgezeigt werden, dass es einen Bedarf an spezifischer Weiterbildung in Bezug auf das Krankheitsbild Delir gibt. Es konnte ebenfalls festgestellt werden, dass ein Bedarf an speziellen Stationen vorhanden ist, die sich um Patientinnen und Patienten mit psychiatrischen Vorerkrankungen und einem akuten Delir kümmern.

PS 17/17-8

Stressbewältigung in der Intensivpflege

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Einleitung: Im Rahmen der Bachelorarbeit wurden die Stresstheorien nach Selye und Lazarus behandelt, um Stress und Stressoren zu definieren, die bei der Versorgung kritisch kranker IntensivpatientInnen von hoher Relevanz sind. Stressoren sind Reize, die eine Stressreaktion auslösen und können als psychische und physische Reaktion einer Person auf Stress definiert werden. Stressreaktionen zeigen sich bei IntensivpatientInnen z. B. als erhöhte Atemfrequenz, erhöhter Blutdruck, Unruhe, oder Einschlafstörungen. Besonders erwachende IntensivpatientInnen befinden sich in einer Extremsituation, die besondere Abwehr- und Anpassungsprozesse erfordert und stellt für diese eine emotionale Belastung und deren Bewältigung eine große Herausforderung dar. Für das Pflegepersonal ist der Stress der PatientInnen ebenfalls eine Herausforderung, da er einen negativen Einfluss auf die Genesung hat. Der derzeitige Wissensstand über evidenzbasierte pflegerische Maßnahmen zur Reduzierung von Stress bei wachen PatientInnen auf der Intensivstation ist allerdings begrenzt.

Methoden: Ziel der Arbeit war, pflegerische Maßnahmen zur Stressreduktion bei wachen IntensivpatientInnen, die mit hoher Evidenz bewertet wurden, zu identifizieren. Welche evidenzbasierten pflegerischen Maßnahmen zur Stressreduktion können bei wachen, intensivpflichtigen PatientInnen, die sich mittels Mimik, Gestik oder Sprache mitteilen können, identifiziert werden? Die Arbeit wurde als hermeneutische interpretative Literaturarbeit verfasst. Die Literaturrecherche erfolgte in PubMed, Cochrane, Google Scholar, der Landesbibliothek Linz und der Universitätsbibliothek der Donau-Universität Krems mit den Suchbegriffen: Stress, Stressreaktionen, Stressmanagement, Patient, Pflege und Intensivpflege. Sie sind über Operatoren wie „und", „oder", „nicht" in verschiedenen Konstellationen kombiniert worden. Der Fokus wurde auf mögliche pflegerische Interventionen und Maßnahmen gelegt und diese anhand von Studien und adäquater Fachliteratur im Zuge des hermeneutischen Zirkels interpretiert.

Resultate: Aus vorhandener Studienlage konnten nur zwei Pflegemaßnahmen mit hoher Evidenz identifiziert werden: Das Intensivtagebuch ermöglicht den Betroffenen, nach ihrem Aufenthalt auf einer Intensivstation, einer posttraumatischen Belastungsstörung durch das Lesen des Tagebuches und Rekonstruieren des Aufenthalts entgegenzuwirken. Die Initialberührung des Konzepts der Basalen Stimulation® kann den IntensivpatientInnen während des Intensivaufenthaltes zugutekommen. Die Berührung einer vorbestimmten Körperstelle signalisiert den Anfang bzw. das Ende einer Pflegetätigkeit. PatientInnen ist es dadurch möglich, sich auf eine kommende Tätigkeit einzustellen, wodurch eine Stressreduktion bewirkt wird. Andere Maßnahmen, wie z. B. Reduzierung von Lärm, das Licht in der Nacht auszuschalten, Besuchszeiten der Angehörigen zu verlängern, waren hauptsächlich auf „Expertenebene" als Empfehlungen identifiziert worden.

Schlussfolgerungen: Neben den zwei evidenzbasierten Maßnahmen konnten zahlreiche Maßnahmen zur Stressreduktion auf Intensivstationen, die auf Expertenmeinungen/-erfahrungen basieren identifiziert werden. Sie zeigen ebenso Erfolge und haben somit ihre Berechtigung. Für die Zukunft braucht es noch weitere Forschung, um deren Wirkung auch wissenschaftlich zu beweisen!

POSTERSITZUNG 18 – RHYTHMOLOGIE 3

PS 18/18-1

Procedural efficiency of high power pulmonary vein isolation compared to standard power ablation

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Introduction: Radiofrequency ablation of atrial fibrillation aims to create durable pulmonary vein isolation (PVI) in an effective and safe manner. Over the years, the advent of the 3D electroanatomic mapping systems and the implementation of the CLOSE protocol have greatly improved the success rate of PVI. High power short duration ablation is purposed to further advance this procedure's efficacy and efficiency. We report on our observation over a 3-year period, assessing the impact of different power output settings, normal power versus high power, in atrial fibrillation ablation on PVI procedural efficiency.

Methods: A total of ninety-four patients undergoing atrial fibrillation ablation at Ordensklinikum Linz Elisabethinen from January 2017 to February 2020 were included. Atrial fibrillation ablation was performed by wide antral circumferential ablation with or without additional ablation at the discretion of the operators according to the findings from the electrophysiology study and left atrial mapping. Ablation was performed in the first group with a mean power output of 33 W to a maximal power output of 40 W using the SmartTouch TCool Nav ablation catheter in 48 patients. Ablation was performed in the second group with a mean power output of 45 W to a maximum power output of 52 W using the Smart Touch TCool Nav ablation catheter or
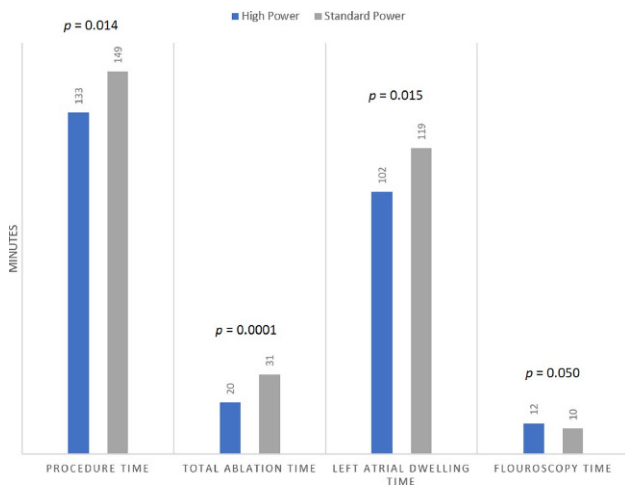
Fig. 1 | PS 18/18-1 Statistical Differences in Intraoperative Time in High Power and Standard Power Group

	High Power Group (Total 58 patients)	Standard Power Group (Total 53 Patients)	P-value
Average Age	60.1	58.8	0.46
Average Body Mass Index (BMI)	29.9	27.9	0.04
Average CHA₂DS₂VASc Score	1.79	1.53	0.28
Male Gender	40	32	0.35
Hypertension	36	23	0.05
Diabetes	4	2	0.48
Coronary Heart Disease	11	6	0.27
Congestive Heart Disease	12	5	0.10

Fig. 2 | PS 18/18-1 Cohort Demographics and Comorbidities

the QDOT Micro Thermocool SmartTouch SF ablation catheter (Biosense Webster, Inc. Irvine, California) in 46 patients.

Results: Baseline characteristics and medical history are summarized in Table 1. There was no significant difference between the 2 groups for the clinical characteristics except for body mass index (BMI). Regarding the procedural parameters for the high power and the standard power groups, the procedure duration appears to be better in the high power group with a mean duration of 133 min and a standard deviation of 35 vs. a mean duration of 149 min with a standard deviation of 32, respectively, $p = 0.014$. Left atrial dwell time was shorter in the high power group with a mean time of 102 min a standard deviation of 35 vs. a mean time of 119 min with a standard deviation of 32; $p = 0.014$. The total ablation time was significantly less in the high power group as compared with the standard power group with a mean ablation time of 20 min with a standard deviation of 35 vs. a mean ablation time of 119 min with a standard deviation of 32; $p = 0.0001$. The fluoroscopy duration was slightly less in the standard power group with a mean duration of 10 min and a standard deviation of 5 vs. a mean duration of 12 with a standard deviation of 6; $p = 0.05$; Fig. 1.

Conclusion: The use of higher power shows a trend towards improved procedural efficiency.

PS 18/18-2

Successful ablation of bigeminal PVCs triggering polymorphic VT in LQT 2 syndrome

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Introduction: A 19-year-old woman with an established diagnosis of Long QT (LQT) 2 syndrome and underlying KCNH2-mutation was referred to our centre for recurrent ventricular tachycardias/fibrillation (VT/VF) refractory to medical treatment. A subcutaneous ICD (Boston Scientific) had been implanted in 2015. Despite being on drug therapy with mexiletine and beta-blocker, she presented with repeated episodes
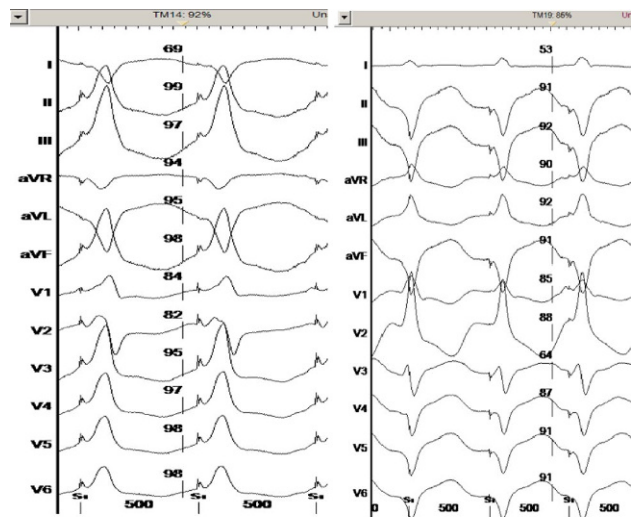
Fig. 1 | PS 18/18-2 Pace mapping used to narrow down the region of interest revealed best pacematch of 92 % for the anterior LV fascicle morphology and 85 % for the posterior LV fascicle
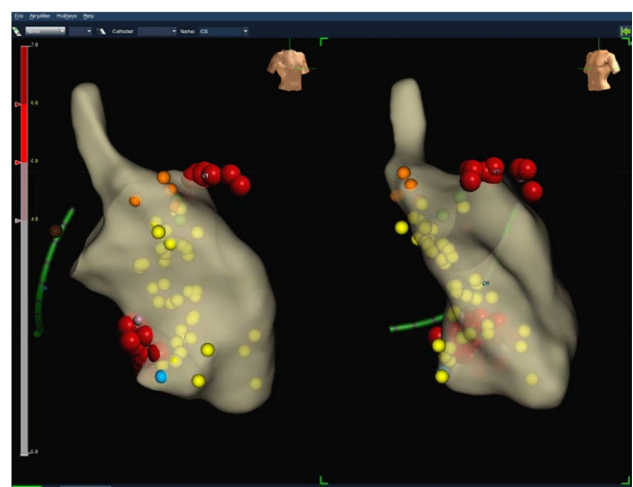
Fig. 2 | PS 18/18-2 3D electroanatomic map (using the EnSite Precision, Abbott mapping system) shoeing RAO and LAO projections and radiofrequency ablation points applied at the posterior and anterior LV fascicles

of premature ventricular complexes (PVC) triggering sustained and non-sustained polymorphic VT/VF which were successfully terminated by appropriate shocks. Interrogation of the ICD revealed frequent episodes of VT/VF initiated by ventricular bigeminy. Holter monitoring demonstrated a PVC burden of 3657 (4 %) per 24 h. The PVCs were of two different morphologies. The first PVC had a right bundle branch morphology with inferior axis suggesting an origin from the left ventricular anterior fascicle. The second PVC had a right bundle branch morphology with a frontal superior axis suggesting an origin from the left ventricular posterior fascicle. A decision was made to perform an invasive electrophysiologic study to map and ablate the PVCs.

Methods: Using a 3D electro-anatomical mapping system (EnSite Precision, Abbott) the left ventricle was mapped and Purkinje potentials of both fascicles were marked. Pace mapping was applied to further narrow down the region of interest. With the best pace match of 92 % obtained at the anterorior fascicle and 85 % obtained at the posterior fascicle (Fig. 1) the earliest activation was identified to be at both left ventricular fascicles and at least 38 ms ahead of the PVCs. Using both the retrograde aortic and the transseptal approach radiofrequency energy was applied at 45 to 50 W at the anterior and posterior fascicular regions targeting areas with identifiable Purkinje potentials. Ablation at these sites produced runs of idioventricular rhythm with QRS morphology similar to the PVCs that initiated VT/VF. Radiofrequency ablation resulted in acute suppression of both PVCs.

Results: Over a follow up period of 6 months after the procedure the patient has fared well with a marked reduction of PVC burden from 3657 in 24 h (4 %) to 66 PVCs in 24 h (0.08 %) and no documented episodes of ventricular arrhythmias.

Conclusion: Ablation of triggering PVCs causing recurrent VT/VF in LQT 2 syndrome is feasible and effective over a short term follow up of 6 months.

PS 18/18-3

Evaluation of pulmonary vein lesions after cryoballoon ablation in redo procedures for recurrent atrial fibrillation using a high resolution mapping system

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Introduction: Aims Pulmonary vein isolation (PVI) is the cornerstone of atrial fibrillation (AF) ablation. Due to recurrence rates of 10–60 %, redo procedures are common. There is, however, no clear consensus on how a repeat AF ablation should be carried out. Cryoballoon ablation is widely used for first time PVIs and is appreciated for short procedure time and reproducible results. This technology is being increasingly used in redo
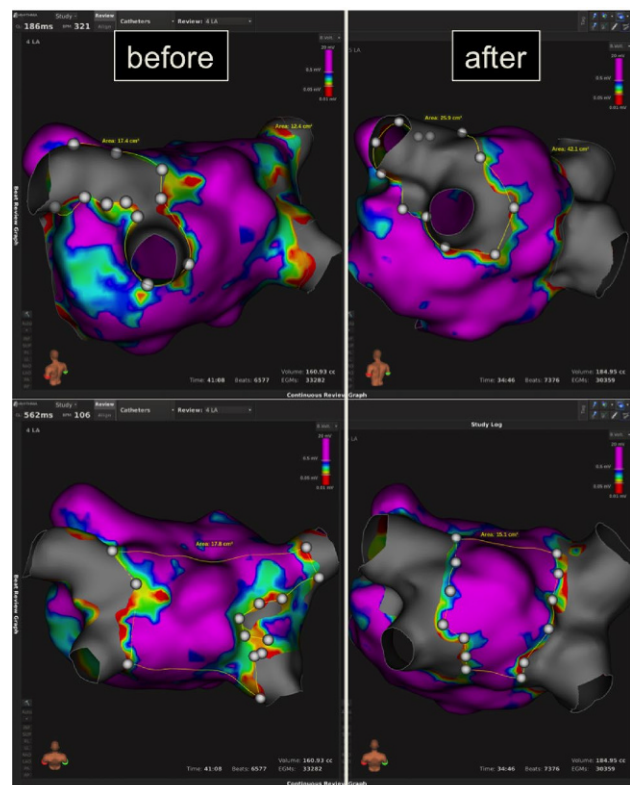
Abb. 1 | PS 18/18-3 Electroanatomic map illustrating the lesions produced by cryoballoon ablation and their measurement

procedures for AF recurrence after PVI, although data for use in this setting is scarce. Therefore, it is crucial to understand how lesion formation behaves in redo procedures carried out with cryoballoon and whether this approach can lead to successful isolation of pulmonary veins (PV) in this group of patients.

Methods: Methods In this case series we retrospectively evaluated seven consecutive patients undergoing redo ablation procedure for AF recurrence after a primary PVI with radiofrequency ablation. In all patients an initial high-resolution electroanatomical voltage map was created and gaps were localized. Subsequently, an empiric cryoballoon PVI of all the PVs was performed using cryoballoon ablation. Finally, a remap was done to assess lesion size and effectiveness of PVI.

Results: Results Of the included patients, 4 were female and 3 male. Mean age was 56 years and clinical characteristics were similar in all patients. A mean of 4 PVs were present in each patient, of which an average of 3.3 veins per patient exhibited at least one gap at the beginning of the procedure. Total lesion size increased significantly after cryoablation by 31.51 cm^2 (± 8.97 cm^2 [CI 23.21–39.81] $p < 0.001$), while the remaining viable tissue on the posterior wall center area decreased significantly by 6.9 cm2 (± 5.79 cm2 [CI 1.55–12.26] $p = 0.02$).

Table 1 | PS 18/18-3 Comparison of pulmonary vein lesion size and posterior wall center area, before and after cryoablation

	Before cryo (cm^2)	After cryo (cm^2)	Mean difference (cm^2)	P
Total PV lesion size	29.89 ± 12.28	61.40 ± 10.40	31.51 ± 8.97	<0.001
Left PV lesion size	14.13 ± 4.23	25.10 ± 5.57	10.97 ± 3.02	<0.001
Right PV lesion size	15.76 ± 9.18	36.30 ± 6.63	20.54 ± 9.79	0.001
Posterior wall center area	22.52 ± 6.48	15.61 ± 3.12	6.90 ± 5.79	0.02

Conclusion: Conclusions This hypothesis generating case series suggests that empiric cryoballoon ablation in redo PVI procedures is a reasonable approach. We demonstrated that the cryoballoon creates large antral wide area circumferential ablation lesions and thereby expands lesion size from previous PVIs.

PS 18/18-4

Atrial fibrillation triggered in the superior vena cava

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Introduction: Pulmonary vein isolation (PVI) is the mainstay of catheter-based atrial fibrillation (AF) ablation. However, recurrence rates remain substantial (at around 30 %). While in most instances pulmonary vein (PV) reconnection may be the cause of recurrence, it has been described that AF triggers outside of the pulmonary veins (PV) exist. An important location of AF triggering foci is the superior vena cava (SVC).

Methods: n. a.**:**

Results: Case reports: We report two cases of AF recurrence after multiple PVI procedures, in which the SVC could be identified to act as trigger for AF. The first case was a 56-year-old male patient who underwent cryoballoon PVI one year earlier and came back for a redo procedure because of recurrence of paroxysmal AF. Despite successful reisolation of the PVs, the patient went spontaneously into AF. A stepwise trigger mapping was performed and the responsible atrial premature beats (APB) could be localized to the SVC. Hence, the SVC was isolated by radiofrequency ablation (RFA) and stable sinus rhythm was achieved. The second case involved a 49-year-old woman. After four PVI procedures and a dual-chamber pacemaker implantation, she was scheduled for a fifth electrophysiologic procedure for recurrent symptomatic AF episodes, documented in the pacemaker. Recurrent bursts of APBs were observed intraprocedurally. Similarly to the previous case, they could be mapped to the SVC. Again, isolation of the SVC was performed, which led to succesful suppression of the APBs and all mode-switch episodes in the pacemaker.

Conclusion: Arrhythmogenic foci in the SVC can trigger atrial tachycardias and atrial fibrillation. In AF redo ablation procedures, isolation of the SVC should be considered if atrial arrhythmias recur despite successful PVI.
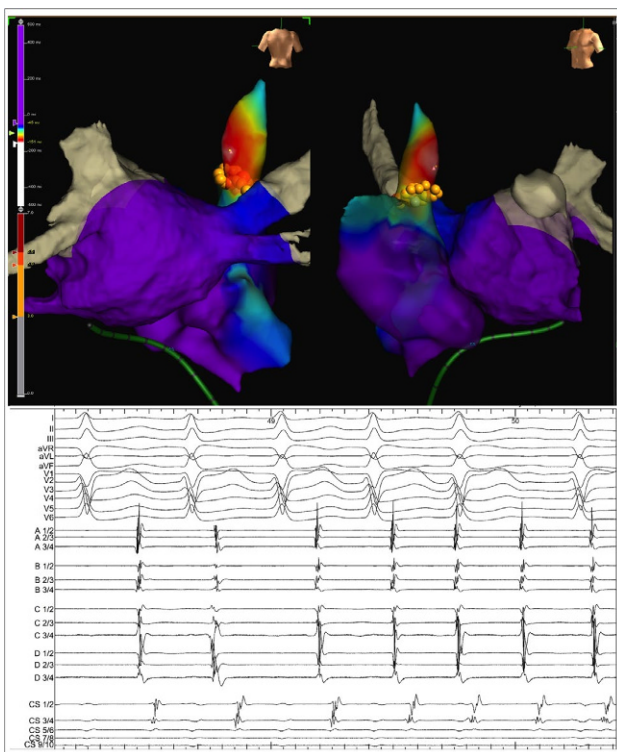
Fig. 1 | PS 18/18-4 Isolation of the superior vena cava (SVC) is illustrated in the electroanatomical map. The intracardiac electrogram shows repetitive bursts of atrial premature beats which were earliest in the SVC
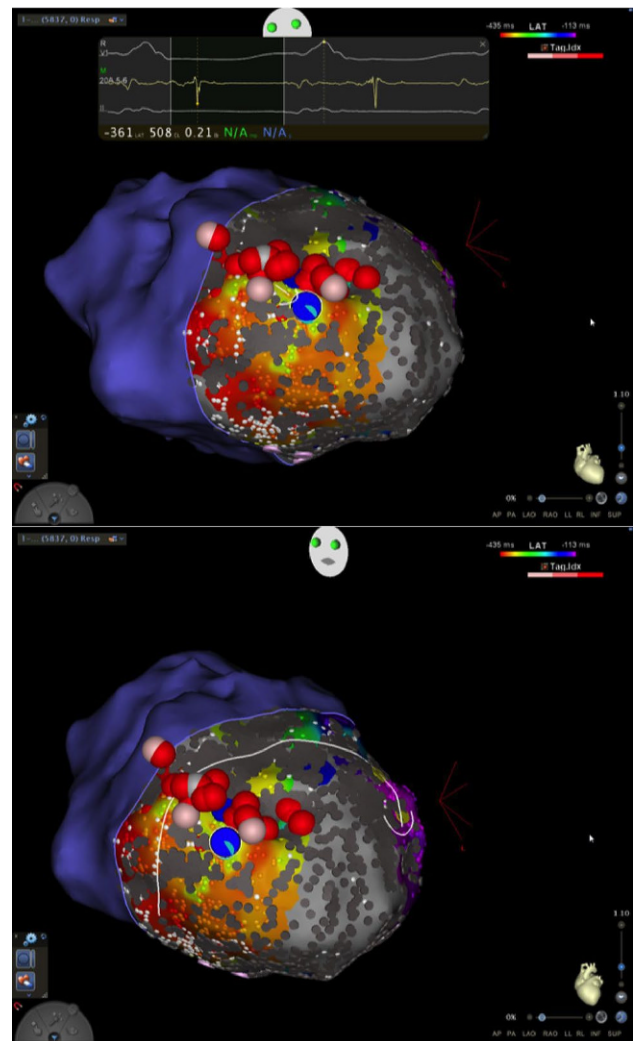
Abb. 1 | PS 18/18-5 Darstellung des diastolischen Pfades/kritischen Isthmus mittels Aktivierungsmap der ventrikulären Reentry-Tachykardie im apikalen Narbenareal in LAO Projektion (nur die diastolische Aktivität wird angezeigt, die systolische Aktivität ist ausgeblendet) und der durchgeführten Ablation (rote Punkte)

PS 18/18-5

Ablation einer inzessanten ventrikulären Tachykardie durch Mapping des diastolischen Pfades unter ECMO-Unterstützung

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Einleitung: Myokardiale Narben bilden das organische Substrat für ventrikuläre Tachykardien (VT) bei Patienten mit ischämischer Kardiomyopathie. Anatomisch findet sich in der Narbe ein Isthmus, eine für die VT Kreiserregung kritische Zone mit langsamer elektrischer Leitung, den es durch die VT-Ablation zu eliminieren gilt.

Methoden: n. a.:

Resultate: Fallbericht: Wir berichten über einen Patienten mit ischämischer Kardiomyopathie und Vorderwandaneurysma im elektrischen Sturm aufgrund einer inzessanten ventrikulären Tachykardie, die auch durch zwei endokardiale Ablationen, eine epikardiale Ablation und eine Sympathektomie therapeutisch nicht beherrscht werden konnte. Als letzter Ausweg wurde in der laufenden VT direkt der diastolische Pfad identifiziert und erfolgreich von endokardial abliert. Weil die VT hämodynamisch nicht toleriert wurde, wurde diese dritte Intervention unter extrakorporaler Membranoxygenierung (ECMO) durchgeführt. Es waren insgesamt drei Ablationszentren an diesen Prozeduren beteiligt. Durch die gut koordinierte Zusammenarbeit konnte der Patient trotz mehrfach therapierefraktären elektrischen Sturms letztendlich erfolgreich behandelt werden

Schlussfolgerungen: Direktes Mapping des diastolischen Pfades und Ablation des kritischen Isthmus bei laufender VT unter ECMO Unterstützung ist eine wirksame Therapieoption bei inzessanten Kammertachykardien. Für die optimale Versorgung von Patienten im elektrischen Sturm ist eine koordinierte, überregionale interhospitale Zusammenarbeit im Rahmen eines VT Netzwerkes zwingend erforderlich.

PS 18/18-6

Left atrial anterior wall microreentrant tachycardia involving an idiopathic fibrosis area: a case report

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Introduction: Left atrial tachycardias most commonly involve macroreentrant circuits around the mitral valve or the
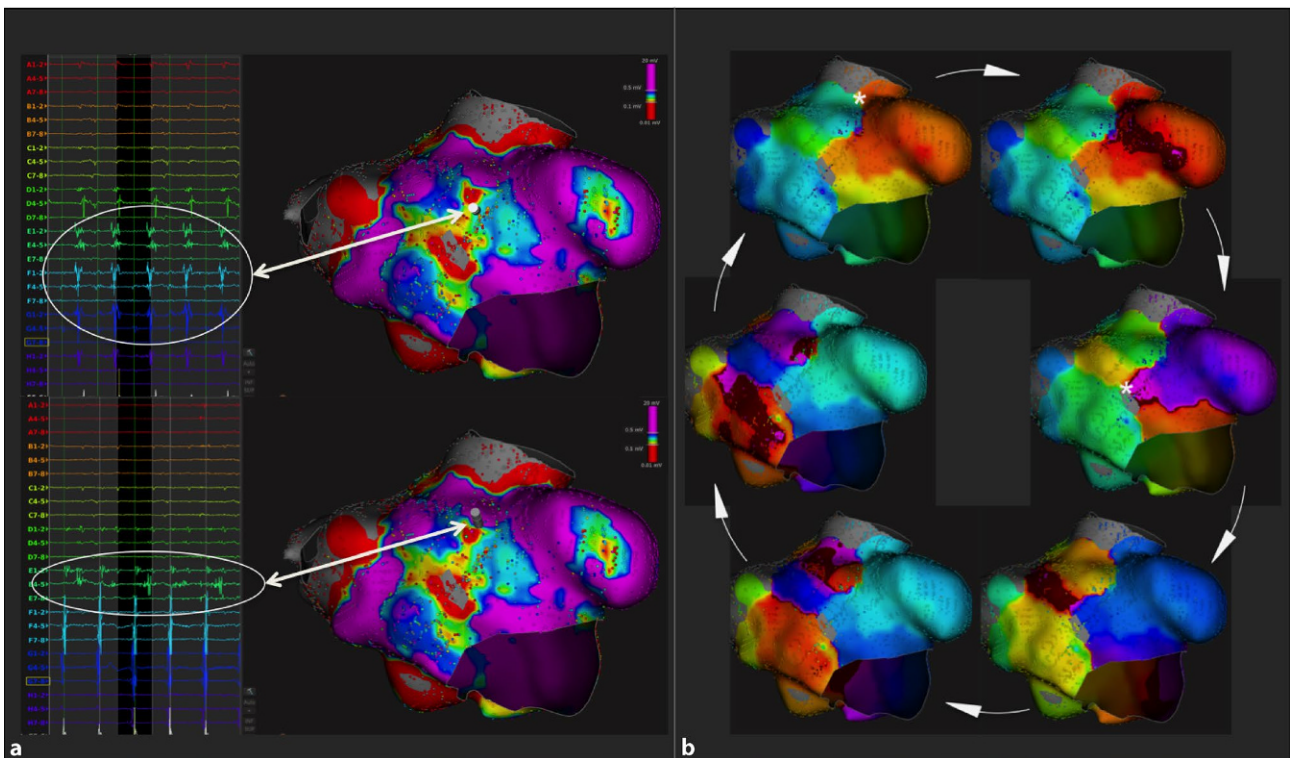
Abb. 1 | PS 18/18-6 Local electrograms in the area of the two slow conduction zones are shown in (**a**). The roving probe (grey catheter tip) indicates the location where the electrogram has been recorded. As can be noticed, local activation is extremely fractionated and almost continuous, spanning a large part of the cycle length. In (**b**) the propagation of the microre-entry arrhythmia is illustrated, showing the circuit revolving around a central point (obstacle). Two areas of significant delay in propagation can be appreciated (marked by white stars), corresponding to the areas of electrogram fractionation

left atrial roof, and sometimes a focal source. Microreentrant tachycardias, on the contrary, are a rare finding, especially in previously unablated patients.

Methods: n. a.:

Results: Case summary: We present a case of an 83-year-old female patient with persistent atypical atrial flutter alongside episodes of atrial fibrillation. She did not undergo any prior ablation procedures. The electroanatomic voltage map revealed an area of fibrosis (low voltage <0.5 mV) at the anterior left atrial wall. The activation map showed a microreentrant circuit revolving around a small area of dense scar (0.5 ×0.5 cm), within the low-voltage area. After identification of the arrhythmia mechanism, successful ablation could be performed.

Conclusion: Microreentry as a mechanism of atrial flutter is rare. Atrial myopathy, leading to the development of scarring and fibrosis, is linked to atrial arrhythmias. In the present case, atrial anterior wall fibrosis facilitated a microreentrant circuit by providing a central obstacle and two zones of slow conduction. High-resolution mapping allowed us to successfully map and ablate the arrhythmia.

PS 18/18-7

Long-term outcome after ablation of atrial fibrillation in patients with reduced ejection fraction

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Introduction: Pulmonary vein isolation (PVI) is an established therapy for symptomatic patients with paroxysmal or persistent atrial fibrillation (AF). The randomized controlled CASTLE AF trial showed that catheter ablation for AF in patients with heart failure is associated with a significantly lower rate of death from any cause or hospitalization for worsening heart failure than optimal medical therapy only. Patients with reduced left ventricular ejection fraction (LVEF) may have a poorer outcome after ablation compared to patients with preserved LVEF. In this study, we aimed to describe long-term outcome of patients with reduced ejection fraction.

Methods: We performed a retrospective analysis of 274 consecutive patients included in the "Graz AF ablation registry" who underwent AF ablation between 2015 und 2018. We grouped patients by LVEF (LVEF ≥55 % grouped as normal versus <55 % as reduced LVEF) at the time of the first ablation procedure and performed group-wise comparison.

Results: 243 patients had normal left ventricular systolic function (nEF, 64 ±5 %) and 31 patients had reduced systolic function (rEF, 49 ±10 %). In rEF patients, atrial enlargement was more frequent (48 vs. 25 %, $p=0.005$). Age (56 ±12 vs. 60 ±12), percentage of females (26 vs. 19 %), BMI (27 ±5 vs. 29 ±4 kg/m^2, $p=0.08$), CHA2DS2-Vasc-Score (1 [0, 2] vs. 1 [0, 2], $p=0.8$), HAS-BLED Score (1 [0, 2] vs. 1 [1, 2], $p=0.56$), arterial hypertension (54 vs. 52 %, $p=0.15$), diabetes (4 vs. 7 %, $p=0.26$), prior stroke (4 vs. 3 %, $p=0.38$) were comparable between both groups. PVI was performed in 100 % of nEF and rEF patients, roof lines in 3 vs. 3 % ($p=0.4$), cavotricuspid isthmus ablation in 21 vs. 20 % ($p=0.19$). Follow up duration was 340 (122, 555) vs. 192 (107, 452) days ($p=0.12$). Complication rate was low in both groups (inguinal aneurysm in 1 nEF patient, 0.4 %), 4 vs.

7 % of patients requiring cardioversion before demission from the ward ($p=0.25$). Repeat ablations were performed in 19 vs. 23 % of patients ($p=0.17$). Single procedure success rate was comparable in both groups: 59 % in nEF patients vs. 67 % in rEF patients (log rank test: $p=0.203$). At the end of follow up, 58 % of nEF patients vs. 65 % of rEF patients were still on antiarrhythmic drug therapy ($p=0.12$).

Conclusion: Catheter ablation for symptomatic AF is reasonable and safe in patients with reduced left ventricular ejection fraction. Outcomes and complication rates are comparable, independent of the patients' LVEF.

PS 18/18-8

Concordance of the Cockcroft-Gault, MDRD and CKD-EPI formulas in patients receiving Non-Vitamin K Antagonist Oral Anticoagulants: Insights from the PREvention oF thromboemolic events– European Registry in Atrial Fibrillation (PREFER in AF Prolongation)

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Introduction: The Cockcroft Gault (CG) formula is recommended to determine a potential renal indication for dose
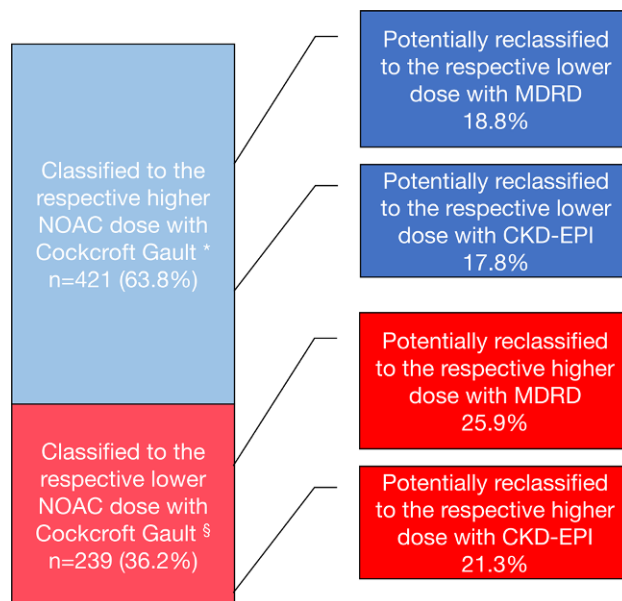
Fig. 1 | PS 18/18-8 Proportion of patients requiring a different dose of edoxaban, rivaroxaban and potentially dabigatran (either lower or higher) when the MDRD or the CKD-EPI formulas are applied instead of CG

reduction of dabigatran, edoxaban and rivaroxaban. Most laboratories adopted the more accurate Modification of Diet in Renal Disease (MDRD) and Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formulas.

Methods: The prospective, observational, multicentre PREvention oF thromboemolic events–European Registry in Atrial Fibrillation (PREFER in AF Prolongation) study included patients with AF receiving a non-Vitamin K antagonist oral anticoagulant (NOAC) for stroke prevention. We investigated the proportion of patients with dissimilar renal dosing indications when applying the CG, MDRD or CKD-EPI formula.

Results: The study included 660 AF patients with chronic kidney disease. The MDRD and CKD EPI formulas provided lower estimated glomerular filtration rates (eGFRs) in patients with mild renal dysfunction, and higher eGFRs in patients with moderate-severe renal dysfunction (e. g. KDIGO class 4, mean difference 5.8 ml/min for MDRD vs. CG, $p < 0.01$ and 4.5 ml/min for CKD-EPI vs. CG, $p < 0.01$). Differences were particularly pronounced in patients with a body weight ≤60 kg and moderate-severe renal dysfunction (mean difference 15.3 ±6.8 ml/min for MDRD vs. CG, $p < 0.01$ and 14.2 ±6.1 ml/min for CKD-EPI vs. CG, $p < 0.01$). In patients in whom the CG formula suggested a potential renal indication for dose reduction of dabigatran, edoxaban or rivaroxaban (eGFR ≤50 ml/min), 26 % and 21 % were reclassified to the respective higher doses when applying the MDRD and CKD-EPIs formula. Conversely, 19 % and 18 % were potentially reclassified to the respective lower NOAC doses when using MDRD and CKD-EPI.

Conclusion: The use of more accurate formulas for estimating creatinine clearance resulted in a potentially different dosing indication in up to a quarter of patients. The differences in eGFR were particularly pronounced in patients susceptible to bleeding complications, i. e. those with moderate-severe renal dysfunction and low body weight.

POSTERSITZUNG 19 – DIVERSE 2

PS 19/19-1

Inflammation is associated with elevated levels of ZMPSTE24 and lamin A/C

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Introduction: Lamins are important intermediate filament proteins forming the inner nuclear membrane thereby regulating its prober function. The biosyntheses of lamin A, especially the last cleavage step, is performed by the zinc metalloproteinase (ZMPSTE24). Failure to cleave defective prelamin A also-called progerin–causes Hutchinson Gilford Progeria Syndrome (HGPS) a well known premature ageing diseases. Minor levels of progerin are also expressed in otherwise healthy non-HGPS cells through alternative splicing. Recently we could observe increased expression of progerin mRNA in patients with dilated cardiomyopathy associated with impaired left ventricular function and found an association with overweight and chronic inflammation. In this study we aimed to elucidate possible correlations of ZMPSTE24 and mRNA expression levels of lamin A and.

Methods: In this retrospective, cross-sectional analysis of prospectively included patients, quantitative expression of lamin A, ZMPSTE24 and progerin mRNA were determined in

blood samples of 110 individuals. Quantitative RT-PCR analyses were performed using primers spanning exon 9 to 12 do determined total LMNA expression and spanning the splice junction site between exon 11 and 12 for progerin quantification as published by us previously (9). Exon spanning primers were verified on agarose gels. Rpl32 was used as reference gene.

Results: Linear regression analyses based on Spearman's correlation ($n = 110$) were performed to calculate whether CRP-serum levels were correlated to total lamin or ZMPSTE mRNA. As shown in figure 1A total lamin mRNA expression is positively related to serum-CRP ($n = 110$; r = 0.24; $p = 0.03$). Furthermore a significant positive correlation was observed between ZMPSTE and CRP ($n = 110$; r = 0.21; $p = 0.01$). We further looked for an association of ZMPSTE, total lamin and progerin mRNA expression. ZMPSTE ($n = 110$; r = 0.33; $p = 0.0004$) correlates significantly with progerin mRNA levels and are positively related to total lamin ($n = 110$; r = 0.82, $p < 0.0001$) expression. No associated is observed between total lamin A and ZMPSTE.

Conclusion: To elucidate possible underlying mechanisms we measured expression levels of total LMNA and ZMPSTE24. The enzyme ZMPSTE24 is known to be involved in different steps of lamin A processing and its proper functioning is crucial for the production of a mature protein. In our cohort we found an association between the inflammation biomarker CRP and the expression of progerin mRNA as well as for ZMPSTE24 mRNA. We previously observed an association between progerin expression, overweight and inflammation. This study suggest that chronic inflammation is associated with increased expression of total lamin and ZMPSTE mRNA. Both correlate with progerin mRNA expression and thus is linked to premature biologic aging.

PS 19/19-2

High-sensitivity cardiac troponin after strenuous exercise–a systematic review and meta-analysis

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Introduction: Rising patterns of high-sensitivity cardiac troponin (hscTn) do not only occur during acute coronary syndrome, but also following strenuous physical activity. To improve our understanding about hscTn release associated with strenuous exercise, we conducted a systematic review of the existing literature and performed a meta-analysis.

Methods: Two independent reviewers searched the literature for studies that reported hscTn change values (hscTnT or hscTnI) in relation to physical activity that were published between Jan 2008 and July 2016. Studies were excluded if they included patients with symptoms of myocardial ischemia prior to measurement of baseline hscTn value.

Results: This meta-analysis included 27 studies (total $n = 1177$) that reported hscTn before and after strenuous exercise. After strenuous exercise, hscTnT increased by 15.2 ng/L or 338 % (19 studies, $n = 688$) (Fig. 1) and hscTnI by 23.2 ng/L or 340 % (11 studies, $n = 519$) (Fig. 2).

Conclusion: Results from this meta-analysis suggest that hscTn can markedly rise after strenuous physical activity.

Study name	Difference in means	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Aagaard et al.; 2014	35.8	4.9	23.9	26.2	45.4	7.3	0.0
Baker et al.; 2014 (NSHD Group)	44.0	6.0	35.6	32.3	55.7	7.4	0.0
Boos et al.; 2014	2.9	1.3	1.8	0.3	5.5	2.2	0.0
Duttaroy et al.; 2012	4.8	1.9	3.6	1.1	8.5	2.5	0.0
Hattasch et al.; 2014	10.8	2.0	4.0	6.9	14.7	5.4	0.0
Huskens et al.; 2015	12.4	1.0	1.0	10.5	14.3	12.7	0.0
Klapcinska et al.; 2013	10.0	4.4	19.1	1.4	18.6	2.3	0.0
Legaz-Arrese et al.; 2015 Cycling	12.4	2.0	4.0	8.5	16.4	6.2	0.0
Legaz-Arrese et al.; 2015 Running	15.0	2.4	5.8	10.2	19.7	6.2	0.0
Legaz-Arrese et al.; 2015 Swimming	14.7	2.5	6.5	9.7	19.7	5.8	0.0
Legaz-Arrese et al.; 2015 Treadmill Run	12.4	12.5	157.2	-12.2	36.9	1.0	0.3
Lippi et al.; 2010	0.7	0.1	0.0	0.4	1.0	5.0	0.0
Mellor et al.; 2014	4.4	1.2	1.6	2.0	6.8	3.5	0.0
Mingels et al.; 2009	38.0	4.9	24.5	28.3	47.7	7.7	0.0
Sanchis-Gomar et al.; 2015 (Placebo Group)	8.4	1.8	3.1	4.9	11.9	4.7	0.0
Scherr et al.; 2011	27.8	0.7	0.4	26.5	29.1	41.8	0.0
Stewart et al.; 2014	22.2	6.7	45.4	9.0	35.4	3.3	0.0
Stewart et al.; 2015	14.7	6.7	45.5	1.5	27.9	2.2	0.0
Tian et al.; 2012	111.1	53.8	2899.6	5.5	216.6	2.1	0.0
Tian et al.; 2014	54.9	35.2	1241.8	-14.2	123.9	1.6	0.1
Wedin et al.; 2015	2.8	0.6	0.4	1.6	4.0	4.6	0.0
	15.2	2.7	7.1	10.0	20.5	5.7	0.0

Heterogeneity: I2 = 99%

Fig. 1 | PS 19/19-2 Forest plot showing pooled estimate of the absolute hs-cTnT change from baseline after strenuous exercise

Study name	Difference in means	Standard error	Variance	Lower limit	Upper limit	Z-Value	p-Value
Eijsvogels et al.; 2012	14.0	4.5	20.5	5.1	22.9	3.1	0.0
Eijsvogels et al.; 2015	80.0	10.9	119.7	58.6	101.4	7.3	0.0
Lippi et al.; 2010 Downhill walking	0.2	0.4	0.1	-0.5	0.9	0.5	0.6
Lippi et al.; 2012	14.0	1.0	1.1	12.0	16.0	13.4	0.0
Lippi et al.; 2012 JCLI	9.4	2.7	7.5	4.0	14.8	3.4	0.0
Mingels et al.; 2009	50.0	9.4	88.6	31.5	68.5	5.3	0.0
Mohlenkamp et al.; 2014	20.0	5.1	25.8	10.0	30.0	3.9	0.0
Om et al.; 2014	86.8	11.5	131.3	64.3	109.3	7.6	0.0
Salvagno et al.; 2014	8.8	3.1	9.5	2.8	14.8	2.9	0.0
Stewart et al.; 2016	20.9	7.5	56.8	6.1	35.7	2.8	0.0
Stewart et al.; 2015	13.8	6.0	35.4	2.1	25.5	2.3	0.0
	23.2	3.9	15.6	15.5	31.0	5.9	0.0

Heterogeneity: I2 = 96.9%

Fig. 2 | PS 19/19-2 Forest plot showing pooled estimate of the absolute hs-cTnI change from baseline after strenuous exercise

PS 19/19-3

Outcome after in- and out-of-hospital cardiac arrest treated at the University Hospital Krems: an observational cohort analysis

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Introduction: At the moment, there are no data available on incidence and outcome of patients suffering from in- and out- of hospital cardiac arrest (IHCA, OHCA), treated at the Department of Internal Medicine I. This study aims to determine outcome data on this patient cohort.

Methods: In the setting of an academic secondary care center, all patients treated after IHCA or OHCA between July 1st and December 31st, 2019 were included. We observed all patients until death or hospital discharge. Main outcome was defined as survival and neurological outcome at the time of discharge. Neurological outcome was evaluated with the Cerebral Performance Categories (CPC). All data are given in totals and percentage or mean and standard deviation if applicable.

Results: During the observational period, an overall number of 31 adult cardiac arrest patients were treated. Of these,

Table 1 | PS 19/19-4 Patients characteristics

No Chest compression ($n=196$)			Chest compression ($n=270$)	
Sex-female	99 (51 %)		Sex-female	93 (34 %)
Died within 24 h	22 (11 %)		Died within 24 h	146 (54 %)
Regular duty	149 (77 %)		Regular duty	187 (69 %)
Weekend	45 (23 %)		Weekend	82 (31 %)
Night duty	113 (58 %)		Night duty	169 (63 %)
Initial Rhythm ($n=190$)		($n=265$)		
Sinus rhythm	151 (79 %)		Sinus rhythm	8 (3 %)
Bradycardia	12 (6 %)		Bradycardia	3 (1 %)
Ventricular fibrillation	7 (4 %)		Ventricular fibrillation	12 (5 %)
Atrial fibrillation	8 (4 %)		Atrial fibrillation	4 (2 %)
VF	3 (2 %)		VF	28 (11 %)
PEA	1 (1 %)		PEA	107 (44 %)
Asystole	2 (1 %)		Asystole	94 (36 %)
Co-morbidities ($n=195$)		($n=270$)		
CVD	146 (75 %)		CVD	230 (85 %)
Hypertension	140 (72 %)		Hypertension	206 (76 %)
Dyslipidemia	101 (52 %)		Dyslipidemia	137 (51 %)
AFIB	60 (31 %)		AFIB	95 (35 %)
T2 DM	57 (29 %)		T2 DM	101 (39 %)
CKD	55 (28 %)		CKD	110 (42 %)
Cancer	44 (21 %)		Cancer	70 (26 %)
COPD	39 (20 %)		COPD	59 (23 %)
Aortic stenosis	21 (11 %)		Aortic stenosis	42 (16 %)
CTEPH	9 (5 %)		CTEPH	33 (12 %)

26 (84 %) patients were male. The mean age was 68 ± 14 years. OHCA occured in 18 (58 %) patients compared to 13 (42 %) patients suffering from IHCA. In 20 patients (64 %) a cardiac cause has been identified. In 20 (64 %) patients a sustained return of spontaneous circulation has been restored. In total 9 (29 %) patients (5 (56 %) IHCA, 4 (44 %) OHCA) were discharged alive with a favorable neurological outcome rated with CPC Score of 1.

Conclusion: In a short observational period of 6 months, a relatively large proportion of patients (29 %) survived with favorable neurological outcome. Further studies are needed to confirm these results and investigate the strengths in the local chain of survival.

PS 19/19-4

Patient's outcome after inhospital emergency call in a tertiary hospital in Austria from 2014 to 2018

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Introduction: In this study, we retrospectively followed all calls to our medical emergency team (MET) and to assessed survival among patients who received a cardiopulmonary resuscitation (CPR) in a tertiary Univiersity hospital. Our MET is one of 3 teams and our responsibility includes 15 departments, cath lab and some out-patient clinics of the General Hospital of Vienna.

Methods: We reviewed demographic data and diagnosis from 2014 until 2018. regression model was used to test the association of clinical characteristics with the primary outcome of survival. Data are mean±standard deviation or median an IQR, as appropriate.

Results: A total of 455 patients were available for analysis (97 [49 %] male, 99[51 %] female mean age 65 ±16 years). One hundred an ninety six of 455 pts (43.1 %) had no chest compressions. One hundred and three (53 %) patients died during the observation period. Out of 270 patients who received chest compression 177 (66 %) were male and 93 (34 %) female with a mean age of 69 ± 14 yrs. 229 patients (85 %) died during the observation period. The cumulative survival of patients with or without chest compression is presented in Fig. 1. After adjustment for covariates, age ($p=0.001$, 95 %CI, 0.95–0.99), cardiovascular disease (CVD) ($p=0.010$, 95 % CI, 0.14–0.77), chronic kidney disease (CKD) ($p=0.000$, 95 %CI, 1.73–5.11), and dyslipidemia ($p=0.059$, 95 %CI, 0.33–1.02) were significantly associated with patients's survival during the first 24 h.

Conclusion: In the present study, our MET faced a "true" CPR situation in more than 59 % of cases. The first 24 h were vital for patient's clinical outcome and survival. Beside age, comorbidities such as CVD and CKD were associated with mortality after CPR.

PS 19/19-5

Produktprobleme bei Selbsttests zur Bestimmung der INR – Analyse der 2004–2020 vom BfArM veröffentlichten Kundeninformationen

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Einleitung: Vermarktung und Marktüberwachung von Medizinprodukten und In-vitro Diagnostika (IVD) sind in Europa durch europäische Direktiven (z. B. Verordnung (EU) 2017/745 über Medizinprodukte, Verordnung (EU) 2017/746 über In-vitro-Diagnostika) geregelt. Bei Vorkommnissen und korrektiven Maßnahmen (Field Safety Corrective Action, FSCA) müssen die Hersteller diese den zuständigen nationalen Behörden (Competent Authority (CA); in Deutschland: Bundesinstitut für Arzneimittel und Medizinprodukte (BfArM) für Medizinprodukte und die meisten IVD, in Österreich: BASG) melden und die Kunden über Kundeninformationen (Field Safety Notice, FSN) informieren, die auch den Behörden zur Verfügung gestellt werden. Selbsttests (und diesen entsprechende Point of Care Tests (POCT); jeweils Analyzer und Teststreifen) zur Bestimmung des INR-Wertes (International Normalized Ratio) sind trotz neuer oraler Antikoagulantien (NOAK) von Bedeutung bei Selbstkontrolle/Optimierung der Therapie mit Vitamin-K-Antagonisten (Marcumar, Warfarin) von Patienten mit schweren Erkrankungen, z. B. implantierten Herzklappen und Herzunterstützungssystemen, Pulmonalarterienembolien nach tiefer Beinvenenthrombose, Lupus Antikoagulans und Unverträglichkeiten gegenüber NOAK. Ziel der Studie war die Untersuchung von FSN/FSCA zu diesen IVD, die seit 15.12.2004 auf der Homepage des BfArM veröffentlicht wurden, in Hinblick auf vorliegende Produktprobleme, damit einhergehende Risiken und Art der FSCA.

Methoden: Für die in die Studie eingeschlossenen IVD erfolgte eine Analyse der vom BfArM bis 2020 auf der Homepage (http://www.bfarm.de/DE/Medizinprodukte/riskinfo/kundeninfo/functions/kundeninfo-node.html) publizierten FSCA und FSN.

Resultate: Es fanden sich 21 FSCA zu INR Selbsttests (einschl. entsprechender POCT) bei insgesamt 2889 FSCA zu IVD im Untersuchungszeitraum. Diese betrafen Teststreifen (8), Analyzer (9) bzw. das Gesamtsystem (4). Berichtete Probleme waren bei Tests abweichende INR-Werte oder Fehlermeldungen (6, z. B. Stabilitäts-/Produktionsprobleme, Kalibrationsänderung) und Hinweis auf die Gebrauchsanweisung bzw. Änderung der Anwendungsbeschränkung (2), bei Analyzern Probleme der Datenübertragung/Datensicherheit (3), andere Software-/Geräteprobleme (4, Fehlermeldungen, Freigabe von Werten außerhalb des Meßbereiches, Probenfehler, eingeschränkte Performance) und fehlerhafte INR-Werte durch hohen Hämatokrit und Gerätereinigung (2) sowie bei Systemen falsch-niedrige INR-Werte bei Erkrankungen (2), verminderte Performance in einer Studie (1) und möglicher Todesfall (1). Korrektive Maßnahmen (Mehrfachnennungen) waren bei Tests/Analyzern/Systemen FSN (bei Rückrufen obligat; oft mit Handlungsanweisungen (6/7/3)), Rückruf (4/6/3), Software-Upgrade (0/5/0), Produktions-/Kalibrationsänderung (2/0/0), Änderung der Gebrauchsanweisung (0/1/0) und Vertriebsstopp (0/0/1).

Schlussfolgerungen: Meldungen zu IVD zur INR Selbsttestung stellen eine kleine, jedoch zur Therapiekontrolle wichtige Gruppe dar. Betroffen sind Analyzer und Teststreifen, die sich in Produktproblemen und korrektiven Maßnahmen unterscheiden. FSN leisten bei FSCA einen wichtigen Beitrag zur Verminderung vom Produkt ausgehender Risiken. Im Laufe des Untersuchungszeitraumes wurden Form und Inhalt der FSN zunehmend verbessert. Das Europäische Marktüberwachungssystem leistet einen wichtigen Beitrag zur Verbesserung der Sicherheit von IVD.
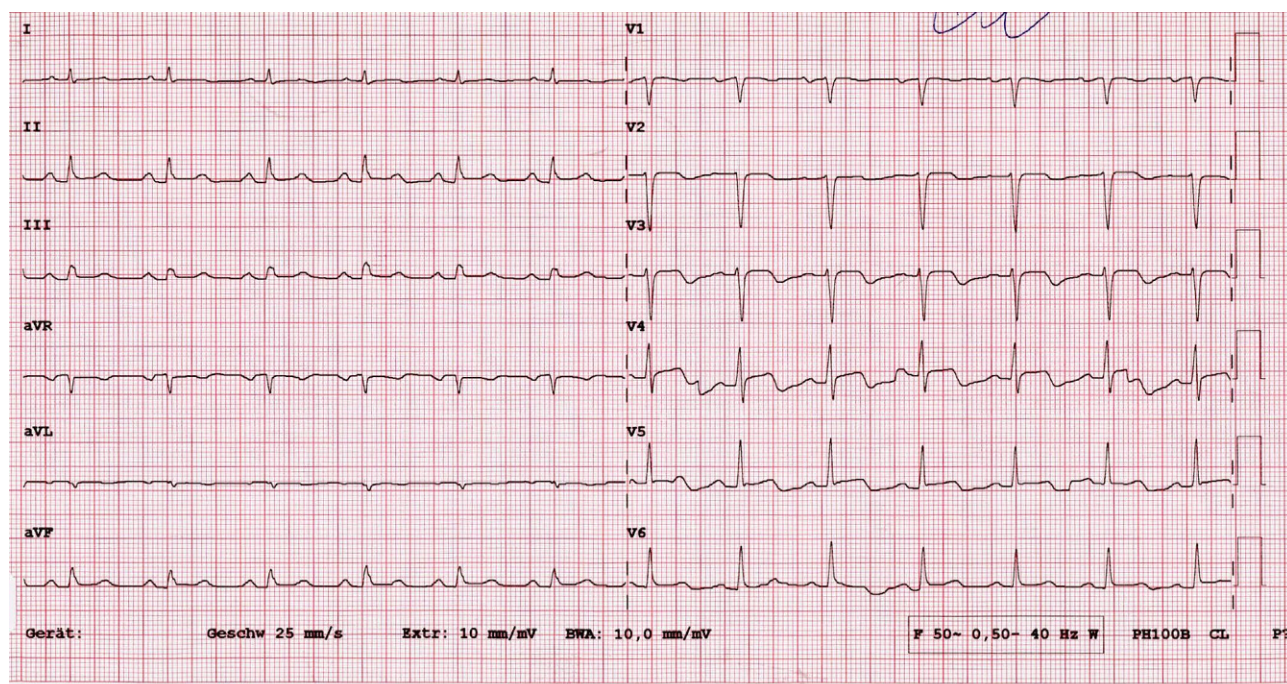
Abb. 1 | PS 19/19-6 Electrocardiogram at the time of patient's admittance

PS 19/19-6

„Wellens' sign" on ECG in a patient suffering from Takotsubo cardiomyopathy

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Introduction: A female patient, aged 61 years, presented at the emergency department for a possible right knee injury after a gardening accident. As she also reported acute dyspnea and chest pain, she was finally transferred to the cardiology department. At the ward, an electrocardiogram (ECG) was recorded that revealed sinus rhythm with 78 beats per minute and normal PQ interval. Remarkable were biphasic T waves from lead V2 to V4 and a ST elevation in lead V5 and V6, respectively (Fig. 1). Serum troponin T and pro-BNP levels were elevated (228 pg/ml; reference value: 0–14 pg/L and 6429 pg/ml; reference value: 0–376 pg/L, respectively). All other relevant laboratory parameters were normal. Echocardiography revealed a reduced ejection fraction of the left ventricle (45 %) with akinesia of the apex and all adjacent wall segments. However, the mid-ventricular and basal segments contracted normally. Due to the clinical presentation, the ECG findings, the elevated troponin levels and impaired regional contractility in echocardiography, coronary angiography was performed and showed normal coronary arteries. However, ventriculography confirmed the suspected diagnosis of Takotsubo cardiomyopathy. Beta-blocker therapy (Carvedilol 12.5 mg twice daily) was initiated and resulted in recovery of left ventricular contractility (ejection fraction of 65 %) and discharge with of the patient after 6 days in hospital.

Methods: –

Results: –

Conclusion: The Wellens' sign describes typical ECG findings accompanied with chest pain in patients suffering from a proximal significant stenosis of the left anterior descending artery ("widow-maker-lesion"). These ECG findings are usually biphasic (seldom completely inverted) T-waves in the presence of preserved R waves in the leads V1 to V4 In rare cases (like ours), these ECG abnormalities can also be found in the absence of coronary artery disease in patients suffering from Takotsubo cardiomyopathy.

PS 19/19-7

Steady-state and pulsatile haemodynamic characterization of aldosterone-renin ratio from physiological to pathological ranges

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Introduction: There has been an extension in antihypertensive treatment lately, since an additional effect on treating resistant hypertension by aldosterone-antagonists has been revealed. This implicates that aldosterone is not only a predominant cause for resistant hypertension, but also plays a pivotal role in the pathophysiology of primary hypertension.

Based on an increased aldosterone-to-renin ratio (ARR), a marker of inappropriate aldosterone activity, the prevalence of primary hyperaldosteronism is claimed to be as high as 10 % to 15 %, whereas Conn's classical adenoma would be concerning only less than 2 %. Aldosterone is known to cause changes in arteriolar vasoactive tone and sodium homeostasis and to increase arterial stiffness and wave reflections, both markers of increased cardiovascular risk. Moreover, other haemodynamic alterations have been described, among them increased peripheral resistance and associated changes in cardiac output. The aim of our study was a noninvasive, 24-hour deep hemodynamic phenotyping of untreated patients across a spectrum of aldosterone-to-renin ratios, starting in the physiological range and extending into pathological ranges.

Methods: We included 28 consecutive patients undergoing etiological assessment for elevated blood pressure (BP), who were naive to any antihypertensive treatment. Blood pressures and haemodynamic parameters were assessed using a brachial oscillometric sphygmomanometer, used for 24 h BP monitoring. The device has been non-invasively and invasively validated for measuring brachial BP, central BP, wave reflections (Augmentation Index–AIx; AIx normalized for heart rate 75–

Tab. 1 | PS 19/19-7 Elevated and normal ARR in steady-state and pulsatile haemodynamic parameters

	ARR <1.1 nd/dl/ µU/ml [mean (SD)]	ARR >1.1 nd/dl/µU/ ml [mean (SD)]	*p*-value
bSBP (mmHg)	145 (18)	153 (11)	0.20
DBP (mmHg)	95 (15)	101 (7)	0.24
bPP (mmHg)	50 (9)	52 (5)	0,59
cSBP (mmHg)	134 (17)	143 (9)	0,13
AIx (%)	21 (9)	31 (6)	0,01
AIx75 (%)	22 (9)	29 (6)	0,03
AP (mmHg)	9 (5)	13 (3)	0,01
aoPWV (m/s)	7,5 (1,7)	8,7 (1,5)	0,08
SV (L)	0,07	0,07	0,72
CO (L/min)	5,42	5,09	0,13
Peripheral resistance (dyn sec cm-5)	1,33	1,49	0,02

Fig. 1 | PS 19/19-7 Correlation of ARR and peripheral resistance

AIx75; augmentation pressure–AP), estimated aortic pulse wave velocity (aoPWV), stroke volume, cardiac output and peripheral resistance. Renin- and Aldosterone concentration were tested in peripheral blood sample using commercially available ChemilumineszenzImmunoAssay. Normal distribution was tested, using Shapiro-Wilk test. Parameters without normal distribution were log-transformed for further analysis. Correlations between quantitative variables were made by calculating the coefficient r of Pearson. A P value <0.05 was considered statistically significant.

Results: Mean age was 47.8 years (range 24–75), 71.4 % were men, 10.7 % smokers. Renal function was preserved, mean serum potassium was 4.1 mmol/l (SD 0.4). ARR was not correlated with 24-hour mean values of brachial or central BP. There was a moderate, direct relationship between ARR and 24-hour measures of wave reflections AIx, AIx75 and AP ($r = 0.53$, 0.42, and 0.47, respectively; $p = 0.004$, 0.025, and 0.01) and aortic stiffness (aoPWV; $r = 0.47$, $p = 0.01$). There was a stronger, positive relationship between ARR and 24-hour peripheral resistance ($r = 0.60$, $p = 0.0007$), and an inverse relationship between ARR and 24-hour cardiac output ($r = -0.48$, $p = 0.01$). These relationships were observed across the entire spectrum of ARR–Figure. Patients with elevated ARR (>1.1 nd/dl/µU/ml) ($n = 10$) had significantly higher values for AP, AIx, AIx75, and peripheral resistance, whereas brachial and central BPs did not differ significantly–Table.

Conclusion: Our results indicate that changes in ARR are associated with profound changes in steady-state as well as pulsatile haemodynamics. Both can be detrimental in the long run. Our haemodynamic results are well in line with previous findings that aldosterone exerts a direct effect on vascular smooth muscle cells and endothelial cells, leading to vascular dysfunction.

Abb. 1 | PS 19/19-8 Levocardiographie

PS 19/19-8

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. Im vorliegenden Fall stellen wir eine andere, nicht ischämiebedingte Ursache vor.

Methoden: In der kardiologischen Ambulanz wurde ein 49 Jahre alter männlicher Patient mit einem pathologischen Ergometrie-Befund ohne bekannte kardiovaskulären Erkrankungen vorstellig. Neben einem Nikotinkonsum war eine positive Familienanamnese bezüglich einer koronaren Herzerkrankung dokumentierbar. In der nach dem WHO-Protokoll durchgeführten Ergometrie zeigten 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.

Resultate: In der Koronarangiographie zeigten sich unauffällige glattwandige linke und rechte Herzkranzgefäße. In der Levocardiographie zeigte sich bei guter Linksventrikelfunktion eine auffällige inferioapikoseptale Ausbuchtung von ca

Abb. 2 | PS 19/19-8 Cardiales MRT

20 ×15 mm. Die Kontrastmittelverteilung in diesem Areal gab deutliche Hinweise auf Trabekel. Zur weiteren Abklärung wurde ein cardiales 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. Dies führte zu einer Dyskinesie, welche zu einer Reduktion der rechtsventrikulären Ejektionsfraktion führte. Ein Thrombus oder ein Links-Rechtsshunt konnten nicht nachgewiesen werden. Da der Patient beschwerdefrei war, wurde eine jährliche echocardiographische Kontrolle und die Durchführung eines Holters alle 2–3 Jahre sowie sofort bei Palpitationen vereinbart.

Schlussfolgerungen: Apikale Divertikel wie durch diesen Fall dargestellt, können zu auffälligen ischämietypischen Bildern führen. Kongenitale ventrikuläre Aneurysmen und Divertikel stellen seltene Anomalien dar, die durch immer höhere Qualität und breitere Verfügbarkeit von kardialen MRTs besser diagnostiziert werden können. Eine einheitliche Nomenklatur liegt nicht vor, ebenso gibt es wegen der geringen Fallzahl keine evidenzbasierten Therapievorschläge. Wünschenswert wäre die Einführung einer einheitlichen Nomenklatur sowie die Durchführung eines europaweiten Registers.

POSTERSITZUNG 20 – INTERVENTIONELLE KARDIOLOGIE 2

PS 20/20-1

Save your brain–does the sentinel cerebral protection device work?

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	Without Cerebral Protection Device	With Cerebral Protection Device	p-Value
Age (years)	80.3±7.1	81.5±7.3	P=0.233
Female (%)	47.3	47.7	P=0.967
Previous stroke (%)	6.9	3.2	P=0.373
Peripheral artery disease (%)	9.8	4.8	P=0.305
Glomerular filtration rate (ml/min)	52	56	P=0.283
Coronary artery disease (%)	63.1	57.1	P=0.370
EuroScore II (%)	6.02±5.43	6.08±6.05	P=0.937
Predilation (%)	48.3	47.6	P=0.925
Postdilation (%)	29.3	31.7	P=0.707
Procedure time (min)	59	66	P=0.152
Baloon-expandable valve (%)	33.2	34.9	P=0.797
Mean pressure gradient (mmHg)	46±14	51±17	P=0.028

Fig. 1 | PS 20/20-1

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Introduction: Transcatheter aortic valve implantation (TAVI) is increasingly used for the treatment of severe symptomatic aortic stenosis (AS), also in low-risk patients. Periprocedural embolic stroke is rare, but potentially associated with considerable morbidity and mortality. Thus, there is great interest in preventing any cerebral embolic event. At present, only one cerebral embolic protection systems (CPS) is commercially available and little is known about its efficacy in preventing stroke during TAVI. The Sentinel CPS is a FDA-approved system consisting of two inter-connected filters that are placed in the brachiocephalic trunk and the left carotid artery via the right radial artery.

Methods: Consecutive patients undergoing TAVI between 11/2018 and 11/2019 were enrolled. Consecutive patients treated by one operator received the Sentinel device, if anatomically possible. Periprocedural stroke rate, as defined by VARC2-criteria, and mortality up to 7 days after procedure was assessed. Descriptive statistics was performed to identify baseline variables associated with elevated risk of stroke and Cox-regression analysis was used to investigate its influence on outcome.

Results: 268 patients (47.4 % female, 81 ±7 years) were included. In 74 patients (27.6 %), a Sentinel CPS was used, in 63 (23.5 %) it was positioned correctly in the brachiocephalic trunk and left carotid artery. Only these patients were considered Sentinel-protected. Patients with and without Sentinel presented with similar baseline characteristics with regard to age (no CPS vs CPS; 80.3 vs 81.5 years; $p=0.233$), sex (female 47.3 % vs 47.7 %; $p=0.967$), previous stroke (6.9 % vs 3.2 %; $p=0.373$), peripheral artery disease (9.8 % vs 4.8 %; $p=0.305$), coronary artery disease (63.1 % vs 57.1 %; $p=0.370$), and kidney function (GFR 52 vs 56 ml/min/m²; $p=0.283$). The EuroScore II (6 % vs 6 %; $p=0.937$), periprocedural predilation (48.3 % vs 47.6; $p=0.925$), postdilation (29.3 % vs 31.7 %; $p=0.707$) and procedure time (59 min vs 66 min; $p=0.152$) were not different. In total, 15 strokes (5.6 %) occurred, of which 9 (3.3 %) were disabling strokes as defined by the VARC2-criteria. In Sentinel-protected patients undergoing TAVI, no periprocedural stroke was observed (no CPS 7.3 % vs 0.0 %; $p=0.026$).

Conclusion: Our results suggest that Sentinel CPS significantly reduces periprocedural stroke rates in patients undergoing TAVI compared to patients without CPS. However, the study population is small and randomized trials are still needed.

PS 20/20-2

The right heart in patients undergoing transcatheter aortic valve implantation: an integrative approach

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Combined: Heart Failure Hospitalization and/or Death

Log-rank: p<0.001
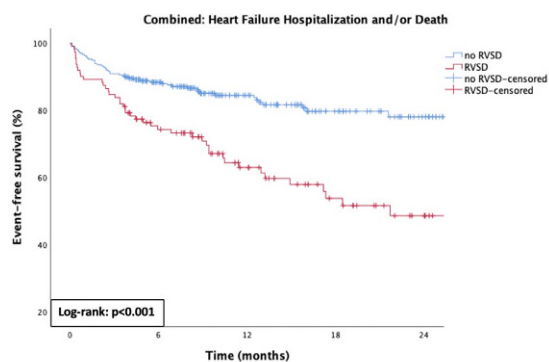
No. at risk	0	6	12	18	24
no RVSD	295	215	124	63	33
RVSD	111	72	42	25	13

Abb. 1 | PS 20/20-2 Kaplan-Meier survival curves

Introduction: Right ventricular (RV) function is strongly associated with outcome in heart failure. Whether it also adds important prognostic information in patients undergoing transcatheter aortic valve implantation (TAVI) is unknown.

Methods: We consecutively enrolled patients with severe aortic stenosis (AS) scheduled for TAVI and performed preprocedural cardiac magnetic resonance (CMR) as well as transthoracic echocardiography (TTE). Kaplan-Meier estimates and multivariate Cox regression analyses were used to identify factors associated with outcome. A composite of heart failure hospitalization and/or cardiovascular death was selected as primary study endpoint.

Results: 406 consecutive patients (80.8 ±7.3 years; 48 % female) were prospectively included, 205 (51 %) underwent CMR imaging. 111 (27 %) patients presented with RV systolic dysfunction (RVSD) assessed by an integrative approach including RV ejection fraction (RVEF) <45 % on CMR and conventional TTE parameters (RV fractional area change <35 %, TAPSE <17 mm, RV free-wall longitudinal strain >–20 %). RVSD was associated with male sex (65 vs. 48 %; $p = 0.002$), New York Heart Association functional status (NYHA ≥III: 84 vs. 58 %; $p < 0.001$), NT-proBNP serum levels (8268 vs. 3296 pg/mL; $p < 0.001$), renal failure (creatinine: 1.5 vs. 1.3 mg/dL; $p = 0.001$), atrial fibrillation (AF: 56 vs. 36 %; $p < 0.001$), and use of loop diuretics (64 vs. 42 %; $p < 0.001$). On CMR, RVSD was associated with left ventricular (LV) volumes (end-diastolic: 186 vs. 136 mL; end-systolic: 117 vs. 52 mL; both $p < 0.001$) and EF (40 vs. 64 %; $p < 0.001$). A total of 94 events (72 deaths, 22 heart failure hospitalizations) occurred during follow-up (mean 14.0 ±10 months). While LVSD (LVEF <50 %) was not significantly associated with outcome, RVSD showed a strong and independent association with event-free survival by multivariate Cox regression analysis (HR 2.24, 95 % CI: 1.26–4.00; $p = 0.003$), which was adjusted for all relevant CMR (LV volumes and EF) and TTE parameters (mitral and tricuspid regurgitation, pulmonary artery pressure), cardiovascular risk factors (sex, NYHA, AF, use of diuretics), and routine biomarkers (NT-proBNP, creatinine).

Conclusion: RVSD rather than LVSD is an important predictor of outcome in patients undergoing TAVI. RV function might thus add useful prognostic information on top of established risk factors.

PS 20/20-3

Acute hemodynamic effects of iatrogenic interatrial shunts after percutaneous edge-to-edge mitral valve repair

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Introduction: Implantable interatrial shunt devices improve pulmonary vascular function in patients with heart failure by transferring richly oxygenated blood to the right heart. Whether iatrogenic atrial septum defects (iASDs) after percutaneous edge-to-edge mitral valve repair (pMVR) are also associated with beneficial hemodynamic effects has not been investigated.

Methods: We consecutively enrolled patients with relevant functional (FMR) and degenerative mitral regurgitation (DMR) scheduled for pMVR. Invasive hemodynamic assessments were performed prior to and immediately after the procedure.

Results: 97 consecutive patients (75.4 ±9.1 years; 58 % female) were prospectively included, 65 (66 %) presented with relevant FMR. At baseline, when compared to the DMR group, FMR was associated with worse left ventricular (LV) function (LV ejection fraction: 39 vs. 49 %; $p = 0.001$), higher NT-proBNP levels (7404 vs. 5214 pg/mL; $p = 0.023$), worse renal function
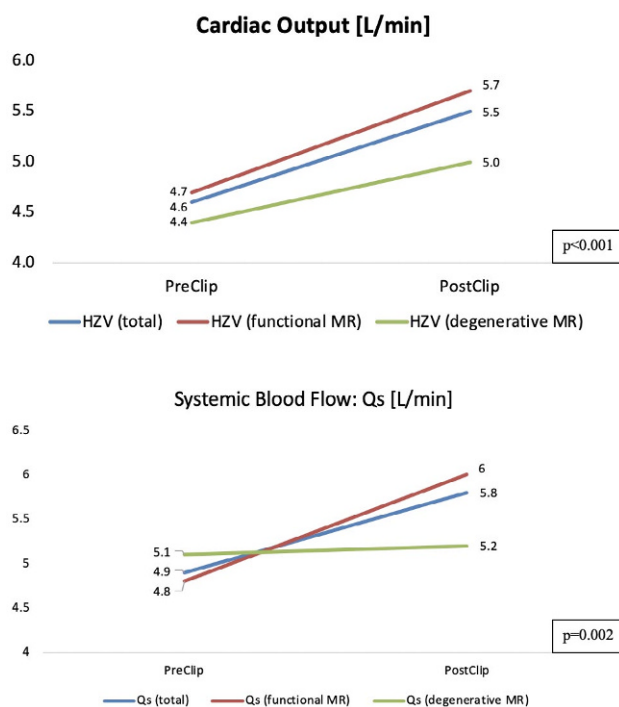
Abb. 1 | PS 20/20-3 Invasive hemodynamics I–Cardiac Output & Qs

Pulmonary vascular resistance [dynes*sec/cm⁵]
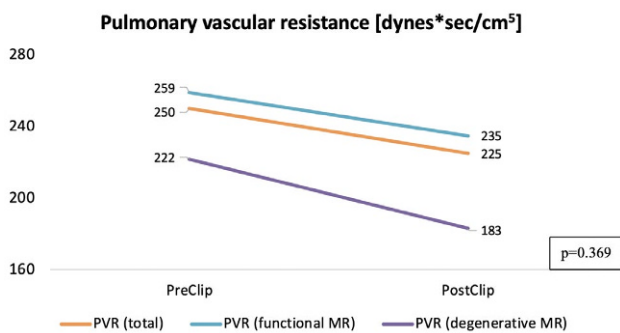
Abb. 2 | PS 20/20-3 Invasive hemodynamics II–PVR

(serum creatinine: 1.7 vs. 1.3 mg/dL; $p = 0.019$), and higher usage of spironolactone (68 vs. 42 %; $p = 0.018$) and sacubitril/valsartan (33 vs. 0 %; $p < 0.001$). Following pMVR, cardiac output (CO) and systemic blood flow (Qs) increased significantly (CO: 4.6 to 5.5 L/min; $p < 0.001$; Qs: 4.9 to 5.8 L/min; $p = 0.002$), with more pronounced changes in the FMR subgroup (ΔCO: 1.0 vs. 0.6 L/min; ΔQs: 1.2 vs. 0.1 L/min; Fig. 1), when compared to DMR. Pulmonary blood flow (Qp) increased by 26 % (4.3 to 5.4 L/min; $p = 0.008$), accompanied by a raise in pulmonary artery (PA) oxygen (O2) saturation from 73 to 77 % ($p < 0.001$). Arterial O2 saturation levels remained unchanged (98.3 to 98.7 %; $p = 0.165$), confirming no significant changes in systemic oxygenation. These changes were associated with a slight decline in pulmonary vascular resistance (PVR: 250 to 225 dynes*sec/cm5; $p = 0.369$, Fig. 2), and a tendency towards improvement of pulmonary compliance (PAC: 3.6 to 4.0 mL/mm Hg; $p = 0.414$).

Conclusion: Invasively measured CO, Qs, Qp, and mixed-venous PA O2 saturation increased immediately after pMVR, alongside with potentially beneficial effects on pulmonary vasculature with marked improvements in PVR and PAC. These changes were more pronounced in the FMR subgroup. Further studies are required to assess long-term hemodynamic effects and underlying mechanisms of persistent iASDs on pulmonary vascular function.

PS 20/20-4

Frailty in a contemporary TAVR cohort

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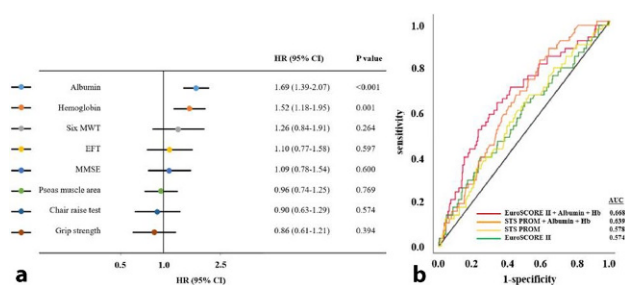
Abb. 1 | PS 20/20-4

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Introduction: Frailty significantly reduces the potential to recover after transcatheter aortic valve replacement (TAVR) and has been linked to excess mortality following treatment. However, studies on frailty are mostly historical and were conducted during early TAVR experience. We aimed to perform comprehensive assessment of physical and mental capability and study its prognostic implications in a contemporary TAVR cohort.

Methods: Between September 2018 and December 2019 consecutive patients underwent frailty assessment prior to TAVR and were prospectively followed. The following frailty markers were collected: all 4 items of the Essential Frailty Toolset (EFT: five-times chair raise test, mini mental status examination (MMSE), serum levels of hemoglobin and albumin), six-minute walk test, five-meters walk time, grip strength as assessed with hand grip dynamometer, and psoas muscle area by computed tomography. Scaled hazard ratios were created for Cox regression analysis to allow better comparison.

Results: In total, 300 patients (81.2 ± 6.9 y/o, 45 % female, EuroSCORE II 4.4 ± 2.2 %, STS PROM 4.3 ± 3.4 %) with complete frailty assessment were included, of whom 93 (31.0 %) were classified as "frail" according to the EFT (Score ≥3 of 5 points). Among patients receiving TAVR ($n = 292$), 9.9 % ($n = 29$) had died 33.3 ± 19.5 weeks following intervention. Interestingly, physical (chair raise test, walking tests, grip strength) and cognitive tests (MMSE) were unable to predict mortality, as were psoas muscle area/body surface area and the EFT (p for all >0.05). Conversely, serum levels of albumin (hazard ratio [HR] 1.69, 95 % confidence interval [CI] 1.39–2.07, $p < 0.001$) and hemoglobin (HR 1.52, 95 % CI 1.18–1.95, $p = 0.001$) were significantly associated with outcome (Fig. 1a), alongside with EuroSCORE II (HR 1.21, 95 % CI 1.05–1.39, $p = 0.008$). When albumin and hemoglobin were integrated in traditional surgical risk scores (by adding 1 point each if albumin <3.5 g/dl and if hemoglobin <12/13 g/dl for females/males, respectively, according to the EFT cut-off values), this significantly improved the prognostic power of EuroSCORE II and STS PROM Score by receiver operating characteristic analysis (p for both <0.001, Fig. 1b).

Conclusion: Serum albumin and hemoglobin are easily accessible laboratory markers that help to risk stratify AS patients undergoing TAVR. Assessment of physical and cognitive performance may become less important as TAVR is moving more towards patients with low to intermediate operative risk.

PS 20/20-5

Fluid overload prior to TAVR–a worrying condition?

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Introduction: Fluid overload (FO) and impaired kidney function are frequent findings in patients scheduled for transcatheter aortic valve replacement (TAVR). The optimal fluid management strategy prior to TAVR with the aim to prevent
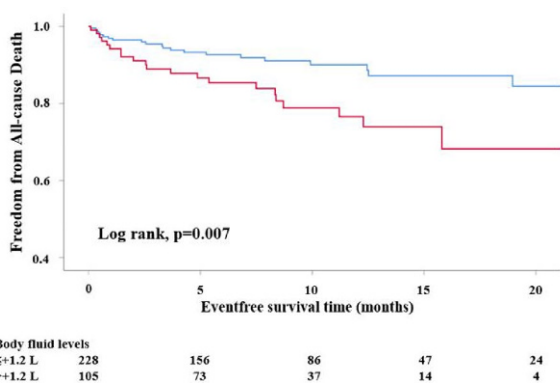
Fig. 1 | PS 20/20-5 Kaplan Meier Estimates

procedural complications, such as acute kidney injury (AKI), is largely unknown. We aimed to study whether FO at the time of TAVR is linked with AKI and mid-term outcomes.

Methods: Consecutive patients underwent fluid status measurement with bioelectrical impedance spectroscopy (BIS), echocardiography, ECG, and clinical and laboratory assessment prior to TAVR. FO was defined according to the third tertile of fluid levels ($\geq$+1.2L). AKI was defined as a $\geq$1.5-fold and/or $\geq$0.3 mg/dL increase in serum creatinine within 7 days post-TAVR. AKI and all-cause death were selected as study endpoints. Binary logistic, Cox regression, and Kaplan Meier analysis were used as statistical methods.

Results: 333 patients (81.5 ±7.2 y/o, 47.4 % female) were included and prospectively followed. FO was linked to advanced stages of disease, characterized by poorer left ventricular ejection fraction, more severe estimated pulmonary artery pressure and tricuspid regurgitation, and higher serum levels of NT-proBNP and creatinine (p for all <0.05). In total, 12.9 % (n=44/333) of patients experienced post-TAVR AKI, and 12.6 % (n=42/333) had died 10.4 ±6.7 months following treatment. By multivariate binary logistic regression analysis, serum creatinine levels (odds ratio [OR] 3.100, 95 % confidence interval [CI] 1.744–5.510, p<0.001), and procedural duration (OR 1.014, 95 % CI 1.004–1.023, p=0.006), but not body fluid levels (p>0.05) were associated with AKI. Conversely, FO was associated with mortality by Kaplan-Meier estimates (log-rank, p=0.007, Fig. 1), and fluid levels were independently linked with survival by multivariate Cox regression analysis (hazard ratio 1.163, 95 % CI 1.044–1.295, p=0.006), alongside with serum albumin levels (p<0.001), procedural stroke (p=0.001), EuroSCORE II (p=0.016), and body mass index (p=0.022). Interestingly, FO was linked to a 3.7-fold increased risk of post-interventional pacemaker dependency (OR 3.656, 95 % CI 1.604–8.334, p=0.002) even after adjustment for pre-existing conduction disturbances and prosthesis characteristics.

Conclusion: FO prior to TAVR is independently associated with mid-term mortality and pacemaker need, but not AKI. When preparing AS patients with FO for TAVR, decongestive treatment appears to be safe without significant risk for AKI. Moreover, FO characterizes patients at high risk for whom early intervention may be considered irrespective of symptoms.

PS 20/20-6

Volume status in percutaneous mitral edge-to-edge repair–a therapeutic target?

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Introduction: Percutaneous edge-to-edge mitral valve repair (pMVR) has emerged as a valuable treatment option for patients with severe mitral regurgitation (MR) at prohibitive surgical risk. Following pMVR, MR reduction oftentimes entails improvement of congestion, whereas some patients fail to respond. We aimed to correlate post-interventional changes in volume status, as assessed by bioelectrical impedance spectroscopy (BIS), with evolution of MR, symptoms, and functional capacity.

Methods: Consecutive patients scheduled for pMVR underwent fluid status measurement using BIS, six-minute walk test (6MWT), echocardiography, and clinical and laboratory assessment on the day before and 3–6 months after pMVR. Patients were categorized into two groups according to post-interventional reduction (Δ<0.0L) or increase ($\Delta\geq$0.0L) in body fluid levels as compared to baseline measurements, respectively.

Results: 50 patients (74.8 ±8.6 y/o, 36.0 % female) undergoing pMVR for functional (70 %, n=35) and primary MR (30 %, n=15) were included and followed-up 19.5 ±11.0 weeks after intervention. Procedural success, defined as a MR≤2, was achieved in 94.0 %. In total, 44 % of patients (n=22/50) showed a decline in fluid levels at follow-up. Subjects with improvement of congestion displayed higher fluid levels at baseline (+2.5 ±2.0L vs. +0.4 ±1.4L, p=0.001), but did not differ significantly from those without reduction in fluid levels with respect to MR mechanism, renal function, other cardiovascular risk factors, and heart failure medication including diuretics (p for all >0.05). An improvement in fluid levels was associated with a higher percentage of MR reduction <2 (40.9 % vs. 14.3, p=0.033), significantly enhanced symptomatic improvement (New York Heart Association functional class reduction≥II levels: 47.6 % vs. 10.7, p=0.004, Fig. 1a), and a greater decline in NT-proBNP
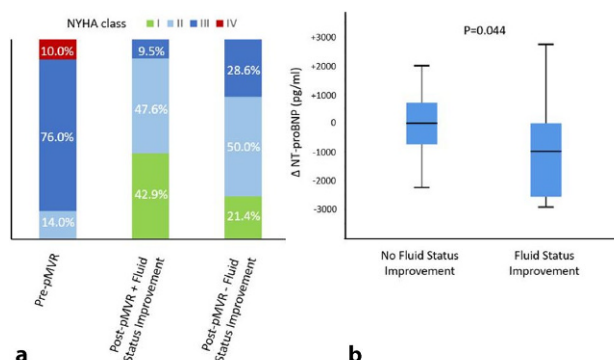
NYHA indicates New York Heart association; pMVR, percutaneous mitral valve repair; NT-proBNP, N-terminal prohormone of brain natriuretic peptide.

Abb. 1 | PS 20/20-6 Evolution of dyspnea and NT-proBNP post-pMVR

levels (Δ–2380 ±6296 vs. Δ–533 ±3879, $p = 0.044$, Fig. 1b), as compared to persistent congestion. Moreover, there was a trend towards increased improvement in walking distance by 6MWT for patients with congestion recovery (Δ+80 ±140 meters vs. Δ+42 ±81, $p = 0.145$).

Conclusion: Decongestion following pMVR is linked to symptomatic and functional improvement, as well as more reduction in MR. Hence, volume status appears to be an attractive therapeutic target for clinicians even after successful pMVR to alleviate symptoms, improve functional capacity, and potentially ameliorate outcomes.

PS 20/20-7

A rare complication after transfemoral aortic valve-implantation (TAVI): Aorto-right ventricular fistula, report of three cases

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Introduction: TAVI is a sharp increasing treatment method for elderly patients with severe aortic stenosis and elevated surgical risk. One oft most feared complications during the acute intervention is annular rupture, that yields a high mortality rate despite prompt surgical intervention. We here report on three patients with concealed annular rupture resulting in aorto-right ventricular fistula successfully treated wit an Amplatzer occluder device (Abbott vascular, Santa Clara, CA).

Methods: Case Summary: Three patients (80 ±10 years, 2 females) with severe aortic stenosis, who underwent TAVI (balloon expandable Edwards Sapiens III, Edwards Lifesciences Corporation, Irvine CA) developed annular rupture with aorto-right ventricular fistula. After the intervention, they suffered from worsening dyspnea (NYHA III+) and pulmonary hypertension (sPAP mean 64 mm Hg). According to a heart team decision, all patients underwent successful transcatheter fistula closure with an Amplatzer Vascular Plaque III device.
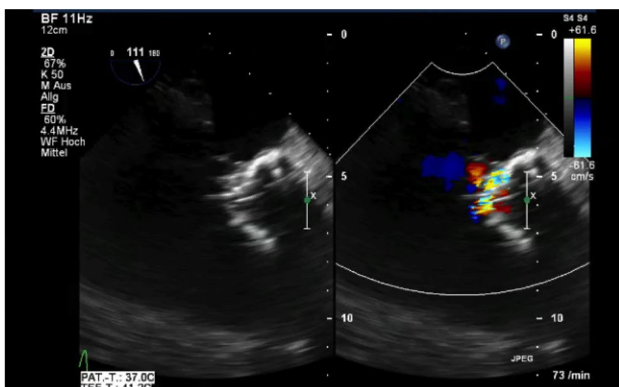
Abb. 1 | PS 20/20-7 Aorto-Right Ventricular Fistula

Results: The last performed TTE (mean follow-up of 25 months ±4 months) showed a good and lasting result with no or just a minimal Re-Shunt, < moderate paravalvular regurgitation and reduced sPAP (mean 38 mm Hg).

Conclusion: Successful transcatheter closure with the use of an Amplatzer occluder device is an efficient technique for the treatment of Aorto-RV fistula after TAVI also in long term follow-up. To our knowledge, this is the largest series of this rare complication so far.

PS 20/20-8

A contemporary definition of periprocedural myocardial injury after percutaneous coronary intervention of chronic total occlusions

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Introduction: Coronary chronic total occlusion (CTO) recanalization represents the most technically challenging percutaneous coronary interventions (PCI). The complexity harbours a significant increased risk risk for complications with CTO PCI as compared to non-CTO PCI. However there are evidenced biomarker cut-off levels that help to identify those patients at risk for an unfavorable clinical outcome. This study aimed to assess the prognostic impact of postprocedural troponin T increase and mortality in patients undergoing CTO-PCI in order to define the threshold, where procedural related myocardial injury drives mortality.

Methods: We enrolled a total of 3712 consecutive patients undergoing PCI for at least one CTO lesion and performed comprehensive troponin T measurements 6, 8, and 24 h after the procedure. All-cause mortality was defined as the primary study endpoint.

Results: Using spline curve analysis, we observed that a more than 18-fold increase of troponin above the upper reference limit (URL) is significantly associated with mortality. In the Cox regression analysis we observed a crude hazard ratio (HR) of 2.32 (95 %CI 1.83–2.93, $P < 0.001$) for a ≥18fold increase
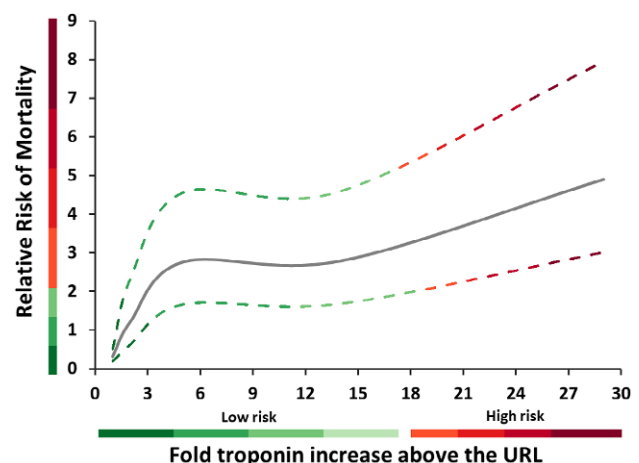
Fig. 1 | PS 20/20-8
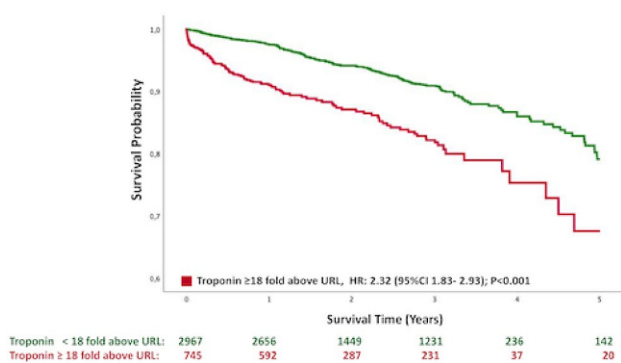
Fig. 2 | PS 20/20-8

compared to patients with post-procedural troponin increase <18-fold of the URL. Results remained virtually unchanged after bootstrap- or clinical confounder-based adjustment.

Conclusion: This large-scale outcome study demonstrates for the first time the prognostic value of post-procedural troponin T elevation after PCI in CTO patients. We could define a threshold for procedure related myocardial injury in CTO patients that differ non- CTO patients and may help guide the postprocedural clinical care in this high risk patient population.

POSTERSITZUNG 21 – PULMONALE HYPERTENSION 2

PS 21/21-1

Hemodynamics in cardiac amyloidosis

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Introduction: Intracardiac filling and pulmonary arterial pressures play a central role in various heart failure entities, as they are strong predictors of outcome. However, their role in patients with cardiac amyloidosis (CA) is less clear. Therefore, we aimed to characterize hemodynamic profiles of CA patients and assess their association with outcomes.
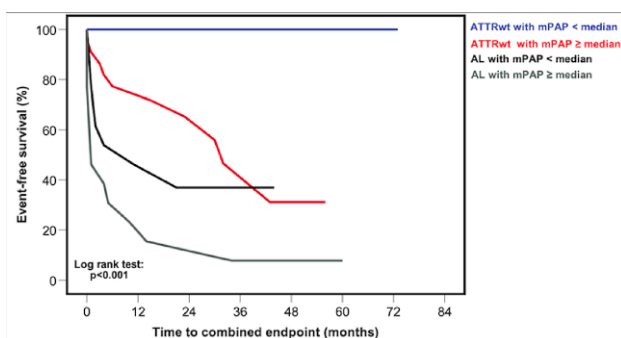
Methods: The present study was conducted within a prospective, national CA registry. CA was diagnosed in accordance with current recommendations. Consecutive CA patients underwent invasive hemodynamic, clinical, laboratory, and echocardiography assessment, as well cardiac magnetic resonance imaging with T1-mapping. Hemodynamic parameters of interest were mean PAP (mPAP), pulmonary artery wedge pressure (PAWP), right atrial pressure (RAP), cardiac index (CI), and stroke volume index (SVi). The main outcome measure was a combined endpoint consisting of hospitalization for heart failure or death from cardiovascular causes.

Results: Between March 2012 and January 2019, 61 patients, 35 (57.4 %) with wild-type transthyretin amyloidosis (ATTRwt) and 26 (42.6 %) with light chain amyloidosis (AL) were included in the present study. Median *N*-terminal prohormone of brain natriuretic peptide was 3552 pg/mL (IQR: 1501–7357) and almost half ($n=30$, 49.2 %) of all patients were in New York Heart Association class ≥III. Patients had marked elevations in pulmonary arterial and intracardiac filling pressures (mPAP: 30.0 mm Hg, IQR: 25.5–36.5; RAP: 11.0 mm Hg, IQR: 7.3–16.8; PAWP: 20.0 mm Hg, IQR: 16.5–24.0), whereas CI (2.4L/min/m2, IQR: 1.9–2.8) and SVi (30.7 mL/m2, IQR: 25.2–41.6) were mostly within normal ranges. Hemodynamic parameters did not differ between ATTRwt and AL patients. In ATTRwt median mPAP [hazard ratio (HR): 1.130, 95 % confidence interval (CI): 1.006–1.269; $p=0.040$] and pulmonary vascular resistance (HR: 1.010, 95 % CI: 1.000–1.020; $p=0.046$) were independent predictors of outcome. However, no hemodynamic parameter was associated with outcome in the AL group. Furthermore, AL patients with mPAP ≥ median had the worst, whereas ATTRwt patients with mPAP < median had the best event-free survival. Of note, there was no difference in event-free survival between patients with ATTRwt and median ≥mPAP and AL patients < median mPAP (Fig. 1).

Conclusion: Cardiac ATTRwt and AL patients feature markedly elevated intracardiac as well as PAPs and show similar hemodynamic profiles. However, hemodynamic parameters are of greater prognostic relevance in ATTRwt, thus potentially providing a new therapeutic target.

PS 21/21-2

Postcapillary pulmonary hypertension: perfomance diagnostics by cardiopulmonary exercise testing and therapeutic management, a case report

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Introduction: Postcapillary pulmonary hypertension (PH) is seen in heart failure patients with preserved ejection fraction (HFpEF), which is to be distinguished from precapillary PH, also known as idiopathic pulmonary arterial hypertension (IPAH). Cardiopulmonary exercise testing (CPET) is a good method to quantify dyspnea symptoms and has functional prognostic value for the progression of pulmonary vascular disease. Important parameters are peak volume of oxygen (VO2), minute ventilation/carbon dioxide production (VE/VCO2)
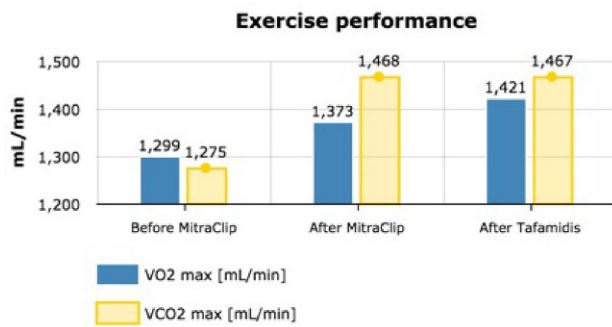
Abb. 1 | PS 21/21-1

Abb. 1 | PS 21/21-2 Exercise performance quantified by VO2 max and VCO2 max by CPET

CPET	Before MitraClip	After MitraClip	After tafamidis 6 months
VO2 max [mL/min] (% of predicted)	1299 (83%)	1373 (88%)	1421 (93%)
VCO2 max [mL/min]	1275	1468	1467
O2-pulse max [mL]	11,9	14,3	15,6
VE/VCO2 slope	35,60	33,89	32,53
6-Minute Walk Test (m)	437	470	458

Abb. 2 | PS 21/21-2 CPET results and 6MWT at baseline and follow-ups

slope, peak volume of carbon dioxide (VCO2) and oxygen pulse (O2-pulse).

Methods: Case presentation: This is the case of a 79-year-old male multimorbid patient with wild type cardiac transthyretin (ATTR) amyloidosis and severe mitral regurgitation (MR), who presented with dyspnea (NYHA 2) in 10/2018. Significant coronary artery disease was excluded by coronary angiography. Right heart catheterization disclosed post capillary pulmonary hypertension: mean pulmonary artery pressure (mPAP) of 42 mm Hg and mean pulmonary capillary wedge pressure (mPCWP) of 25 mm Hg. ECG showed atrial fibrillation, bifascicular block and negative T-waves in aVR, V1, V2, V3. High grade left ventricular concentric hypertrophy (IVS = 26 mm), severe MR (EROA = 35 mm2) and enlargement of left and right atria (LA = 68 mm, RA = 68 mm) were demonstrated by transthoracic echocardiography. Transesophageal echocardiography showed no thrombi in left atrium including left atrial appendage. Management: In 12/2018 the patient received a MitraClip with no complications and was started on tafamidis treatment in 04/2019. Patient underwent regular follow-ups. The dynamic response of the heart after MitraClip implantation and medication treatment for cardiac amyloidosis was evaluated by comparing the patient's exercise capacity before and after treatment with CPET.

Results: Cardiopulmonary exercise testing results: CPET by a cycle ergometer was used to measure the dynamic response of the heart before and after the procedure. A ramp protocol, adapted to reach maximal capacity after 8–10 min was chosen for the test. The parameter of main interest was maximal oxygen uptake (VO2 max [ml/min]), the major factor known to limit exercise capacity. Results of CPET parameters at baseline, after MitraClip implantation and after 6 months of tafamidis treatment are illustrated in Table 1 and Fig. 1.

Conclusion: Cardiopulmonary exercise testing is a useful diagnostic tool for progress monitoring and risk stratification in patients with post capillary pulmonary hypertension.

PS 21/21-3

Vorkommen von Vorhofflimmern in PAH-Patienten unter laufender Treprostinil-Therapie

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Einleitung: Eine Pulmonalarterielle Hypertonie (PAH) liegt nach den ESC/ERS-Guidelines 2015 vor, wenn der mittlere PA-Druck ≥25 mm Hg und der PCW ≤15 mm Hg, sowie ein PVR >3 Wood vorliegen [1]. Ein chronisch erhöhter Pulmonalarteriendruck kann in weiterer Folge zu einer Dehnung des rechten Atriums führen, was wiederum zu einem „electric remodelling" in diesem führt und somit als Auslöser für Vorhofflimmern (VHF) in Frage kommt. Eine Rhythmusstörung in einem bereits sensiblen Patientenkollektiv kann zu einer weiteren klinischen Dekompensation des Patientenzustandes führen [2]. Mit dieser retrospektiven Datenauswertung unserer Patienten wollen wir untersuchen wie häufig VHF in diesem Kollektiv vorkommt und welche klinischen Folgen daraus gezogen werden können.

Methoden: Für diese retrospektive Datenauswertung wurden alle Patienten, die unter Kombinationstherapie inklusive i. v. Treprostinil stehen und in unserem Studienkollektiv vorhanden sind eingeschlossen.

Resultate: Unser Kollektiv umfasst 80 PAH-Patienten. Als Ursache für die Pulmonalarterielle Hypertonie zeigt sich bei 17 Patienten eine CTEPH, bei 63 Patienten eine PAH (IPAH und assoziierte Formen). Insgesamt besteht bei 33 (41,3 %) der Patienten ein VHF (7 davon Paroxysmales VHF und 26 ein permanentes VHF). Die Patienten mit VHF sind im Schnitt älter als die Patienten ohne VHF (74,4 vs 63,2 Jahre). Das NT Pro BNP in pg/ml ist in Patienten mit VHF etwas höher, als in nicht VHF-Patienten (2963 vs. 2713). Patienten mit VHF legen im 6 min Walktest durchschnittlich 135 m weniger zurück (311 vs. 446 m). Die TRPG und der im Rechtsherzkatheter gemessene mittlere Pulmonal-arterielle Druck sind bei VHF-Patienten geringer. (TRPG 52,1 vs. 61,0, mPAP 40,3 vs. 51,5) Der links-atriale Durchmesser in der Echokardiographie ist im Durschnitt um 8,6 mm größer (47,6 vs. 39,0). Der mittlere rechts-atriale Druck liegt um

Tab. 1 | PS 21/21-3 PAH und Vorhofflimmern

	VHF	keinVHF
n = 80	33 (41,3 %)	47 (58,8 %)
Alter in Jahre	74,4	63,2
Paroxysmales VHF	7 (21 %)PermanentesVHF	26 (79 %)
NT Pro BN Pin pg/ml	2963 (32pts.)	2713 (46pts.)
6min Walking TestinMeter	311 (13pts.)	446 (17pts.)
TRPG	52,1 (31pts.)	61 (43pts.)
LA in mm	47,6 (32pts.)	39,0 (43pts.)
RA Mitteldruck in mmHg	12,0 (27pts.)	8,0 (40pts.)
PA Mittel druck in mmHg	40,3 (33pts.)	51,5 (45pts.)

PAH = pulmonalearterielle Hypertonie, VHF = Vorhofflimmern,-NT-Pro-BNP = N-terminalesprobrainnatriureticpeptide, TRPG = tricuspidregurgitationpeakgradient, LA = linkes Atrium, pts. = Patienten,-RA = rechtsatrial, mmHg = milimeter Quecksilber, PA = pulmonal-arteriell

4,0 mm Hg höher im VHF-Kollektiv (12,0 vs 8,0). 22 (66,6 %) der 33 Vorhofflimmerpatienten erhalten eine Vorhofflimmermedikation (die verwendeten Medikamente sind Concor, Lanitop, Isoptin, Sedacoron).

Schlussfolgerungen: Circa zwei Fünftel unserer Patienten unter Kombinationstherapie inklusive i. v. Treprostinil leiden zusätzlich unter VHF. Vergleicht man die erhobenen Surrogatparameter (NT Pro BNP, 6-MWT, mittlerer RA-Druck) mit der Risikostratifizierungstabelle des ESC sieht man, dass das VHF Kollektiv im Bereich des intermediären Risikos liegt. Dies erhöht die 1-Jahres-Mortalität von <5 % in einen Bereich von 5–10 %. Das Auftreten von Vorhofflimmern in unserem Kollektiv nimmt mit zunehmendem Alter der Patienten zu und hat einen Einfluss auf die Risikostratifizierung. Trotz adäquater Frequenzmodulation führt Vorhofflimmern zu einem NT-Pro-BNP Anstieg.

PS 21/21-4

Körperliche Leistungsevaluierung bei PatientInnen mit pulmonaler Hypertension

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Einleitung: Funktionstests erlauben Rückschlüsse auf die funktionelle Leistungsfähigkeit unter Alltagsbedingungen und dienen zur Überprüfung des Therapieerfolges. Gut etabliert und in der klinischen Praxis routinemäßig angewandt ist der 6-Minuten-Gehtest (6MWT), der jedoch Zeit und entsprechende räumliche Ressourcen benötigt. Eine wertvolle Alternative zum 6MWT könnte der 1-minütige Sit-to-Stand Test (STST) darstellen. Bisher wurde der STST bei Patienten mit COPD validiert, bei PatientInnen mit anderen kardiopulmonalen Erkrankungen fehlen entsprechende Daten. Ziel dieser Studie ist, die Äquivalenz des STST mit dem 6MWT auch bei PatientInnen mit pulmonaler Hypertonie (PH) zu testen und zu prüfen ob der STST künftig – aufgrund seiner kürzeren Dauer und leichteren Durchführbarkeit – den 6MWT gegebenenfalls ersetzen könnte.

Methoden: Von insgesamt 100 geplanten StudienteilnehmerInnen haben wir bisher 18 in die klinische Studie aufgenommen, die allesamt im Zuge des Ambulanzbesuchs für die Studie rekrutiert wurden. Alle TeilnehmerInnen absolvierten beide Tests und es wurden direkt vor und nach, sowie drei Minuten nach den Tests Vitalparameter wie Blutdruck, Puls und periphere Sauerstoffsättigung erhoben. Weiters wurde sichergestellt, dass ein Abstand von mindestens 30 min zwischen beiden Tests eingehalten wird. Die ProbandInnen waren alle bereits diagnostiziert mit PH und älter als 18 Jahre. Nicht gehfähige PatientInnen wurden aus der Studie ausgeschlossen.

Resultate: 56 % der bisherigen StudienteilnehmerInnen waren Frauen und das mittlere Alter aller ProbandInnen betrug 67 ±10 Jahre. Erste Resultate zeigen, dass der STST bei PatientInnen mit PH eine gute Korrelation mit dem 6MWT zeigt (r=0,61, P=0.0075). Die mediane Wiederholungszahl lag beim STST bei 17,5 Wiederholungen/min (25.–75. Perzentile 14–22,5/min) und der Median der Gehstrecke lag bei 417,5 m (25.–75. Perzentile 341–493 m). Tendenziell hatten ältere PatientInnen

schlechtere Werte sowohl im STST (r=–0,464, P=0.0526), als auch im 6MWT (r=–0,451, P=0.0603). Bei der Selbsteinschätzung der Atemnot mittels Borg Dyspnoe Skala liegt der Median der Angaben bei allen zwei Tests bei fünf Punkten. Bei beiden Tests kam es zu einem vergleichbaren Herzfrequenzanstieg. Beim STST betrug dieser Anstieg im Mittel 17 ± 13 Schläge/min, beim 6MWT 20 ±17 Schläge/min. Weiters ließ sich ein Blutdruckanstieg bei beiden Tests feststellen, wobei auch hier weitestgehend ähnliche Ergebnisse festgestellt wurden: Systolisch kam es zu einem mittleren Anstieg um 16 mm Hg beim STST und um 7,9 mm Hg beim 6MWT; diastolisch betrug der Anstieg im Mittel 3,16 mm Hg beim STST und 2 mm Hg beim 6MWT.

Schlussfolgerungen: Die Ergebnisse unserer Studie lassen sich in Einklang mit Resultaten bisheriger Studien bringen, in denen ebenfalls gezeigt werden konnte, dass der STST eine wertvolle Alternative zum 6MWT darstellt.

PS 21/21-5

Effects of tafamidis on exercise capacity, cardiac function and myocardial amyloid deposition in patients with transthyretin amyloid cardiomyopathy

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Introduction: Transthyretin amyloid cardiomyopathy (ATTR-CA) is caused by deposition of amyloid fibrils in the myocardium. The deposition occurs when transthyretin (TTR) becomes unstable and misfolds. Tafamidis is a kinetic stabilizer of transthyretin that prevents tetramer dissociation and amyloidogenesis by wild-type and mutant TTR.

Methods: Thirty-eight patients with diagnosis of transthyretin amyloid cardiomyopathy from our national amyloidosis registry were treated with tafamidis (20 mg or 61 mg) for a period of six months. In our explorative analysis we aimed to evaluate the effects of tafamdis by changes from baseline of the serum *N*-ter-

		Tafamidis	No treatment	Difference	p-Value
Cardiac Biomarkers		n=38	n=38		
NT-proBNP, ng/L	Baseline, median	2871.0	2420.0	-1042.5	0.015
	CFB to 6 months, median	-591.5	+451.0		
Functional Status		n=36	n=22		
6MWT, m	Baseline, mean	381.64	393.45	+29.27	0.175
	CFB to 6 months, mean	+5.72	-23.55		
Echocardiogram		n=38	n=18		
LA, mm	Baseline, mean	63.90	63.41	-2.51	0.178
	CFB to 6 months, mean	+0.08	+2.59		
LV, mm	Baseline, mean	41.82	42.00	-1.03	0.733
	CFB to 6 months, mean	-1.56	-0.53		
IVS, mm	Baseline, mean	20.95	21.24	+0.41	0.866
	CFB to 6 months, mean	+0.82	+0.41		
Longitudinal strain, %	Baseline, mean	10.80	12.43	+0.80	0.893
	CFB to 6 months, mean	-0.23	-1.03		
MRI		n=25	n=10		
ECV, %	Baseline, mean	50.41	51.76	-2.20	0.180
	CFB to 6 months, mean	-1.79	+0.41		

CFB, Change from baseline; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; 6MWT, Six-minute walk test; LA, Left atrium; LV, Left ventricle; IVS, Interventricular septum; MRI, Magnetic resonance imaging; ECV, Extracellular volume.

Abb. 1 | PS 21/21-5 Change from Baseline

minal prohormone of brain natriuretic peptide (NT-proBNP) concentration, 6-minute walking distance, as well as cardiac structure and function, compared to untreated amyloidosis patients.

Results: The analysis showed a significant reduction in the serum NT-proBNP concentration in tafamidis-treated patients compared to an increase in untreated patients (median difference, –1042.5 pg/mL, $p = 0.015$). Tafamidis also improved the walking distance during the 6-minute walk test at month six, while reduction in untreated patients was observed (mean difference, +29.27 m, $p = 0.175$). Echocardiographic findings revealed a decrease in left ventricular size (mean, –1.56 mm) as well as improvements regarding the left atrial size (mean difference, –2.51 mm) and the global longitudinal strain (GLS) (mean difference, +0.80 %) in tafamidis treated compared to untreated patients. T1 mapping in cardiac MRI showed a decrease in extracellular volume (ECV) (mean, –1.79 %) in patients receiving tafamidis, while an increase in ECV in untreated patients was observed (mean, +0.41 %). Due to insufficient power, the imaging parameters did not differ significantly between tafamidis treated and untreated patients.

Conclusion: Treatment with tafamidis for a period of six months in patients with transthyretin amyloid cardiomyopathy results in a significant improvement in NT-proBNP levels and may have positive effects on exercise capacity, cardiac function and myocardial amyloid deposition compared to untreated amyloidosis patients.

PS 21/21-6

Lowering of mean pulmonary arterial pressure is a prognostic marker in pulmonary hypertension patients treated with subcutaneous Treprostinil

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Introduction: Treprostinil (TRE), a prostacyclin analog, is effective for the treatment of pulmonary arterial hypertension and chronic thromboembolic pulmonary hypertension (CTEPH). We hypothesized that change of hemodynamics is of prognostic value. In our prospective registry we evaluated effects of first-line subcutaneous (sc) TRE in patients with severe pulmonary hypertension (PH) and analyzed the prognostic value of hemodynamic changes from baseline on long-term follow-up.

Methods: Data was collected from patients with pre-capillary PH in WHO functional class III or IV, mean right atrial pressure of ≥ 10 mm Hg, and/or cardiac index ≤ 2.2 liters/min/m2. Patients received first-line scTRE. Dose adjustments were performed individually according to clinical symptoms and side effects.

Results: Between 1999 and 2018 138 patients were included into the study. Of these, 18 (13 %) patients underwent double lung transplantation, and 59 (42.8 %) died of any cause. Overall survival rates at 1, 5, 10, and 15 years were 91 %, 57 %, 31 % and 29 %. The strongest predictor of outcome was change in mPAP after one year of scTRE. Change in mPAP –18.4 ± 7.9 mm Hg ($P = 0.012$) was associated with the best subsequent survival of 12.7 ± 1.5 years.

Conclusion: The data suggest that patients benefit from aggressive lowering of mPAP in the first year of treatment.

POSTERSITZUNG 22 – BASIC SCIENCE 3

PS 22/22-1

Sex-independent regeneration of neonatal mouse hearts following myocardial infarction

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Introduction: Myocardial regeneration remains a major challenge in cardiology. The recent establishment of mammalian models of cardiac regeneration offers new avenues to study regenerative strategies. To determine the sex-dependency in mouse neonatal cardiac regeneration we compared female and male mouse neonates upon experimental left anterior descending artery (LAD) ligation.

Methods: The sex was visually determined in neonatal mice and confirmed by PCR by amplification of the two copy Rbm31y Y-linked gene instead of amplification of the single copy Rbm31x X-linked gene. Following LAD ligation on postnatal day 1 (P1) we assessed cardiac function by echocardiography in a timecourse experiment at one day post injury (dpi), 7 dpi, 14 dpi, and 21 dpi. Finally, hearts were harvested to evaluate the size of infarction by Massons-Trichrome and H. E. staining. The initial area of infarction was determined by Tunel staining.

Results: No significant difference was found in the initial area of infarction between male and female mice 24 h after experimental LAD ligation. These comparable index ischemic injuries translated into completely recovered hearts irrespective of the sex. Male and female mice exhibited a conserved cardiac function and morphology 21 days after LAD ligation.

Conclusion: Our analysis revealed no marked differences in the process of neonatal cardiac regeneration between male and female mice and therefore proofs a sex-independent recovery at that age.

PS 22/22-2

CDR1as expression in the ischemic heart

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Introduction: Circular RNAs (circRNAs) are highly stable large non-coding RNAs that are produced by backsplicing. As circRNAs are widely expressed in different tissues under certain conditions, their expression and function in ischemic heart disease are of particular interest. Most circRNAs act as miRNA or protein sponges, by repressing their function on multiple tandem binding sites. Cerebellar degeneration-related protein 1 transcript (Cdr1as) is currently the most well-characterised circRNA and particularly expressed in brain tissue. Its predominant function in the heart is to act as a miR-7 sponge, thereby reducing cardiac fibrosis and dilation. Up-regulation of CDR1as
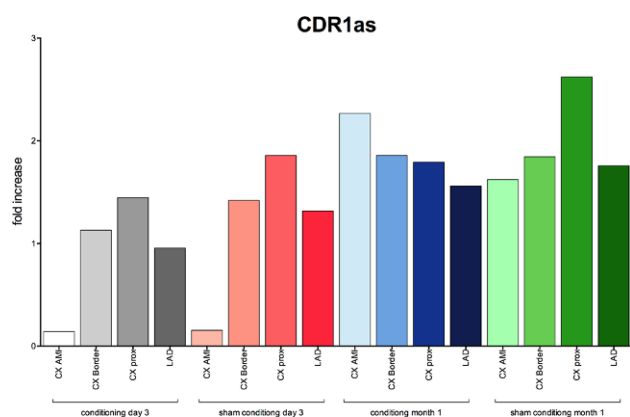
CDR1as

Abb. 1 | PS 22/22-2

has been proven to be pro-apoptotic in hypoxic murine cardiomyocytes. The aim of the study was to examine the role of ischemic preconditioning on CDR1as expression in a translational animal model of ischemic cardiomyopathy.

Methods: We performed cardiac remote intrinsic preconditioning by three times for 10 min in the mid left anterior descending coronary artery (LAD) by percutaneous balloon occlusion followed by each 10 min reperfusion in 14 pigs (group Preco), and sham procedure in 11 pigs (group control). One day later (2nd window of protection) all pigs underwent percutaneous reperfused acute myocardial infarction (AMI) by 90 min occlusion of the left circumflex coronary artery (LCX). Tissue samples from the LCX AMI, border and remote and the conditioning area (distal LAD) were collected on day 3 ($n = 5$ and $n = 5$, group Preco and Control, resp.) and month 1 after AMI ($n = 8$ and $n = 5$, group Preco and Control, resp.). CDR1as was assessed using qPCR in all myocardial regions. Cardiac MRI was conducted on day 3 and month 1 to assess infarct size and left ventricular function parameters.

Results: Scar size was not significantly reduced after one month (mean ± SD: day 3 vs. month 1: 17.61 ± 3.32 vs. 10.97 ± 2.39 %, $p = 0.184$). Left ventricular ejection fraction was also not significantly regulated throughout the follow up period. However, left ventricular end diastolic volume (LVEDV) and left ventricular end systolic volume (LVESV) were decreased on day 3 (LVEDV mean ± SD: day 3 vs. month 1: 80.78 ± 17.53 vs. 82.25 ± 25.47 ml, $p = 0.001$; LVESV mean ± SD: day 3 vs. month 1: 40.92 ± 9.98 vs. 44.73 ± 20.16 ml, $p = 0.014$). Except from scar size after one month (mean ± SD: 8.43 ± 3.74 vs. 13.65 ± 0.79 %, group Preco and Control, resp., $p = 0.038$) cMRI parameters did not differ between both groups. We observed a 7.09 times decreased expression of CDR1as in the LCX infarct zone on day 3 in the conditioning group ($p < 0.01$) and 6.46 times decreased expression in the sham conditioning group ($p < 0.0001$). In contrast on month 1 CDR1as expression was 2.27 times increased in the conditioning group and 1.62 times increased in the sham conditioning group in the LCX infarct zone. CDR1as expression was significantly up-regulated in both groups on month 1 compared to day 3 ($p = 0.0008$ and $p = 0.0014$, group Preco and Control, resp.) No significant difference in CDR1as expression between the preconditioning and sham conditioning group was observed in all myocardium regions. In tissue samples obtained from the CX border, CX remote and distal LAD region CDR1as was not significantly regulated.

Conclusion: In contrast with murine ischemic heart, our in vivo translational model of AMI suggests a down-regulation of CDR1as in the infarct zone on day 3 and only minor up-regulation on month 1, whereas the expression in other areas of the heart was not significantly regulated. This might be explained by the fact that 1) we differentiated the infarct zone from other zones in the ischemic heart; 2) we applied ischemic preconditioning and 3) our model is an in vivo translational one.

Presence of cardiac progenitor cell markers and no Amyloid-beta chains in cardiac samples of pediatric patients

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Introduction: Background Due to significant advances in pediatric cardiac surgery, more pediatric patients undergo surgery and are surviving into adolescence and adulthood. Several molecular markers have been tested and their presence has been proven in adult failing hearts, but no such data exist in the hearts of pediatric patients of various ages and comorbidities. These markers include antigens related to hematopoietic and cardiac progenitor cells or cardiac amyloid. For example, patients with Down Syndrome suffer from an accumulation of Amyloid-beta in the brain leading to an early-onset Alzheimer's disease, but no investigation has been carried out in the hearts of these patients Hypothesis We hypothesized that beta-Amyloid chains would be present in the cardiac tissue of pediatric patients with congenital heart disease, especially in Down Syndrome. We further hypothesized that we could detect cardiac and hematopoietic progenitor cell markers in the histological samples and correlate with the the underlying pathology and the age of the pediatric patients.

Methods: We performed immunohistology and immunofluorescence protocols on 20 paraffin-embedded slices of cardiac tissue from various regions of the heart of 18 patients under-
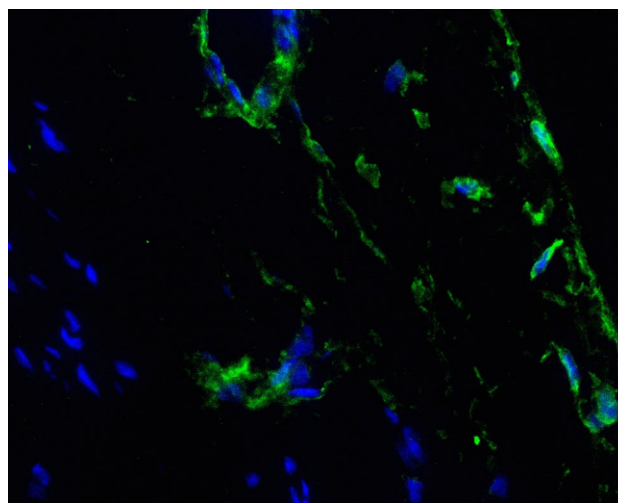
Abb. 1 | PS 22/22-3 Islet-1 in the cardiac tissue of pediatric patients
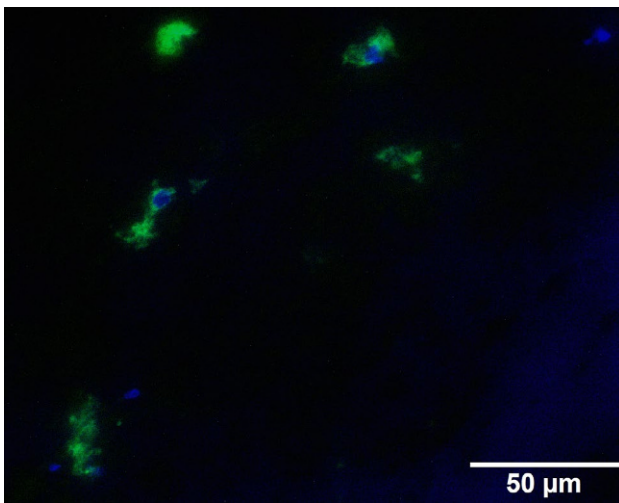
Abb. 2 | PS 22/22-3 Sca-2 in the cardiac tissue of pediatric patients

going routine heart surgery. The patient collective consisted of dilatative cardiomyopathy ($n=4$) and hypoplastic left heart syndrome ($n=4$), left ventricular hypertrophy due to aortic stenosis ($n=3$), right ventricular hypertrophy ($n=3$) due to Tetralogy of Fallot, atrial/ventricular septum defects with Down syndrome ($n=2$) and three patients ($n=3$) with other conditions. We used an Amyloid-beta antibody by Abcam for immunohistochemical and immunofluorescence staining and an Islet-1 Antibody by Biorbyt and Sca-1 (7HCLC) recombinant polyclonal antibody by Invitrogen for immunofluorescence staining.

Results: Neither immunofluorescence no immunohistochemistry could find presence of Amyloid-beta in the myocardial samples, also not in patients with Down syndrome. The cardiac progenitor cell marker Islet-1 was detected in a total of 10 samples from 9 patients. Sca-1 was detected in 5 samples from 4 patients. Three of these patients also showed the presence of Islet-1 (Figure). The oldest patient where the presence of cardiac progenitor cells was detected was aged 7 years and 5 months. Both the highest number of Islet-1 positive cells and Sca-2 positive cells were detected in two patients under 1 years of age with DCMP.

Conclusion: Although the sample size was limited, cardiac progenitor cell markers could be found in in half of the pediatric ventricular samples, suggesting the presence of a regenerative capacity of the pediatric population with congenital heart disease. The presence of Amyloid-beta and possibly Amyloid alpha in pediatric patients needs to be further explored.

PS 22/22-4

Progesterone promotes coronary artery dissection

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Introduction: Spontaneous coronary artery dissection (SCAD) is a common cause of myocardial infarction (MI) in young women. Pathologic remodelling of the coronary extracellular matrix (ECM) causes intimal tear and bleeding resulting in coronary occlusion. Underlying molecular mechanisms of SCAD are poorly understood. Mainly females are affected, with an incidence peak after pregnancy. Progesterone triggers ECM remodelling in the uterus enabling embryonal implantation. We therefore hypothesized that progesterone mediates SCAD development via ECM remodelling.

Methods: Spontaneous coronary artery dissection (SCAD) is a common cause of myocardial infarction (MI) in young women. Pathologic remodelling of the coronary extracellular matrix (ECM) causes intimal tear and bleeding resulting in coronary occlusion. Underlying molecular mechanisms of SCAD are poorly understood. Mainly females are affected, with an incidence peak after pregnancy. Progesterone triggers ECM remodelling in the uterus enabling embryonal implantation. We therefore hypothesized that progesterone mediates SCAD development via ECM remodelling. Human aortic smooth muscle cells (SMC) and endothelial cells (ECs) were treated with progesterone and analysed for ECM gene expression. To further elucidate the effects of progesterone on a single receptor, we performed lentiviral transfection of SMCs for stable upregulation of progesterone receptor (PR). Subsequently, we analysed ECM proteins. To identify the transcription factor responsible for ECM changes, we performed next-generation sequencing after lentiviral PR transfection and progesterone stimulation. To confirm our hypothesis in vivo, we performed unilateral carotid artery clamping with adenoviral injection of PR. Arteries were compared with the contralateral arteries using Doppler sonography and histological analyses.

Results: Progesterone regulates the transcription of a number of ECM-modulating genes including Fibrillin, MMP-9, ADAMTS5, Col4a1, Col5a1 and TGFbeta known for their involvement in arterial dissections in SMCs. In endothelial cells, Progesterone treatment induced expression of genes associated with endothelial dysfunction including eNOS and Endothelin1. In vivo, progesterone transfection resulted in modulation of extracellular matrix proteins and thus, predisposition for arterial dissection.

Conclusion: Progesterone induces ECM remodelling and predisposes coronary arteries for dissection via Fibrillin, MMP9 and ADAMTS5. We thereby uncover a novel risk factor for the development of SCAD, furthering our understanding of disease development and potentially leading to novel therapeutic approaches and prevention strategies.

PS 22/22-5

Intravenous amiodarone exerts solvent-related cardiodepressant effects in atrial and ventricular human myocardium

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Introduction: Amiodarone (AM) is one of the most frequently used antiarrhythmic drugs in cardiac arrhythmias. For

patients with pre-existing structural heart disease, as well as in an emergency setting there often is no suitable alternative for AM, yet there are reports of acute hypotension after intravenous application. However, the exact mechanical properties as well as rate dependent contractile effects have not been characterized in detail before for this substance.

Methods: Isolated human atrial ($n = 25$) and (failing) ventricular ($n = 24$) trabeculae were stimulated in an organ bath under physiological conditions (1 Hz stimulation, 37 °C, 2.5 mmol/l Ca2+) and subjected to increasing concentrations of AM (0.01 to 1000 μM/l) compared to corresponding solvent controls. Force frequency relationship (FFR) was investigated at submaximal AM concentrations (100 μM/l) with stepwise stimulation rate increases from 1 Hz to 3 Hz.

Results: AM exerts a dose dependent negative inotropic effect which can be attributed mainly to its solvent, which is a mixture of polysorbat 80 and benzyl alcohol. These effects were observed in both atrial (26.14 ± 5.14 % vs. 18.50 ± 8.55 % force compared to baseline) as well as ventricular tissue (43.95 ± 10.23 % vs. 42.84 ± 5.98 % force compared to baseline). However, even in submaximal concentrations, AM did not cause any deterioration of the FFR. Similarly, diastolic function, which was assessed by measurement of diastolic tension compared to baseline and relaxation times RT50 % and RT90 %, was widely unaffected by AM.

Fig. 1 | PS 22/22-5

Abb. 2 | PS 22/22-5

Conclusion: Under physiological conditions the solvent underlies rapid esterase degradation, therefore these findings underpin clinical practice of AM application as slow infusion and to avoid bolus infusion of the substance. Using a less cardiodepressant solvent like it is described for example for the sulfoalkyl-ether cyclodextrine would be beneficial and ease its use in emergency situations. Besides, AM did not show a pronounced cardiodepressant effect on its own in these experiments, therefore the oral application that comes without the solvent should be hemodynamically safe.

PS 22/22-6

Neutrophil extracellular traps promote fibrous vascular occlusions in chronic thrombosis

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Introduction: In some instances, venous thrombi transform into fibrotic vascular obstructions leading to chronic deep vein thrombosis and/or chronic thromboembolic pulmonary hypertension (CTEPH). Infection has previously been associated with fibrotic thrombosis, however mechanisms remain unclear. Since neutrophils are the first line of defense against pathogens, role of neutrophil inflammation and neutrophil extracellular traps (NETs) in chronic thrombosis was investigated.

Methods: Mouse model of inferior vena cava ligation was used to study the transformation of acute to chronic thrombus. Two mouse models were studied: first with staphylococcal infection and second with fibroblast-specific transforming growth factor-β (TGF-β) overactivity (TBRIIΔk). Markers of neutrophil inflammation and NETs were measured in plasma and tissues of patients with CTEPH. RNA sequencing (RNA-seq) was employed to study the transcriptome of fibroblasts isolated from pairs of pulmonary artery adventitia and thrombus excised during pulmonary endarterectomy.

Results: Mice with infection presented with larger thrombi containing more neutrophils and NETs, but less resolution. This phenotype was reversed with DNase1 administration. In patients with CTEPH, neutrophil counts, markers of neutrophil activation, and ex vivo NET formation were increased. RNA-seq analysis revealed significant fold differences in 24 genes, with TGF-β being the central regulator. TBRIIΔk mice exhibited enhanced thrombus fibrosis and delayed resolution.

Conclusion: Our results uncover the role of neutrophil inflammation and NETs in enhancing TGF-β signaling which leads to fibrotic thrombus remodeling. Targeting thrombus NETs with DNases may serve as a new therapeutic concept to treat thrombosis and its sequelae.
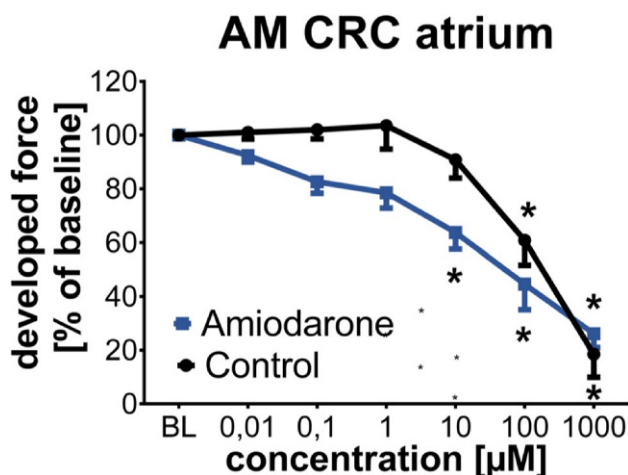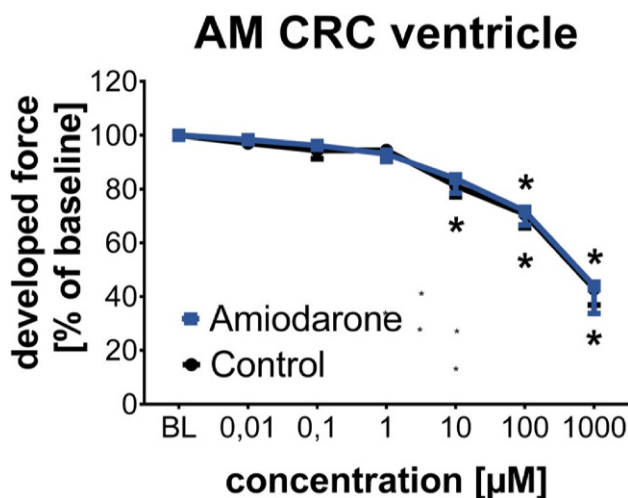
PS 22/22-7

Exploratory analysis of CDR1as/ciRS-7, miR-7 and miR-671-5p in congenital heart disease

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Introduction: The molecular mechanisms of pediatric congenital heart diseases are understudied due to a lack of viable human samples. Long noncoding RNAs (lnRNAs), such as FEN-DRR and Bvht are suggested to be involved in cardiac stem cell proliferation and differentiation. The aim of our study was to investigate the presence of the pro-apoptotic and antifibrotic circular RNA (circRNA) CDR1as/ciRS-7 and miR-671-5p which directly downregulates CDR1as/ciRS-7 in pediatric left ventricular myocardial samples (waste tissue samples during routine cardiac surgery).

Methods: Heart tissues removed during surgery of 10 pediatric patients with right ventricular heart failure of different ages were collected and immediately preserved in RNAlater (Invitrogen) and stored at -80 °C. RNA was isolated using RNEasy Mini Kit (Qiagen). Reverse transcription for miRNA and mRNA was accomplished using QIAGEN miScript RT kit and Quantitect RT kit, respectively. qPCR was performed using miScript SYBR® Green PCR Kit and Quantitect SYBR Green PCR Kit, respectively. For miRNA and mRNA, let-7a and Beta-Actin were used as housekeeping genes. To calculate expression levels, we used ΔCT instead of $\Delta\Delta CT$ since no control condition exists to calculate fold change.

Results: CDR1as was highly expressed in all age groups with a trend towards higher expression in the children with ages 100–200 days: 5–10 yrs ($1/\Delta CT$ 0.56 ± 0.18, $n=4$), 100–200 days ($1/\Delta CT$ 1.89 ± 0.71, $n=4$) and <50 days ($1/\Delta CT$ 0.94 ± 0.41, $n=2$). miR-671-5p was detected in all samples at much lower levels than CDR1as: 5–10 yrs ($1/\Delta CT$ 0.44 ± 0.15, $n=4$), 100–200 days ($1/\Delta CT$ 0.33 ± 0.09, $n=4$) and <50 days ($1/\Delta CT$ 0.32 ± 0.001, $n=2$). A non-significant negative correlation could be observed between miR-671-5p and CDR1as/ciRS-7 ($r=-0.33$, $p=0.34$). None of the investigated underlying diseases or syndromes (Trisomy 21 ($n=2$), Tetralogy of Fallot ($n=3$), Double Outlet Right ventricle ($n=2$), other ($n=3$)) showed significant differences in either CDR1as or miR-671-5p expression. The lncRNAs FEN-DRR and Bvht as well as miR-7 were not detected in sufficient quantities in our samples to enable statistical analysis.

Conclusion: This is the first time to show the high expression of circRNA CDR1as in human pediatric patients with congenital heart diseases, resulting in right heart failure. Future investigations of samples of our biobank will reveal time- and disease-dependent regulations of different circRNAs, or other lncRNAs, which might become potential targets of future gene therapies.
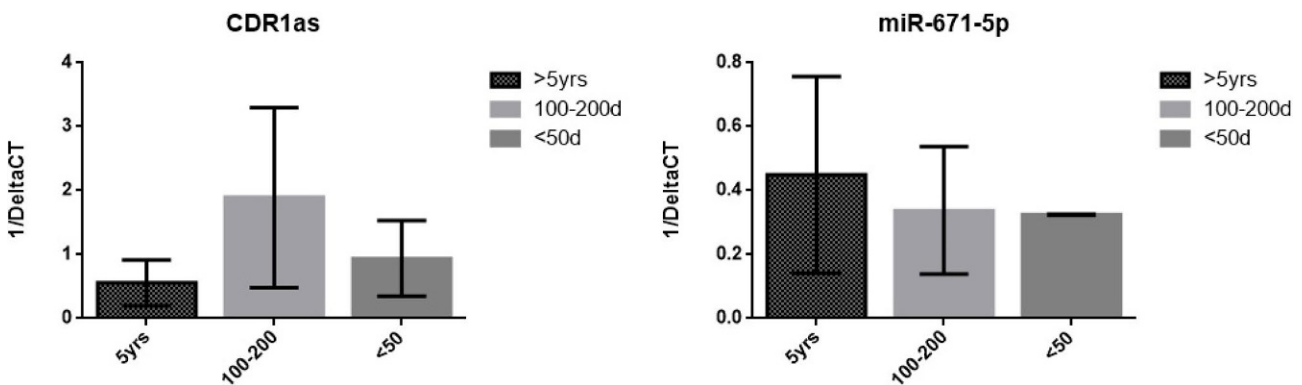
Fig. 1 | PS 22/22-7
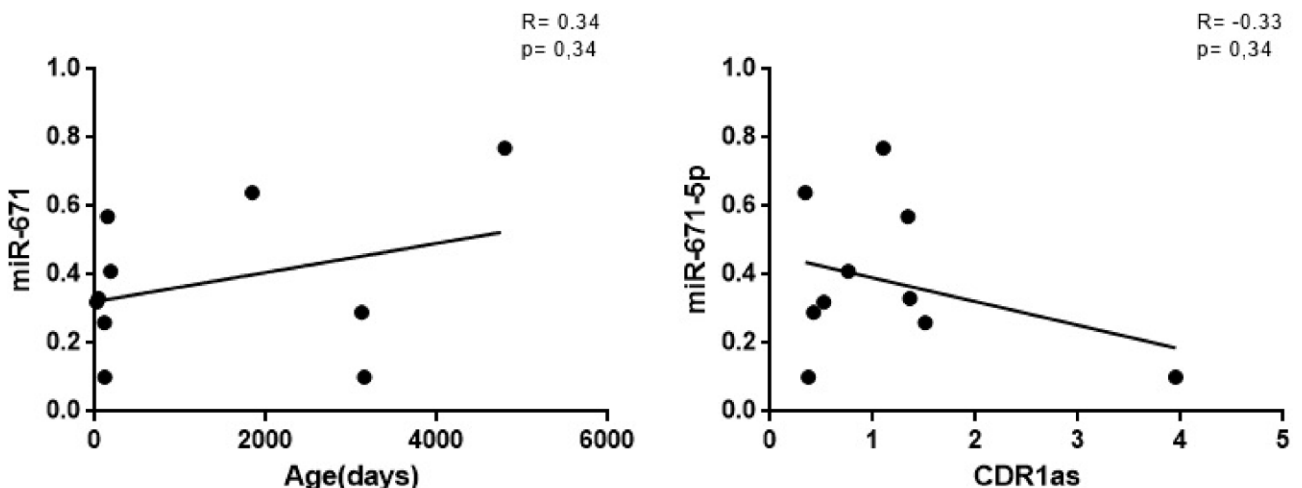
Fig. 2 | PS 22/22-7

PS 22/22-8

Importance of Tenascin-C in vascular remodeling following myocardial infarction in mice

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Introduction: Post myocardial infarction (MI) remodeling is known to be mainly driven by neurohormonal stimuli such as the activation of local and systemic renin-angiotensin-aldosterone system (RAAS). More recently, we demonstrated that the upregulation of the extracellular matrix protein Tenascin-C (TNC) following myocardial infarction promotes cardiac fibrosis and dilatation, suggesting its importance in adverse cardiac remodeling. The present study aims to further elucidate the impact of TNC on vascular (dys)function in a mouse model of MI.

Methods: MI was induced by permanent ligation of the left anterior descending coronary artery in TNC-KO and wildtype (WT) A/J mice. One or seven days after MI, animals were sacrificed and lung, aorta and serum were taken for further analyses. Vascular reactivity was assessed in isolated aortic segments using a wire myograph setup. Circulating levels of TNC were evaluated using an ELISA kit and ACE activity measurements were performed in lung lysates and serum.

Results: We found that the maximum responses to phenylephrine in aortic rings were significantly higher in infarcted TNC-KO mice compared to infarcted WT animals seven days post MI (TNC-KO MI: 10.64 ± 1.50 mN vs. WT MI 7d: 5.63 ± 1.70 mN; $p < 0.01$). Furthermore, we found that TNC serum levels were markedly increased one day and seven days post MI in comparison to sham-operated mice. Of note, we observed a trend of lower ACE activity in lung samples of TNC-KO animals compared to infarcted WT animals. In serum samples we could not see any differences in ACE activity between the groups.

Conclusion: In summary, circulating levels of TNC were increased even one day post-MI and associated with an increase of ACE activity in the lung. In addition, TNC KO induced preserved contractile function in aortic segments in response to adrenergic stimulation. Thus, these results support our hypothesis that TNC may regulate ACE and subsequent cardiac and vascular remodelling post MI, however further studies are warranted to clarify the underlying mechanisms.

PS 22/22-9

Isl-1+Sca-1 +c-kit+ porcine cardiac progenitor cell hypertrophy model: Endothelin-1 and Angiotensin II leads to gene expression changes of MiR-21, MiR-29a, GATA4 and MEF2c in vitro

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Introduction: Cardiac disease models and drug testing are frequently implemented in the porcine translational animal model. However, porcine cell culture models are rarely used to test major disease progresses and pathways. The heart comprises not only cardiomyocytes, endothelial cells and smooth muscle cells, but also cardiac progenitor cells (CPC) that are able to form new cardiac cells and possibly contribute to the regeneration cardiac tissue. In this study we investigated the

Fig. 1 | PS 22/22-9 I) characterization of pCPC. II) time course of hypertrophy induction in pCPCs. (Zlabinger et al.; Cells 2019, 8(11), 1416)

Fig. 2 | PS 22/22-9 gene expression of hypertrophy related genes. (Zlabinger et al.; Cells 2019, 8(11), 1416)

involvement of pCPC in hypertrophic cardiac disease modeling by evaluating disease associated gene and protein expression. MEF2c and GATA4 have shown to be increased in cardiac hypertrophy and are together with miR-21 and miR-29a involved in fibrosis related pathways.

Methods: Isl1+Sca1+cKit+ porcine CPCs (pCPCs) were isolated, characterized (Fig. 1, I) and in vitro hypertrophy was induced by stimulation with endothelin-1 (ET-1) and angiotensin II (Ang II). One treatment group also included a preceding transfection of a cardiac reprogramming plasmid aiming to induce differentiation into cardiac myocytes (Fig. 1, II). For the evaluation of hypertrophy induction, cell size measurements and gene expression analysis (MEF2c, GATA4, miR-21, miR-29a) as well as immunofluorescence staining for protein expression patterns (BNP, MCP-1, Cx43) were implemented.

Results: We could detect an increase in cell size in all hypertrophy treated groups compared to unstimulated pCPC. Gene expression of GATA4, MEF2c and miR-29a was significantly increased in Ang II treated pCPCs, accompanied by a higher protein expression of MCP-1. ET-1 stimulated pCPC showed an increased expression of GATA4, MEF2c and BNP compared to control cells. We could also detect increased levels of GATA4, MEF2c, miR-21 and miR-29a in differentiated pCPC in the Ang II treatment group and a higher expression of MEF2c, GATA4 and BNP in the ET-1 treatment group.

Conclusion: We could show that ET-1 and Ang II is able successfully induce hypertrophy in Isl1+Sca1+cKit+ pCPC with an increased cell size and upregulation of hypertrophy associated genes (Fig. 2) and proteins. Our model is suitable to study other hypertrophy and cardiac disease associated pathways and potential new biomarkers.

POSTERSITZUNG 23 – CHIRURGIE 2

PS 23/23-1

Reversal of pulmonary hypertension in pediatric patients with restrictive cardiomyopathy

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Introduction: Fixed pulmonary hypertension is a frequent complication of restrictive cardiomyopathy and a contraindication for cardiac transplantation due to an unacceptable risk of posttransplant failure of the unconditioned right ventricle. Continuous flow left ventricular assist devices have shown to reverse pulmonary hypertension in cardiac transplant candidates with heart failure with reduced ejection fraction. Reversal for pulmonary hypertension by left ventricular assist device implantation has so far not been reported in pediatric patients with restrictive cardiomyopathy. We report our experience with left ventricular assist device implantation in a bridge-to-candidacy approach in two pediatric patients with restrictive cardiomyopathy and fixed pulmonary hypertension.

Methods: A Medtronic HeartWare HVAD (Medtronic, Minneapolis, MN) was implanted as left ventricular assist device in two pediatric patients (female, 13 and 12 years of age; body weight 52.6 and 35.7 kg; body surface area 1.5 and 1.2 m2) with restrictive cardiomyopathy and fixed pulmonary hypertension (mPAP 40 and 95 mm Hg, PVR 2.4 and 18.6 WU), as bridge-to-candidacy for transplantation. Both patients, who were in NYHA class IV despite optimized medical therapy, underwent extensive vasodynamic testing prior to assist device implantation to test for reversibility of pulmonary hypertension. HVAD implantation was pursued through a median thoracotomy and cardiopulmonary bypass was installed in a standard fashion. To gain space for left ventricular assist device placement, an enhancement plastic of the left-sided pericardium was performed with a Gore-Tex patch. Due to the setting of restrictive cardiomyopathy after apical coring an additional myectomy was performed to remove partially obstructing parts of myocardium before inserting the inflow cannula in alignment with the mitral valve. The outflow graft anastomosis was performed to the ascending aorta in an end-to-side fashion. No additional valve procedures were necessary. The patients left the ICU after 6 and 11 days and were discharged 29 and 25 days respectively post ventricular assist device implantation. Medical treatment of pulmonary hypertension was pursued with bosentan and with bosentan and sildenafil respectively.

Results: During the time on device support, no ventricular assist device related complications occurred and readmission was not necessary. The patients were thoroughly monitored at our ventricular assist device outpatient clinic. Eligibility for heart transplant listing was achieved after 57 and 119 days of device support. A distinct decline of both mean pulmonary artery pressure and pulmonary vascular resistance values was examined in pre-transplant catheterization (mPAP 22 and 57 mm Hg; PVR 1.5 and 3.3 WU). Subsequent orthotopic heart transplantation was performed in both patients without any adverse events after 78 and 272 days of device support. Post-

transplant catheterization revealed sustained reversal of pulmonary hypertension (mPAP 18 and 20 mm Hg).

Conclusion: The described bridge-to-candidacy approach appears to be an effective treatment option to achieve transplant eligibility in a pediatric patient population with restrictive cardiomyopathy and fixed pulmonary hypertension. Prolonged left ventricular assist device support with a HeartWare HVAD lead to a decrease of both mean pulmonary artery pressure and pulmonary vascular resistance in both pediatric patients with terminal heart failure due to restrictive cardiomyopathy and allowed eligibility for transplant listing, as fixed pulmonary hypertension was reversed in a 12-week period of support. From a technical point of view, the implantation of a fully implantable device is possible in pediatric patients with restrictive pathologies, however requires additional extended myectomy to avoid later suction events. The quality of life is noticeably better than with paracorporal devices. No ventricular assist device related complications occurred and the subsequent heart transplantations were uneventful in both patients. Post-transplant vasodynamic testing revealed sustained reversal of PH.

PS 23/23-2

5 years later, an update on the comparison of porcine and pericardial bioprostheses after isolated aortic valve replacement

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Introduction: Long-term outcomes of aortic valve replacement may be determined by the choice of bioprosthesis. The clinical relevance of whether pericardial heart valves offer a favourable haemodynamic profile compared to porcine valves is still controversially debated. Our working group presented results of two commonly implanted bioprostheses at a single centre in a previous study. This is an update on findings 5 years later.

Methods: All consecutive patients undergoing isolated aortic valve replacement with either a Carpentier-Edwards Magna pericardial prosthesis or a Medtronic Mosaic porcine prosthesis between 2002 and 2008 were analysed in regard to preoperative characteristics, valve-related complications as well as short- and long-term survival.

Results: The Medtronic Mosaic was implanted in 162 patients and 292 patients received the Carpentier-Edwards Magna. The mean sizes of implanted valves were 22.5 ± 1.5 mm for the Mosaic and 21.8 ± 1.8 mm for the Magna ($P = 0.001$). The long-term survival rate was 75 and 46 % after 5 and 10 years for the Medtronic Mosaic, which was comparable with the Carpentier-Edwards Magna (76 and 48 %; $P = 0.444$). Valve-related adverse events were similar between groups in regard to endocarditis, strokes, bleedings and arrhythmias. Both prostheses were free from valve thrombosis. The need for postoperative pacemaker implantation and the presence of structural valve deterioration (SVD) was significantly increased in the Mosaic group ($P = 0.026$, $P = 0.032$). However, there is a tendency towards shorter ICU stay for the Mosaic group with 3 versus 5 days in average ($P = 0.052$).

Conclusion: Both types of aortic bioprostheses offer excellent results after isolated aortic valve replacement. Despite relevant differences in the presence of SVD and PM dependency, long-term survival was still comparable for both bioprostheses in this update five years later.

PS 23/23-3

Hemolysis due to stuck valve leaflet after transcatheter valve-in-valve mitral procedure

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Introduction: We present a case of an 81-year-old woman with a symptomatic severe mitral valve regurgitation combined with a grade II mitral valve stenosis due to a biological prosthesis degeneration–size 29 mm. The patient underwent a double valve replacement 16 years ago and the aortic bioprosthesis was competent.

Methods: Due to age and high operative risk the decision for transapical mitral valve-in-valve intervention was made. A 29 mm balloon-expandable bioprosthesis was implanted. After initial expansion, the valve showed an eccentric transvalvular leakage due to underexpansion of the anterior part of the valve probably due to the existence of the aortic bioprosthesis. A secondary balloon-inflation with +4 ml was performed. After that the patient left the operation theatre with a good functioning valve with minimal paravalvular leakage.

Results: The days after the patient showed up with anemia and needed repeated blood transfusions. She also suffered from acute renal impairment. In a control echocardiography the eccentric insufficiency-jet reappeared. This was associated to a stuck leaflet of the mitral prosthesis next to the bioprosthetic aortic valve. The patient underwent reoperation for sustained hemolysis. The underexpansion of the mitral valve associated with the presence of the aortic bioprosthesis was confirmed. The operation–a mitral valve replacement–was successful and the patient was discharged after six weeks of hospitalization.

Conclusion: Mitral valve-in-valve at the presence of an aortic valve prosthesis can potentially be associated with valve underexpansion which may lead to valve dysfunction.

PS 23/23-4

Lower hemisternotomy–an infrequently used but versatile approach to minimal invasive cardiac surgery

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Introduction: Lower hemisternotomy is an infrequently used approach to minimally invasive cardiac surgery. Aim of this study was the retrospective evaluation of the lower hemisternotomy approach in our institution.

Methods: Between June 2014 and September 2019 55 patients (30 male; median age 72 [63–75] years) underwent cardiac surgery via a lower hemisternotomy in our institution. Patient characteristics, procedures performed, peri- and postoperative complications as well as 30-day mortality were evaluated.

Results: Isolated mitral valve surgery with or without arrythmia correction surgery was performed in 11 patients, mitral and tricuspid valve surgery in 28 patients, tricuspid valve surgery in four patients, aortic valve replacement in five patients, two patients underwent triple valve surgery, two patients aortocoronary bypass surgery (CABG), one patient a tricuspid valve procedure with CABG, LAA occlusion and removal of a Lambl's Excrescence on the aortic valve, one patient underwent removal of an left sided atrial myxoma and one patient foreign body removal from the right ventricle. Median EuroScore II was 3.4 % [2.1–6.0]. Median cross-clamp time was 67 min [44–99]. Median procedural duration was 169 min [138–201]. Direct cannulation was used in 92.7 % ($n=51$) of patients. Major complications included aortic dissection during the index hospital stay in two patients, deep sternal wound infection in 1 patient, redo for bleeding in 1 patient. 30-day mortality was 3.6 % (2 patients).

Conclusion: In properly selected patients lower hemisternotomy provides excellent access to a broad variety of cardiac procedures with appealing cosmesis. Direct aortic and venous cannulation is feasible. Experienced aortic complications resulted from thereafter abolished cardioplegia delivery catheters. In our experience cardiopulmonary bypass as well as cross-clamp times were comparable to full sternotomy procedures.

PS 23/23-5

Anomalous origin of the right coronary artery–TAVI or no TAVI?

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Introduction: Transcatheter aortic valve implantation (TAVI) has become a routinely used therapy option in a selected patient population at high risk for conventional surgery. Different anatomically variants require a careful evaluation and planning of the procedure. Therefore, we are reporting a case study of a TAVI procedure with an anomalous origin of the right coronary artery.

Methods: An 84-year-old man diagnosed with a severe symptomatic aortic stenosis and worsening dyspnea during exertion was referred to our heart team. The preprocedural computed tomography revealed an anomalous origin of the right coronary artery from the left coronary sinus (ARCA). The proximal part of the RCA showed an intramural course between the aorta and the pulmonary truncus. It was decided that the amount of calcification in the left cusp was moderate and a compression of the intramural right coronary artery through the expansion of the transcatheter valve is unlikely.

Results: The heart team performed a transfemoral TAVI under general anesthesia. A balloon-expandable valve was implanted using the standard delivery system through the right femoral artery. The implantation was without any complications. Injection of contrast agent in the aortic root showed filling of both coronary arteries. Ten minutes after implantation, the

patient became hemodynamically instable. Transesophageal echocardiography revealed a decrease in right ventricular function. Electrocardiogram remained without any ST-elevation or depression. Nevertheless, the heart team decided to perform an aortocoronary bypass on the right coronary artery using a vena saphena magna graft. Weaning from cardiopulmonary bypass was uncomplicated and right ventricular function recovered quickly. After a short intensive care unit stay, the patient was transferred to the normal ward and could be discharged to the referring hospital on the 7th postoperative day. Unfortunately, the patient died suddenly on the 15th postoperative day. Postmortem examination revealed a serous pericardial effusion as the only pathological finding.

Conclusion: Anomalous origin of the right coronary artery with an intramural course is a challenging indication for TAVI. In our case we could observe severe ischemia after implantation and immediate intervention was necessary.

PS 23/23-6

Transfemoral aortic valve implantation with right-sided aortic arch–a case report

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Introduction: Transcatheter aortic valve implantation (TAVI) has become a routinely used therapy option in a selected patient population at high risk for conventional surgery. The configuration and anatomy of access ways differ between each patient. A rare anatomical variant is the right-sided aortic arch which aggravates transcatheter access to the aortic valve and results in a challenging implantation.

Methods: An 82-year-old man diagnosed with a severe symptomatic aortic stenosis and worsening dyspnea during exertion was referred to our heart team. Comorbidities included hypertension, coronary artery disease (without the need for intervention) and status post stroke. Pre-intervention magnetic resonance tomography revealed a right-sided aortic arch (Fig. 1).

Results: The heart team performed a transfemoral TAVI under local anesthesia with sedation. A balloon-expandable valve was implanted using the standard delivery system through the right femoral artery. Particular attention was paid to the positioning and manipulation of the extra-stiff guidewire and the delivery system within the aortic arch. We could achieve an aligned view of the coronary cusps, the ascending aorta and arch with LAO 10° and cranial 5° in fluoroscopy. The native valve could be crossed without difficulties and the valve was successfully implanted. The femoral access was closed by a percutaneous closure system. In-hospital stay was uneventful and the patient could be discharged home on the 6th postoperative day. Echocardiography showed a good prosthesis function with a mild aortic paravalvular leak.

Conclusion: Especially in anatomically challenging cases, careful preprocedural evaluation and planning are crucial for a successful transcatheter intervention.

Abb. 1 | PS 23/23-6 MRT Scan–right sided aortic arch

PS 23/23-7

Mid- and long-term results of the ATS mechanical prosthesis

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Introduction: Conventional mechanical bileaflet valves are designed with cavity pivots prone to areas of stasis and generate potential to thromboembolic formations. The ATS prosthesis is an open pivot valve which lacks recesses or cavities and thus avoids stasis. Furthermore, its design allows continuous passive washing via unimpeded blood flow unlike cavity pivots which rely on mechanical sweeping and high-velocity leakage jets.

Methods: 90 consecutive patients have undergone aortic valve replacement with the ATS mechanical prosthesis from first implementation until 2017 at the General Hospital of Vienna. Data on survival, complication rates and surgical specifics were acquired within a retrospective analysis using the Viennese documentation system and through a cross-sectional telephone follow up.

Results: 29 % of study participants were female ($N=26$), 71 % male ($N=64$) with a mean age of 51 +/- 11 years and a mean EuroScore II of 6.5 +/- 6.9 %. 64 % suffered from systemic blood pressure ($N=58$), 27 % from coronary heart disease ($N=24$), 12 % from diabetes ($N=11$), 14 % from chronic lung disease ($N=13$) and 17 % from chronic kidney disease ($N=15$). Virtually 63 % ($N=57$) had a preoperative normal left ventricular function. 14 % ($N=13$) needed urgent procedures. Aortic valve insufficiency was the main surgical indication in 42 % ($N=38$) followed by aortic stenosis. 24 % ($N=22$) underwent previous cardiac surgery. Concomitant procedures were performed in 67 % ($N=60$). Patients remained at the intensive care unit for 6 days in average and were hospitalized with a mean of 14 days. Over-all mortality was 16 % with actuarial survival estimates of 85.6 +/- 0.04 % at 5 years and 78.9 +/- 0.06 at 10 years. The most common valve-related complications were post-procedural arrhythmias concerning 30 % ($N=27$) of the study population. 8 % ($N=7$) needed pacemaker implantations in the long-run. Endocarditis occurred in 1 patient, 1 patient suffered from stroke and 1 patient experienced a transient ischemic attack. OAC-related bleedings were present in 2 patients. No valve thrombosis was observed.

Conclusion: We conclude that open pivot valves such as the ATS mechanical prosthesis offer acceptable long-term results. Patients may profit from its refined design with the lack of recesses and cavities which may prevent the development of thromboembolic formations.

POSTERSITZUNG 24 – RHYTHMOLOGIE 4

PS 24/24-1

Subcutaneous defibrillator implantation under regional anesthesia: low risk, low pain

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Introduction: Subcutaneous implantable cardioverter-defibrillator (S-ICD) became an established option in the prevention of sudden cardiac death for patients prone to develop ventricular arrhythmias. The majority of these patients are at a high risk for general anesthesia (GA) due to heart failure with reduced ejection fraction and associated severe comorbidities. However, the intra- and postprocedural pain management remains challenging since the ideal implantation site of the pulse generator is between two muscular layers of the well innervated left anterolateral chest wall and the lead placement

involves a periosteal tunneling along the left sternal edge. The ultrasound guided serratus anterior plane block (SAPB) with or without parasternal plane block was described in single case reports, mostly accompanied by GA though. In an interdisciplinary approach with cooperation of cardiology, anesthesiology and cardiac surgery we aimed to develop a feasible method of risk and pain reduction in a severely ill patient cohort.

Methods: We present a series of three cases who underwent elective S-ICD (EMBLEM MRI S-ICD, Boston Scientific, USA) implantation under an extended local and regional anesthesia including ultrasound-guided SAPB, bilateral parasternal plane blocks and sedation. All patients were male, mean age was 44.8 years (38–52). Indications included arrhythmogenic right ventricular cardiomyopathy, ischemic cardiomyopathy for primary prophylaxis and dilated cardiomyopathy; mean left ventricular ejection fraction was 28.7 % (15–51 %). All patients had an ASA (American Society of Anesthesiologists) score of 3. Intra- and postoperative pain assessment as well as analgesia requirements were collected.

Results: Intraoperatively none of the patients required GA. Due to the combination of short- and long-acting local anesthetic agents the first signs of discomfort were reported between 14–18 h postoperatively, localized mainly to the site of the generator pocket. Oral nonsteroidal anti-inflammatory drugs provided sufficient pain relief. Patients were fully mobilized 1–2 h post implantation and discharged on the following day, except for the first patient, who had a history of disabling stroke 1 month preprocedural and was transferred to a neurorehabilitation unit.

Conclusion: Implantation of S-ICD under ultrasound-guided regional anesthesia is feasible and enables rapid mobilization along with early discharge of patients with severe cardiomyopathies. A multidisciplinary approach is required for the periprocedural management to avoid unnecessary and risky general anesthesia or the need for opioid analgesia.

PS 24/24-2

Diagnoserate nach Implementierung einer strukturierten Abklärung von Patienten mit Synkopen

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Einleitung: Die Abklärung von Patienten mit Synkopen bleibt in bis zu 40 % ohne Diagnose (1). Die Implementierung einer strukturierten Abklärung von Patienten mit Synkopen führt zu einer Verbesserung der Diagnoserate. Ziel: Reevaluierung der Diagnoserate 18 Monate nach Implementierung eines strukturierten Synkopenpfades in der internistischen Notfallaufnahme eines Schwerpunktkrankenhauses.

Methoden: Wir verglichen die Diagnoserate von Patienten mit Synkope, die vor Implementierung einer strukturierten Abklärung (2016), unmittelbar danach (2017) und nach 18 Monate (2019) in unserer Notfallaufnahme vorstellig wurden.

Resultate: Es waren 130 vs. 65 vs. 89 Patienten mit der Diagnose Synkope vorstellig. Vor Implementierung einer strukturierten Abklärung kam es in 60 % der Fälle zu einer Diagnosefindung. Unmittelbar nach Implementierung eines Abklärungspfades erhöhte sich die Diagnoserate auf 80 % ($p = 0{,}013$). 18 Monate danach erhöhte sich die Rate auf 82 %.

Schlussfolgerungen: Eine strukturierte Abklärung von Patienten mit Synkope führt zu einer signifikanten Verbesserung der Diagnoserate. Der Erfolg bleibt auch nach 18 Monaten bestehen.
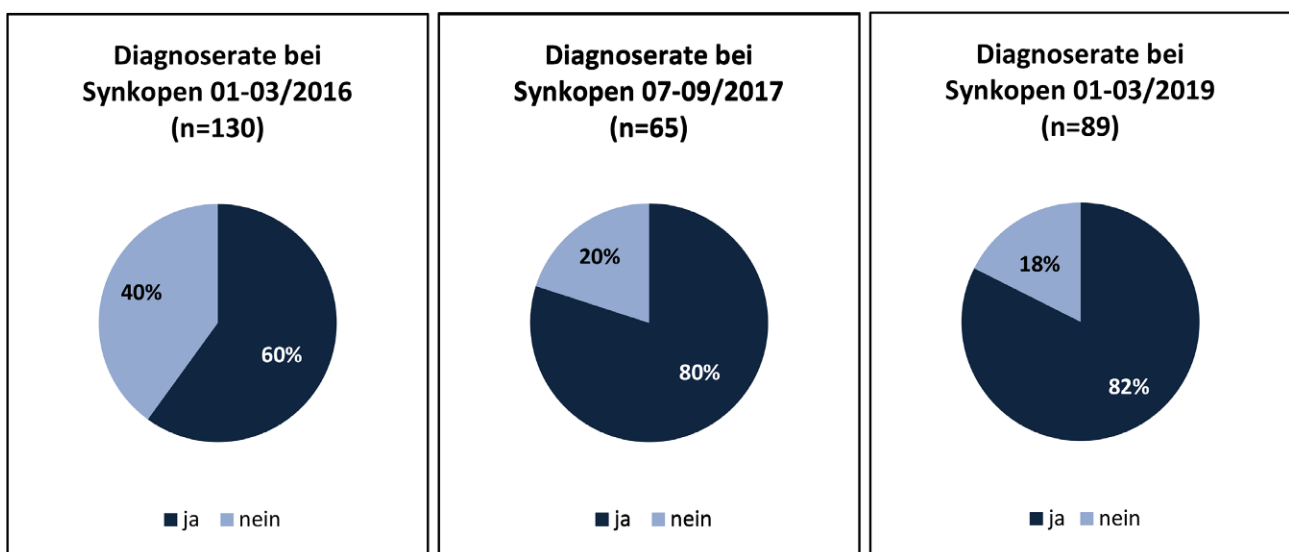
Abb. 1 | PS 24/24-2 Diagnoserate

PS 24/24-3

Altersverteilung unterschiedlicher Synkopenarten in einer internistischen Notfallaufnahme

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Einleitung: Die Art der Synkope ist je nach Altersgruppe sehr unterschiedlich. Sie hängt auch davon ab, in welchem klinischen Kontext die Evaluierung stattfindet.

Methoden: Im Rahmen der Implementierung einer strukturierten Synkopenabklärung führten wir eine Reevaluierung der Patienten durch, die in der internistischen Notfallaufnahme eines Schwerpunktkrankenhauses in der Zeit von Jänner bis März 2019 mit einer Synkope vorstellig wurden. Dabei untersuchten wir die Altersverteilung der unterschiedlichen Synkopenarten.

Resultate: Die häufigste Synkopenart, die orthostatische Synkope ($n=50$) wies einen Altersdurchschnitt von 63,8 a auf mit einer Häufung ab 50–59 a u. ab dem 70 a auf. Die Reflexsynkope ($n=16$) zeigte einen Altersdurchschnitt von 47,8 a. Sie trat in den jüngeren Altersgruppen (20–39 a) vermehrt auf. Die kardiale Synkope ($n=7$) hatte einen Altersdurchschnitt von 75,7 a. Sie trat nur in den Altersgruppen 60–89 a auf. Unklare Synkopen ($n=16$) waren im Schnitt 65,8 a alt. Es gab eine geringe Häufung in der Gruppe 20–29 a und ab dem 60 a.

Schlussfolgerungen: In unserem Kollektiv aus einer Notfallambulanz zeigte sich, dass kardiale Synkopen im höheren Lebensalter und Reflexsynkopen häufiger bei jungen Patienten auftreten. Orthostatische Synkopen traten tendentiell vermehrt bei Patienten im mittleren u. höherem Alter auf. Unklare Synkopen häuften sich im höheren Alter. Das ist durch die multifaktorielle Genese u. die zunehmende Multimorbidität erklärbar. Die erhobenen Daten decken sich mit internationalen Studien (1).

PS 24/24-4

Single-center Austrian experience of atrioventricular synchronous pacing with a leadless ventricular pacemaker following transcatheter aortic valve implantation

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Introduction: Accelerometer-based AV synchronous pacing by tracking atrial activity is feasible using a leadless ven-
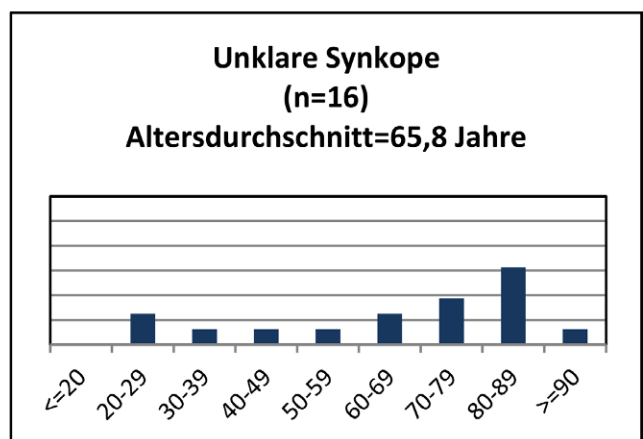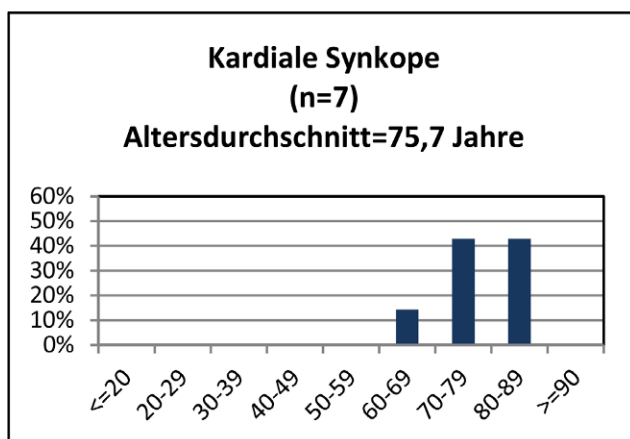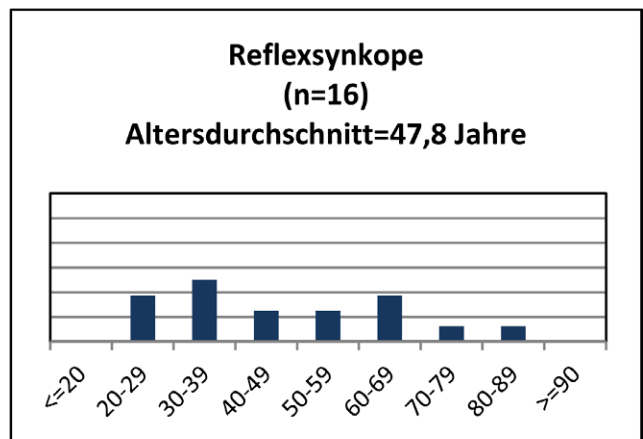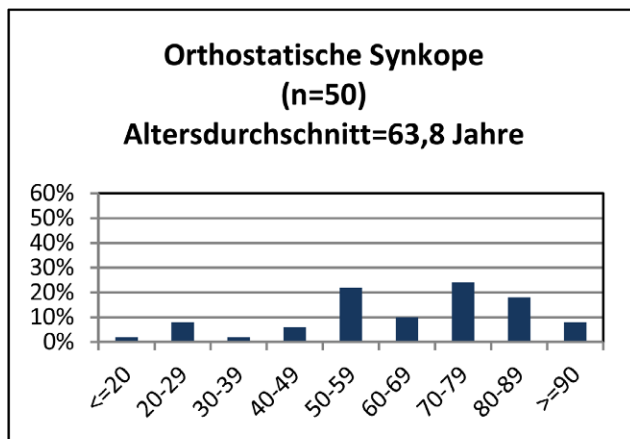
Abb. 1 | PS 24/24-3 Altersverteilung nach Synkopenart

Primary Efficacy Analysis Cohort (n=3)
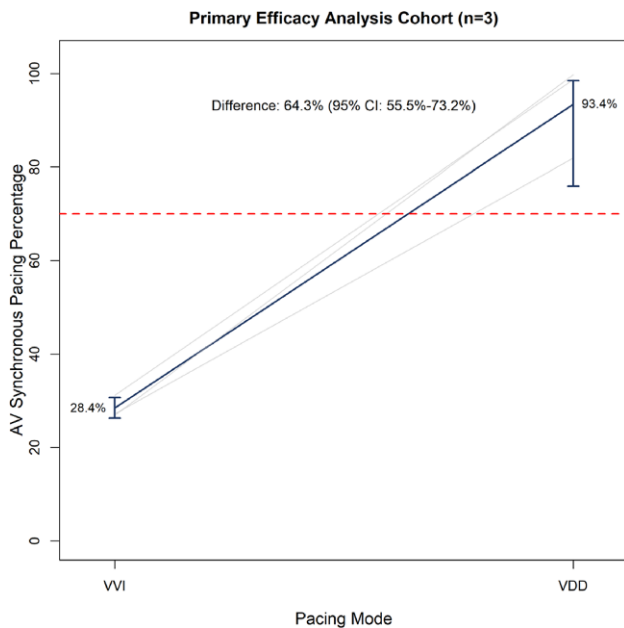
Fig. 1 | PS 24/24-4 AV Synchronous Pacing in Patients with TAVI and Complete AV Block

tricular pacemaker. Patients may be implanted with a leadless pacemaker for intermittent or permanent AV block (AVB) following transcatheter aortic valve implantation (TAVI). Objective of this investigation was to characterize AV synchronous algorithm performance in patients with prior TAVI at a single Austrian center.

Methods: MARVEL2 (Micra Atrial tRacking using a Ventricular accELerometer) was an acute study to assess the efficacy of atrial tracking with an algorithm downloaded into a Micra leadless pacemaker in patients with a history of AVB. AV synchrony was assessed during 20-minute resting periods in VVI and VDD mode. The algorithm switched from VDD to VVI-40 pacing during intact AV conduction.

Results: Four patients (2 male, mean age = 79.5 years) had a TAVI prior to AV synchronous pacing assessment. During study procedures, 3 patients had normal sinus rhythm with complete AVB, 1 had 1:1 AV conduction. Mean time since Micra implant was 10.3 months. Mean AV synchrony at rest in TAVI patients with complete AVB was 93.4 % (Figure). In the patient with 1:1 conduction, the mode-switching algorithm appropriately facilitated AV conduction. The patient with 1:1 had low ventricular pacing despite P–R > 300 ms.

Conclusion: The algorithm provided AV synchronous pacing in complete AVB patients with a prior TAVI and mode switching performed as intended. These limited results are consistent with those previously reported for broader MARVEL 2 study cohort.

PS 24/24-5

Fallbericht: „Fake-News" in der Notfallmedizin

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Einleitung: Speziell im Bereich der Notfallmedizin sind Kommunikation und Dokumentation unerlässlich, wenn es um

eine umfassende Anamnese-Erhebung geht, welche beispielsweise die weiteren Therapie-Schritte bestimmen kann. Da es hier oft mehrere ineinander greifende Glieder gibt, welche die Rettungskette ausmachen, kann auf diesem Wege Information mitunter verloren gehen, aber auch verfälscht werden, was in weiterer Folge Therapie-Schritte verzögern kann, wenn diese Fehl-Informationen spät korrigiert werden. Es kann aber auch passieren, dass fehlerhafte Informationen zu spät oder gar nicht korrigiert werden, was im schlimmsten Fall falsche Behandlungsschritte bedeuten kann. Es handelt sich hierbei konkret um den Fall eines 55-jährigen männlichen Patienten, welcher per Notarzthubschrauber status post Reanimation an die kardiologische Erstversorgung des Universitätsklinikums St. Pölten transferiert worden war.

Methoden: Laut notärztlichem Protokoll erlitt der Patient beim Hausarzt (welchen er wegen generellem Unwohlsein aufgesucht hatte) einen Atem-Kreislauf-Stillstand, wobei umgehend eine Reanimation begonnen wurde, so dass bei Eintreffen des Notarzt-Teams ein stabiler Sinusrhythmus festgestellt werden konnte. In weiterer Folge konnte der Patient in kardio-respiratorisch stabilem Zustand an unsere Erstversorgung transferiert werden. Gemäß der Aussage des Notarztes war im Zuge des BLS durch den Hausarzt keine Defibrillation möglich, weswegen als Grundpathologie eine PEA auf Basis eines cerebralen Geschehens vermutet wurde; weiters war es – laut Protokoll – in der Hausarzt-Praxis zu keiner Adrenalin-Gabe gekommen. Nach Eintreffen an unserer Erstversorgung wurde eine Computertomographie (CCT, CT-Angiogr., CT-Thorax) durchgeführt, um etwaige Grundpathologien zu diagnostizieren; hierbei ließen sich jedoch keine Auslöser für den Herz-Kreislaufstillstand feststellen. In den folgenden Tagen konnte der Patient von der Intensivstation an die Normalstation mit telemetrischer Überwachung verlegt werden. Da auch in zusätzlichen diagnostischen Verfahren (Röntgen, Echokardiographie, Holter-EKG) keine konklusiven Ergebnisse ersichtlich waren, wurde eine Coronarangiographie durchgeführt. Diese zeigte zwar Wandunregelmäßigkeiten, jedoch keine fassbaren signifikanten Stenosen. In weiterer Folge wurde die Implantation eines Loop-Recorders für den Patienten vereinbart.

Resultate: Vor der Implantation hielt eine behandelnde Oberärztin noch einmal Rücksprache mit dem Hausarzt, welcher die unmittelbare Reanimation durchgeführt hatte. Dieser schilderte die Ereignisse um die Reanimation allerdings anders, als es aus dem notärztlichen Protokoll zu entnehmen war: Der Patient kollabiert beobachtet im Wartezimmer der Arztpraxis und reagiert nicht auf Ansprache oder taktile Reize. Es wird eine Reanimation durch den Arzt und dessen Gattin begonnen, inkl. Maskenbeatmung und Sauerstoff-Insufflation. Ein hinzugebrachter halbautomatischer Defibrillator (Modell Responder AED) misst einen schockbaren Rhythmus, daher wird unmittelbar ein Schock abgegeben, nach welchem ein kräftiger Carotis-Puls palpabel ist. Beim Eintreffen des Rettungsteams wird im EKG eine Sinustachykardie mit einer Frequenz von 150/min gemessen, wobei das Rettungsteam inkl. Notarzt keinen Puls tasten kann. Die Reanimation wird daher unter der Arbeitshypothese „PEA" fortgesetzt, bis erneut ein Puls getastet werden kann. Der nun intubierte und stabilisierte Patient wird in der Praxis echokardiographiert, wobei sich eine gute LVEF ohne regionale Wandbewegungsstörungen zeigt. Während des Abtransports kommt es zu keinen weiteren Vorkommnissen.

Schlussfolgerungen: Aufgrund der geänderten Fallsituation, wurde auf die Implantation eines Loop-Recorders verzichtet. Stattdessen wurde dem Patient ein subkutaner ICD (A219 Emblem MRI mit einer Sonde) implantiert, mit unauffälligem Kontroll-Befund. In weiterer Folge konnte der Patient zwei Tage nach Implantation in kardio-respiratorisch stabilem Zustand nach Hause entlassen werden. Eine Woche nach Entlassung

Abb. 1 | PS 24/24-5 Aufnahme-EKG 1

Abb. 2 | PS 24/24-5 Aufnahme-EKG 2

Introduction: For PVI complete ablation lines around the ipsilateral pulmonary veins (PVs) are required. Since there is a highly complex and multilayered arrangement of myocardial fibers around the orifice of the PVs it is questionable if a lasso-catheter in the PV can verify the ablation line outside the PVs. The area between the lasso-catheter positioned within the PV and the ipsilateral circumferential ablation line outside the PVs was examined with high density mapping (HDM) in order to uncover residual electrical activity.

Methods: Patients scheduled for the first PVI underwent a circumferential radiofrequency ablation of the ipsilateral pulmonary veins. The PVI was verified 1) by an exit- and entryblock via all 20 electrodes of the Lasso catheter, 2) a negative adenosine test and 3) the unexcitability along the ablation line. After successfully completing all 3 tests an additional HDM was performed.

Results: We included 50 consecutive patients (62 ± 10 yrs, 36 males), 41 patients had paroxysmal or persistent AF, 9 patients longlasting persistent AF. Lasso-controlled PVI was achieved in all patients, verified by all 3 tests. Additional HDM with 6073 ± 1873 EGMs revealed residual electrical activity within the ablation line in 32 patients (64 %), in 18 patients (36 %) the line was complete in HDM. In 20/32 patients (63 %) residual electrical activity was found in one region and in further 12/32 patients (37 %) in several regions. The electrical activity was detected most frequently in the right carina (22 patients, 69 %). The PVs themselves, distal to the level of the position of the Lasso-catheter, appeared isolated in all 50 patients (100 %) in the HDM. During initial follow-up 7 patients had AF in the blanking period, 4 of them were patients with longlasting persistent AF.

Conclusion: HDM is able to uncover residual electrical activity in a hitherto blind spot of Lasso-controlled PVI. Therefore, an additional HDM of this area close to the ipsilateral ablation line should be considered.

PS 24/24-7

Komplikationen bei jungen ICD-Patienten – eine retrospektive Analyse

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Einleitung: Gewisse kardiologische Erkrankungen erfordern die Implantation eines Kardioverter-Defibrillators (ICD) bei Patienten unter 40 Jahren. Aufgrund ihres aktiven Lebensstiles könnte die Komplikationsrate bei jungen Patienten höher sein als in der restlichen Population.

Methoden: Die ICD-YOUNG Studie ist eine retrospektive Analyse von konsekutiven Patienten ≤40 Jahre, die zwischen Juli 2006 und Dezember 2017 in unserem Zentrum einen konventionellen ICD, einen subkutanen ICD (s-ICD) oder eine Sondenrevision erhielten. Alle Wiederaufnahmen aufgrund von Sondendefekten oder Batterieerschöpfung wurden dokumentiert.

Resultate: Von 586 Patienten, die einen ICD erhielten, waren 35 Patienten (6,0 %) ≤40 Jahre alt. Das mittlere Alter dieser jungen Patienten war 30,0 ±7,2 Jahre und 48,6 % waren weiblich. Die meisten Patienten (62,9 %) erhielten einen ICD als Sekundärprophylaxe und 11,4 % erhielten primär einen s-ICD. Der mediane Beobachtungszeitraum war 7,3 Jahre (Quartilen: 1,8, 12,0), mit einer kürzeren Beobachtung von s-ICD-Patienten im Vergleich zu Patienten mit konventionellem System (Median 2,9 vs. 9,0 Jahre). Während des Beobachtungszeitraums konn-

wurde eine telefonische Befragung beim Patienten durchgeführt, hierbei gab der Patient an, sich zwar müde und schwach zu fühlen, aber ansonsten keine Probleme hinsichtlich des für ihn neuen ICD-Systems zu haben. Die letztendliche Ursache für die Diskrepanz zwischen der Aussage des Hausarztes und des Notarztes hinsichtlich der Reanimation konnte schlussendlich nicht eruiert werden. Ungeachtet dessen zeigt dieser Fall recht anschaulich, dass selbst bei einer lückenloser Rettungskette mitunter massive Widersprüche in Fremdanamnesen entstehen, welche sich in weiterer Folge auf die therapeutischen Schritte auswirken können. Eine mögliche Vorbeugung hierfür wäre eine Erhebung der Personalien aller beteiligten (First-Responder, Zeugen, Rettungsteam) um im Bedarfsfall ein umfassenderes Bild der Anamnese zu erhalten und derartige „Fake-News" zu vermeiden.

PS 24/24-6

Residual electrical activity in the blind spot of lasso-guided antrum isolation detected by ultra-high-density mapping

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ten bei 37,1 % der Patienten anhaltende ventrikuläre Rhythmusstörungen erfolgreich durch den ICD therapiert werden. 19,5 % aller Patienten in der konventionellen ICD-Gruppe mussten sich aufgrund von Störungen der rechtsventrikulären Sonde einer Sondenrevision unterziehen, während in der s-ICD-Gruppe keine Sondenrevision durchgeführt werden musste. Die Zeit bis zum ersten Aggregatwechsel aufgrund von Batterieermüdung war ähnlich bei jungen und übrigen Patienten (Median 5,4 vs. 6,0 Jahre, $p = 0,23$).

Schlussfolgerungen: Junge Patienten, die einen ICD benötigen, haben eine hohe Rate an Sondendefekten. Für viele junge Patienten ist die s-ICD-Implantation eine neue Alternative zur konventionellen ICD-Implantation mit einer geringeren Rate an Sondendefekten.

POSTERSITZUNG 25 – AKUTES KORONARSYNDROM 2

PS 25/25-1

The age-specific impact of cellular immunity on long-term outcome after acute coronary syndrome

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Introduction: The identification of patients at risk for fatal cardiac adverse events after the acute phase of acute coronary syndrome (ACS) is still a challenging field in clinical practice. Especially the aging general population fosters a clear need for age specific prognostication after ACS which seems of major importance in terms of personalized risk stratification. The inflammatory burden during the acute phase of ACS proved to be directly associated with mortality is serval investigations–however its age-specific predictive potential remains unknown. Therefore, we aimed to elucidate the prognostic potential of cellular immunity during the acute phase of ACS on the patient outcome from a long-term perspective.

Methods: Patients presenting with ACS admitted between 12/1996 and 01/2010 were included within a clinical registry. Patient characteristics were elucidated at the time of hospitalization and blood samples including analysis for peripheral immune cell count were taken during the acute phase of ACS. Patients were followed prospectively until the primary study endpoint (= mortality) was reached.

Results: A total of 832 patients were founds eligible for study inclusion und were stratified in individuals <65 years ($n = 416$) and ≥65 years ($n = 416$). Both the total leucocyte count (<65 years: 10.6 G/l [IQR: 8.3–13.8] vs. ≥65 years: 9.5 G/l [IQR: 7.2–12.1]; r = –0.229, $p < 0.001$) and the fraction of lymphocytes (<65 years: 21.7 % [IQR: 16.2–27.3] vs. ≥65 years: 15.9 % [IQR: 11.4–22.3]; r = –0.429, $p < 0.001$) proved to be higher in individuals <65 years, while the fraction of neutrophil granulocytes (<65 years: 69.4 % [IQR: 52.3–76.2] vs. ≥65 years: 74.3 % [IQR: 66.5–80.3]; r = 0.184, $p < 0.001$) and monocytes (<65 years: 6.6 % [IQR: 5.4–8.3]) vs. ≥65 years: 7.5 % [IQR: 5.9–9.5]; r = 0.123, $p < 0.001$) was higher in elderly patients ≥65 years. After a median follow-up time of 8.6 years, a total of 516 (40.9 %) individuals died. We found that the total leukocyte count (adj. hazard ratio [HR] of 1.69 [95 %CI: 1.24–2.31; $p = 0.001$) as well the fraction of neutrophil granulocytes (adj. HR of 3.22 [95 %CI: 1.27–8.16; $p = 0.013$) showed a strong and independent association with mortality in

individuals ≥65 years. Moreover, while the fraction of lymphocytes (adj. HR of 0.52 [95 %CI: 0.41–0.68; $p = 0.001$) was strong inversely associated with fatal events, there was no predictive effect observed for the fraction of monocytes in the elderly population ($p = 0.790$). Of utmost interest, there was no prognostic effect observed in patients <65 years for any of the tested cell types.

Conclusion: While the total leucocyte count and the fraction of both neutrophil granulocytes and lymphocytes proved to be a strong and independent predictor for mortality after AMI in individuals ≥65 years, there was no prognostic effect observed in their younger counterparts. As routinely available values in clinical practice, they can be easily used to identify patients at risk for fatal events and contribute to proper secondary prevention after ACS from an age-specific perspective.

PS 25/25-2

Epinephrine treatment is associated with intestinal injury in patients with cardiac arrest

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Introduction: Besides early and good quality cardiopulmonary resuscitation (CPR) including timely defibrillation in patients with a shockable rhythm, current guidelines suggest the use of epinephrine in all patients with cardiac arrest. However, there is currently no evidence for increased survival in good neurological condition upon discharge, some observations even suggest possible harm due to impairment of microvascular flow. Whether epinephrine treatment might be associated with impaired intestinal flow with possible harmful consequences is currently not well understood. We therefore aimed to analyze the association between epinephrine treatment and intestinal injury evidenced by circulating intestinal fatty acid binding protein (iFABP) levels in patients undergoing CPR.

Methods: We have included all patients with sustained return of spontaneous circulation after cardiac arrest that were admitted to our intensive care unit. Blood was taken on admission, CPR-specifics were noted including medication and iFABP levels were analyzed using specific ELISA.

Results: We included 52 patients admitted to our medical ICU after cardiac arrest. Blood was taken on admission and levels of iFABP were analyzed. Patients were 64 years of age and predominantly male (76.9 %). During the 6-month follow up, 50 % of patients died and 38.5 % of patients had a CPC-score between 1 and 2 defined as favorable neurological outcome. iFABP levels were significantly lower in survivors (233.9 (89.5–399)) than in non-survivors (282.7 (85.9–11.500)) pg/mL ($p < 0.05$). There was no difference regarding iFABP levels and time to ROSC, while those patients receiving ≥3 mg of epinephrine showed a dramatic increase in iFABP levels (509.85 (64.9–15.000) pg/mL) versus those receiving <3 mg of epinephrine (262.45 (94.64–780.27) pg/mL). In addition, those receiving ≥3 mg of epinephrine had a 1.9-fold risk of dying ($p < 0.001$) as compared to thise receiving <3 mg.

Conclusion: Epinephrine treatment, especially in higher doses, was associated with a strong increase in circulating

markers of intestinal damage and mortality in this observational study. Further analyses are required to examine a potential intestinal injury of high epinephrine doses in patients undergoing CPR.

PS 25/25-3

Monocyte subsets predict mortality after cardiac arrest

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Introduction: After successful cardiopulmonary resuscitation (CPR) with return of spontaneous circulation (ROSC), many patients show signs of an overactive immune activation. Monocytes are a heterogenous cell population that can be distinguished into three subsets. The aim of this prospective, observational study was to analyze whether monocyte subset distribution is associated with mortality at 6 months in patients after cardiac arrest.

Methods: We included 53 patients admitted to our medical ICU after cardiac arrest. Blood was taken on admission and monocyte subset distribution was analyzed by flow cytometry and distinguished into classical monocytes (CD14++CD16–), intermediate monocytes (CD14++CD16+CCR2+) and non-classical monocytes (CD14+CD16++CCR2–).

Results: Median age was 64.5 (IQR 49.8–74.3) years and 75.5% of patients were male. Mortality at 6 months was 50.9% and survival with good neurological outcome was 37.7%. Of interest, monocyte subset distribution upon admission to the ICU did not differ according to survival. However, patients that died within 6 months showed a strong increase in the pro-inflammatory subset of intermediate monocytes (8.3% (3.8–14.6)% vs. 4.1% (1.5–8.2)%; $p=0.025$), and a decrease of classical monocytes (87.5% (79.9–89.0)% vs. 90.8% (85.9–92.7)%; $p=0.036$) 72h after admission. In addition, intermediate monocytes were predictive of outcome independent of initial rhythm and time to ROSC and correlated with the CPC-score at 6 months (R=0.32; $p=0.043$).

Conclusion: Monocyte subset distribution is associated with outcome in patients surviving a cardiac arrest. This suggests that activation of the innate immune system may play a significant role in patient outcome after cardiac arrest.

PS 25/25-4

Influence of myocardial damage on serum procalcitonin in ST-elevation myocardial infarction

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Introduction: Myocardial tissue injury due to acute ST-elevation myocardial infarction (STEMI) initiates an inflammatory response with a release of systemic inflammatory biomarkers including C-reactive protein (CRP) and white blood cell count (WBCc), which, however, hampers the usefulness of these routine biomarkers to identify concomitant infections. The clinical role of Procalcitonin (PCT), a promising marker of bacterial infections, to detect concomitant infections in acute STEMI is unknown, mainly because it is unclear whether myocardial injury per se induces a systemic PCT release. Therefore we aimed to investigate release kinetics of serum PCT in the acute setting of STEMI and possible associations with myocardial injury markers as comprehensively assessed by cardiac magnetic resonance (CMR) imaging.

Methods: In this prospective observational study, we included 141 STEMI patients treated with primary percutaneous coronary intervention (PCI). Concentrations of PCT, high-sensitivity CRP (hs-CRP), WBCc and high-sensitivity cardiac troponin T (hs-cTnT) were measured serially at day 1 and day 2 after infarction. CMR imaging to assess infarct size (IS), extent of microvascular injury (MVI) and occurrence of intramyocardial haemorrhage (IMH) was performed within the first week following STEMI.

Results: Median concentrations of PCT were 0.07 µg/l at both time points. In 140 patients (99%), both PCT values were within the normal range (≤0.5 µg/l). Whereas hs-CRP, WBCc, and hs-TnT were significantly correlated with CMR markers of myocardial damage, PCT did not show significant correlations (all $p>0.10$) with IS (PCT24h: r=0.07; PCT48h: r=0.13) or MVI (PCT24h: r=–0.03; PCT48h: r=0.09). Furthermore, PCT failed to discriminate between large and small IS or MVI or between presence and absence of IMH (AUC values:0.46–0.55).

Conclusion: In the acute phase after PCI for STEMI, circulating PCT remained unaffected by the extent of myocardial and microvascular tissue damage as visualized by CMR imaging. These data highlight the clinical potential of PCT to identify concomitant infections and to guide antibiotic treatments in STEMI patients.

PS 25/25-5

A follow-up study of clopidogrel, prasugrel, or ticagrelor use in patients with acute coronary syndrome after acute coronary syndrome in Austria from 2015 to 2017

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Introduction: The beneficial use of dual antiplatelet therapy (DAPT) with acetylsalicylic acid (ASA) and P2Y12 inhibitors has been established for patients after acute coronary syndrome (ACS). However, the optimal duration of DAPT is under debate. The aim of the present study was to investigate the long-term utilization and clinical outcome of clopidogrel, prasugrel, and ticagrelor in patients with ACS in Austria.

Methods: We analysed data from 13 Austrian health insurance funds for the years 2015 to 2017, on patients with a hospital discharge diagnosis of ACS. The primary end point was recurrence of ACS or death.

Results: From 49124 P2Y12 inhibitor naive patients with a hospital discharge diagnosis of ACS, 25.147 subjects filled a prescription of P2Y12 inhibitor within 30 days after the index event. 10.626 (42.9 %) subjects were identified with a prescription for clopidogrel, 4788 (19.3 %) for prasugrel, and 9383 (37.8 %) for ticagrelor, respectively. Occurrence of an endpoint was highest in the elderly patients. After adjustment for age, sex, and pre-existing medication as proxy for comorbidity the hazard ratio for ACS of 0.70 (95 % [CI: 0.61; 0.79]) was similar for prasugrel vs. clopidogrel and 0.70 (95 % [CI: 0.64; 0.77]) for ticagrelor vs. clopidogrel.

Conclusion: Ticagrelor was the most frequently prescribed P2Y12 inhibitor among patients below 70 years, and clopidogrel in those aged ≥75 years. Ticagrelor and prasugrel were associated with a lower number of deaths compared to clopidogrel. The risk of recurrence of ACS or death after an ACS is highest in patients ≥75 years.

PS 25/25-6

Comparison of enzymatic infarction size between patients with ST-segment elevation myocardial infarction versus patients with non-ST-segment elevation myocardial infarction and acute occlusion of the left circumflex artery

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Introduction: Aim of this study was to evaluate the enzymatic infarction size differences between ST-elevation myocardial infarction (STEMI) and non-ST-elevation myocardial infarction (NSTEMI) in patients with acute occlusion of the left circumflex artery (LCX), and the importance of the posterior leads (V7–V9) in electrocardiogram (ECG) in order to detect pretended NSTEMIs as STEMIs.

Methods: One hundred sixty-five patients with acute myocardial infarction with angiographically proven acute occlusion of the LCX as culprit lesion from 2003 until 2016 were included in a prospective registry. The patients were divided in two groups, those with STEMI and those with NSTEMI. We compared the excursion of markers for myocardial cell necrosis in these two groups. Considering Bonferroni-correction to counteract multiple comparison errors we assume significance at/under a p-value of $p = \alpha/6 = 0.05/3 = 0.0083$.

Results: Out of one hundred sixty five patients, who met our inclusion criteria, 88 had STEMIs and 77 had NSTEMIs. The enzymatic infarction size was significantly bigger in STEMIs regarding two biomarker peak value means (CK peak: 1633.1 U/l vs. 2956.6 U/l; $p = 0.001$) (Trop-I peak: 44.6 µg/l vs. 99.2 µg/L; $p = 0.001$) in favour of STEMI. CK-MB peak (213.8 U/l vs. 200.7 U/l; $p = 0.044$) was not significantly different between the two groups. Mean values at hospital admission were non-significantly higher in NSTEMI patients (CK: 645.6U/l vs. 630.0 U/l [$p = 0.9$], CK-MB: 108.6 vs. 101.6U/l [$p = 0.9$], Trop I: 22.4 µg/l vs. 5.2 µg/l [$p = 0.1$]).

Conclusion: At hospital admission, cellular necrosis appears to be approximately equal, which would support current guidelines of execution of additional ECG chest leads (V7–V9) to eventually detect NSTEMIs as hidden STEMIs of the posterior wall.

PS 25/25-7

Adherence to cardiac rehabilitation after acute coronary syndrome and its impact on patient outcome–a nationwide perspective

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Introduction: Secondary prevention after acute coronary syndrome (ACS) mirrors a key position in the reduction of morbidity and mortality in this highly vulnerable patient population. Especially cardiac rehabilitation proved to be one of the most beneficial therapeutic approaches for the reduction of
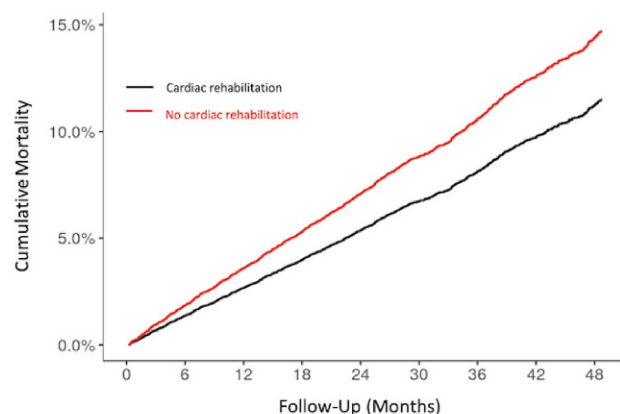
Fig. 1 | PS 25/25-7

re-events and overall modification of cardiovascular factors. However, profound epidemiological measures on adherence to a recommended cardiac rehabilitation after ACS remain scare, but seem of major importance in terms of preventing fatal cardiac adverse events. Therefore, we aimed to investigate adherence to cardiac rehabilitation after ACS and its impact on patient outcome from an Austrian nationwide perspective.

Methods: Within this population-based national observation all patients presenting with ACS between 04/2011 and 8/2015 in Austria were enrolled. Patient characteristics and co-morbidities were assessed via the Austrian national health insurance system and elucidated according to ICD10 definitions. Adherence to recommended cardiac rehabilitation was instigated according to health insurance documentation. Patients were followed prospectively until the primary study endpoint (=mortality) was reached. Cox Regression hazard analysis was used to investigate the impact of non-adherence to cardiac rehabilitation on patient outcome and was adjusted for a comprehensive subset of confounders within the multivariate model.

Results: During the observation period a total of 16.518 patients (median age: 64 years [54–74]; male: 68.4 % [$n=11.306$]) met the inclusion criteria. Of alarming importance 86.6 % ($n=14.305$) of all patients presenting with ACS did not perform any cardiac rehabilitation as recommended by current guidelines. During patient follow-up until 01/2018 a total of 1774 (10.7 %) individuals died. Adherence to recommended cardiac rehabilitation had a strong an independent inverse association with mortality with an adjusted hazard ratio of 0.73 (95 %CI: 0.54–0.98; $p=0.036$) (see Fig. 1).

Conclusion: The present nationwide investigation highlighted an overall low adherence to recommended cardiac rehabilitation after ACS. Since cardiac rehabilitation after ACS was associated with a 27 % risk reduction for fatal cardiovascular events during the observation period, awareness in terms of cardiac rehabilitation and associated intensified risk factor modification should be promoted, in order to prevent fatal atherothrombotic events.

PS 25/25-8

High sensitivity C-reactive protein is associated with worse infarct healing after revascularized ST-elevation myocardial infarction

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Introduction: The inflammatory response due to myocardial tissue injury in the setting of acute ST-elevation myocardial infarction (STEMI) is essential for proper local infarct healing. However, an excessive inflammatory response may aggravate myocardial damage and hampers infarct healing processes. The present study aimed to investigate the association of systemic inflammatory biomarkers with infarct size (IS) dynamics post-STEMI, using cardiac magnetic resonance (CMR) imaging.

Methods: This prospective observational study included 245 STEMI patients treated with primary percutaneous coronary intervention (pPCI). Peak values of high-sensitivity C-reactive protein (hs-CRP), white blood cell count (WBCc) and fibrinogen were determined serially until 96 h after pPCI. Infarct healing, defined as relative IS reduction from baseline to 4 months after STEMI, was assessed using late gadolinium enhanced CMR imaging.

Results: IS significantly decreased from 16 % of left ventricular mass (LVM) (Interquartile range [IQR]:8–24) at baseline to 10 % (IQR: 5–17) at 4 months ($p<0.001$). Relative IS reduction was 35 % (IQR: 8–50). Whereas peak WBCc ($p=0.926$) and peak fibrinogen ($p=0.161$) were not significantly associated with relative IS reduction, peak hs-CRP showed a significant association with IS reduction ($p=0.003$). In multivariable logistic regression analysis, the association between peak hs-CRP and relative IS reduction remained significant after adjustment for baseline IS, hypertension, hs-cardiac troponin T and N-terminal pro B-type natriuretic peptide (odds ratio:0.35 [95 % confidence interval:0.19–0.63]; $p=0.001$).

Conclusion: In STEMI patients treated with pPCI, hs-CRP was independently associated with 4 months IS reduction as determined by CMR, suggesting a pathophysiological interplay between inflammation and adverse infarct healing in survivors of acute STEMI.

POSTERSITZUNG 26 – HERZINSUFFIZIENZ 3

PS 26/26-1

ECG changes and their prognostic impact in patients with TakoTsubo syndrome

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Introduction: Takotsubo Syndrome (TTS) is defined by transient, circumferential regional wall motion abnormalities in the absence of a culprit coronary artery lesion. Within this study, we investigated ECG changes in patients diagnosed with TTS and report on the prognostic impact of the respective ECG patterns.

Methods: METHODS AND RESULTS: This single-center study included 101 consecutive patients diagnosed with TTS between 2006 and 2017 in the Wilhelminenhospital of Vienna. Upon 12-lead admission ECG, 59 patients had ST-segment elevation (58 %, STEMI), 9 patients had only ST-segment depression (9 %, NSTEMI) and 27 T-wave inversion (27 %, NSTEMI). Patients with a symptom-onset of less than 12 h often presented with ST-segment elevation (61.6 % vs. 40.0 %, $p=0.12$), while patients with a symptom-onset of more than 12 h typically presented with T-wave inversion (46.7 % vs. 23.3 %, $p=0.05$). T-wave inversion was also associated with significantly higher values of NT-pro-BNP ($p=0.024$), while high-sensitive cardiac troponin I (hs-CTnI) and creatine kinase (CK) were comparable between the groups. There was no difference in ECG changes among the different types of TTS (apical, midventricular, apical-midventricular and basal type). In a second step, patients with ST-segment elevation were divided into three groups based on the pattern of ST-segment elevation: Group A (concave type, $n=35$), group B (straight type, $n=8$) and group C (convex type, $n=10$), respectively. There was no difference in three-year all-cause mortality based on the type of ST-segment changes ($\chi2(2)=0.839$, $p=0.657$) or the pattern of ST-elevation ($\chi2(2)=0.501$, $p=0.778$).

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Upon 12-lead admission ECG, 59 patients had ST-segment elevation (58 %, STEMI), 9 patients had only ST-segment depression (9 %, NSTEMI) and 27 T-wave inversion (27 %, NSTEMI). Patients with a symptom-onset of less than 12 h often presented with ST-segment elevation (61.6 % vs. 40.0 %, $p = 0.12$), while patients with a symptom-onset of more than 12 h typically presented with T-wave inversion (46.7 % vs. 23.3 %, $p = 0.05$). T-wave inversion was also associated with significantly higher values of NT-pro-BNP ($p = 0.024$), while high-sensitive cardiac troponin I (hs-CTnI) and creatine kinase (CK) were comparable between the groups. There was no difference in ECG changes among the different types of TTS (apical, midventricular, apical-midventricular and basal type). In a second step, patients with ST-segment elevation were divided into three groups based on the pattern of ST-segment elevation: Group A (concave type, $n = 35$), group B (straight type, $n = 8$) and group C (convex type, $n = 10$), respectively. There was no difference in three-year all-cause mortality based on the type of ST-segment changes ($\chi 2(2) = 0.839$, $p = 0.657$) or the pattern of ST-elevation ($\chi 2(2) = 0.501$, $p = 0.778$).

Conclusion: The present study describes typical ECG changes in TTS and highlights their impact on cardiac biomarkers and clinical outcomes. There was no prognostic difference among the different repolarization abnormalities as assessed on admission ECG.

PS 26/26-2

Single and joint impact of type 2 diabetes and of congestive heart failure on albuminuria

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Introduction: Albuminuria is a characteristic feature of diabetic nephropathy, and urine albumin excretion is also increased in patients with congestive heart failure (CHF). However, no data are available on the single and joint associations of type 2 diabetes (T2 DM) and CHF with albuminuria. This issue therefore was addressed in the present study.

Methods: We investigated 181 patients with CHF, of whom 84 had T2 DM (CHF+/T2 DM+) and 97 did not have diabetes (CHF+/T2 DM–) and 413 controls without CHF, of whom 79 had T2 DM (CHF–/T2 DM+) and 334 did not have diabetes (CHF–/T2 DM–).

Results: The albumin/creatinine ratio (ACR) was 34 ± 117 in CHF–/T2 DM- patients. Compared to this group it was higher in CHF–/T2 DM+ patients (56 ± 110; $p = 0.002$), in CHF+/T2 DM- patients (98 ± 311; $p < 0.001$) and in CHF+/T2 DM+ patients (286 ± 779; $p < 0.001$), in whom in turn it was higher than in CHF–/T2 DM+ ($p < 0.001$) or in CHF+/T2 DM- ($p = 0.001$) patients. The ACR did not differ significantly between CHF–/T2 DM+ and CHF+/T2 DM- patients ($p = 0.345$). In multivariate analysis of covariance, T2 DM and CHF proved to be independent predictors of the ACR after adjustment for age, sex, body mass index, LDL-C, smoking and hypertension ($F = 6.392$; $p = 0.012$ and $F = 13.14$; $p = <0.001$, respectively).

Conclusion: We conclude that T2 DM and CHF are mutually independent determinants of albuminuria.

PS 26/26-3

Lebensqualität nach überstandener Riesenzellmyokarditis – Eine Single-Center-Erhebung

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Einleitung: Die Riesenzellmyokarditis – Giant Cell Myocarditis (GCM) – ist eine seltene und besonders schwere Form der Myokarditis, die typischerweise junge Menschen betrifft und eine hohe Mortalität aufweist[1]. Die Einführung neuer intensivmedizinischer Therapien wie zB extrakorporale Membranoxygenierung (ECMO) haben dazu geführt, dass PatientInnen bis zur Klärung der endgültigen Diagnose am Leben erhalten werden konnten und somit eine kausale Therapie (Immunsuppression) gestartet werden kann.

Methoden: Alle zwischen 2008 und 2019 biopsierten und mittels Histologie oder Genexpressions-Analyse auf Riesenzellen [2] diagnostizierten Patienten (3w, 4 m, mittleres Alter 39+/–10 Jahre) wurden erfasst. Die aktuelle Einschätzung der Lebensqualität, des Gesundheitszustandes, der Arbeitsfähigkeit, sowie sozio-demographische Daten der Überlebenden wurden mittels eines standardisierten Fragebogens (WHOQOL_100) erfasst. Anschließend wurden diese Patienten (2w, 3 m) noch telefonisch mittels eines nicht-standardisierten Interviewleitfadens (20 Fragen) über ihr derzeitiges Befinden und die Einhaltung der Therapieempfehlungen befragt.

Resultate: In 2 Patienten (2w) wurde die GCM histologisch, in 5 (1w, 4 m) mittels Genexpression diagnostiziert. Zwei Patienten (1w, 1 m, Alter 28 +/- 2 Jahre) mit kardiogenem Schock erhielten eine ECMO, 1 Patient (w) einen BIVAD. Alle Patienten wurden mit einer kombinierten immunsuppressiven Therapie (Cortison, Cyclosporin A) für mindestens 12 Monate behandelt, die erst nach negativer Kontrollbiopsie (keine Riesenzellen bzw. keine Genexpression für Riesenzellen) ausgeschlichen wurde. In keinem Fall kam es zu einem Rückfall. Bei Diagnosestellung wiesen die Patienten eine LV-EF von 23+/–13 % (10–45) auf, am Ende der immunsuppressiven Therapie 58+/– 10 % (45–70). Zwei Patienten sind verstorben (1w, 1 m), 2 Patienten (1w, 1 m) zeigen eine Defektheilung mit noch eingeschränkter LVEF. Der Rest weist eine Restitutio ad integrum auf (1w, 2 m). Von den Überlebenden waren zum Zeitpunkt der Befragung (12/2019) noch 2 Patienten (1w, 1 m) unter immunsuppressiver Therapie, während 3 Patienten (1w, 2 m) als geheilt galten. Mit Ausnahme eines Patienten (1 m) sind alle Überlebenden wieder in den Arbeitsprozess eingegliedert. Die Patienten zeigen innerhalb von 2 bis 3 Jahren post GCM eine hohe gesundheitsbezogene Lebensqualität mit wenig Einschränkung. Die Therapieempfehlungen zur Weiterbehandlung werden eingehalten. Aufgrund von Komorbiditäten geben 2 Patienten (1w, 1 m) noch Beeinträchtigungen ihres täglichen Lebens an.

Schlussfolgerungen: Durch rasche bioptische Diagnostik und Einsatz moderner Methoden wie ECMO und Immunsuppression hat die GCM eine geringere Mortalität als in der Literatur berichtet. Die wenigen Fälle zeigen großteils eine gute Prognose. In den ersten Jahren nach der Erkrankung zeigt sich schon wieder eine gute Lebensqualität.

PS 26/26-4

Neurohumoral memory in end stage heart failure patients undergoing LVAD implantation and heart transplantation

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Introduction: Neurohumoral dysregulation has been identified as a pathophysiologic hallmark of heart failure (HF), nevertheless conventional concepts do not provide a clear understanding on how neurohumoral activation is initiated and maintained. Contribution of the periphery, response to therapeutic interventions and extent of potential reversibility are unknown. Circulatory support by a left ventricular assist device (LVAD) and heart transplantation (HTx) restore the detrimental hemodynamic condition of cardiac failure instantly. This study aimed on assessing the neurohumoral response to LVAD and HTx.

Methods: Patients with end stage HF undergoing LVAD implantation or HTx were included from an interdisciplinary prospective registry of the cardiology and cardiac surgery departments established for the optimal management of advanced HF patients at the Medical University of Vienna. Venous blood samples were obtained immediately before the surgical intervention (i.e. LVAD or HTx) and after hemodynamic stabilization at approximately 6 months. Routine laboratory measurements including NT-proBNP and active renin concentration (ARC) were performed. Additionally the regulation of the Renin-Angiotensin-System (RAS) was analyzed by measuring all circulating RAS metabolites by mass spectrometry.

Results: A total of 39 patients were included into the study, 9 patients received an LVAD and 30 patients underwent HTx. Neurohumoral dysregulation improved in both groups, reflected by a significant decrease in circulating NT-proBNP and ARC for LVAD [NT-proBNP: 10187 (IQR 2265–12991) vs 1806 (IQR 1209–2426), $p = 0.004$ and ARC: 659 (IQR 40–1910) vs 145 (IQR 30–333), $p = 0.375$] and HTx patients [NT-proBNP: 4059 (IQR 1196–8814) vs 1244 (IQR 560–3145), $p < 0.001$ and ARC: 636 (IQR 103–2103) vs 94 (IQR 54–302), $p = 0.004$] (Fig. 1A). Notably, neither NT-proBNP nor ARC levels reached the normal range after the restoration of the hemodynamic condition. 0 % of patient showed normal NT-proBNP and only 33 % and 18 % patients normal ARC values after LVAD and HTx, respectively, indicative for continuing dysregulation in terms of a neurohumoral memory. Detailed RAS-patterns are displayed in Fig. 1B. Both groups showed a marked improvement in angiotensin patterns with significantly decreased total metabolite concentrations and AngII levels [LVAD 97 (IQR: 5–101) pmol/l vs 3 (IQR: 1–6) pmol/l, $p = 0.036$ and HTx 44 (IQR: 1–145) pmol/l vs 9 (IQR: 6–24) pmol/l, $p = 0.007$], which is the main effector peptide of RAS responsible for HF progression.

Conclusion: Neurohumoral dysregulation is significantly improved after LVAD implantation and HTx in end stage HF patients, however normal ranges of neurohormones were not achieved suggesting a neurohumoral memory. The evolution of neurohumoral memory and the underlying mechanisms for incomplete normalization warrant further investigation. Continuation of HF therapy in individuals showing high levels of circulating neurohumoral biomarkers despite LVAD or HTx seems reasonable.
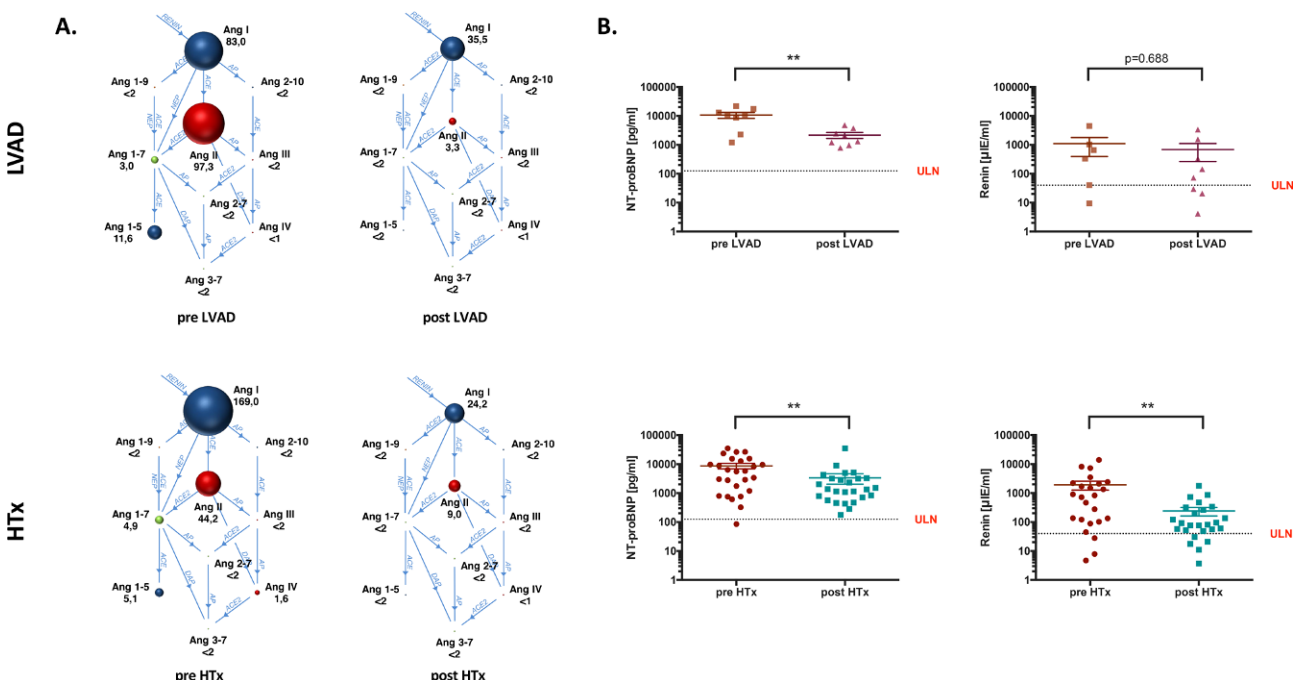
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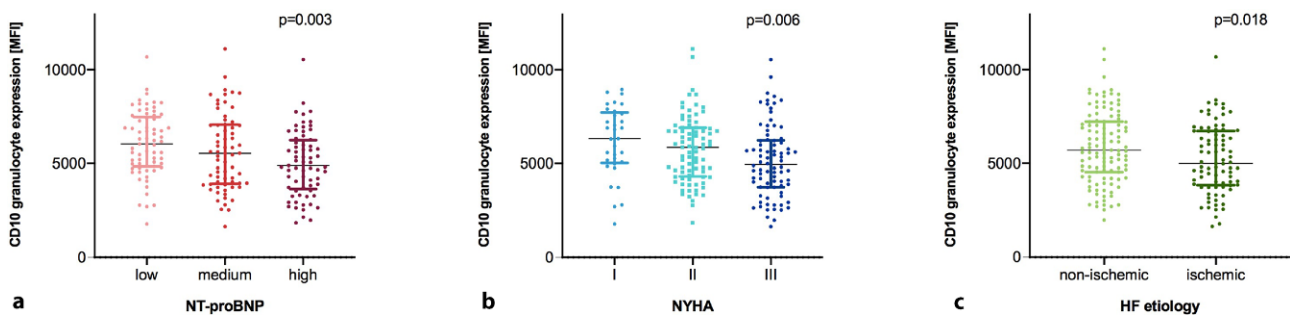
Fig. 1 | PS 26/26-5

PS 26/26-5

Neprilysin expression and activation status on neutrophils in chronic HFrEF patients

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Introduction: Inhibition of neprilysin by the angiotensin receptor-neprilysin inhibitor has shown remarkable success in the treatment of heart failure with reduced ejection fraction (HFrEF). However, the exact mechanism of action is still a matter of debate. Neprilysin (NEP) is a ubiquitous transmembrane endopeptidase markedly expressed on the cell surface of neutrophils, in this context also known as CD10. Originally, CD10 has been identified as a cell surface marker to discriminate mature from immature neutrophils. Other inflammatory neutrophil surface markers such as CD11b, CD64 and CD66b are commonly used to determine the activation status of neutrophils. In our previous explorative study, NEP expression on neutrophils correlated inversely with heart failure (HF) severity and mortality. This study aimed to explore the relationship between NEP expression and typical neutrophil activation markers and to confirm its relation to HF severity reflected by NT-proBNP, NYHA class and mortality in a bigger cohort of HFrEF patients.

Methods: We prospectively enrolled 208 consecutive patients with stable HFrEF. Mean fluorescence intensity (MFI) of CD10 expression on peripheral blood neutrophils was deter-

mined by flow cytometry of whole blood samples. For a subset of 117 patients the expression of CD11b, CD64 and CD66b was measured. EDTA anticoagulated blood (100 µl) was stained using a combination of six antibodies with fluorescence minus one sample as control [CD10 (#332777), CD45 (#560178), CD16 (#335035), CD11b (#555388), CD64 (#561191), CD66b (#562254); BD Biosciences, San Jose, CA, USA].

Results: Median age was 64 years (IQR 54–72), 57 (27 %) patients were female. Median NT-proBNP values were 1819 pg/ml (IQR 740–4264). Approximately half of the study population with 115 (55.3 %) patients had a non-ischemic etiology of HF. Median MFI of NEP (CD10) on neutrophils was 5542 (IQR 4168–6903). Interestingly, non-ischemic HFrEF was characterized by higher neutrophil NEP expression compared to ischemic HFrEF [5703 (IQR 4548–7235) vs 4994 (IQR 3844–6718), $p = 0.018$]. Neutrophil NEP expression correlated highly significant with CD11b expression ($r = 0.61$, $p < 0.001$) but not with CD64 and CD66b [$p =$ ns]. Neutrophil NEP expression decreased with HF severity reflected by NYHA stage ($p = 0.006$) and tertiles of NT-proBNP [$p = 0.003$] (Fig. 1). Thirty-two (15 %) patients died during a median follow up of 10 (IQR: 4–44) months. In the univariate analysis increased NEP expression on neutrophils was associated with better overall survival [HR per 1-IQR increase of MFI 0.49 (95 % CI: 0.28–0.85), $p = 0.011$]. This association remained significant after adjustment for age, kidney function and NT-proBNP [adj. HR per 1-IQR increase of MFI 0.55 (95 % CI: 0.31–0.97)]. Kaplan-Meier analysis demonstrates the impact of neutrophil NEP expression on outcome graphically (Fig. 2).

Conclusion: These data provide evidence that neutrophil NEP expression is inversely correlated with HF severity and mortality in stable HFrEF patients and is upregulated in patients with non-ischemic cardiomyopathy. There was a strong relationship between neutrophil NEP regulation and the activation marker CD11b, proposing a link between neutrophil NEP expression and systemic inflammatory response.
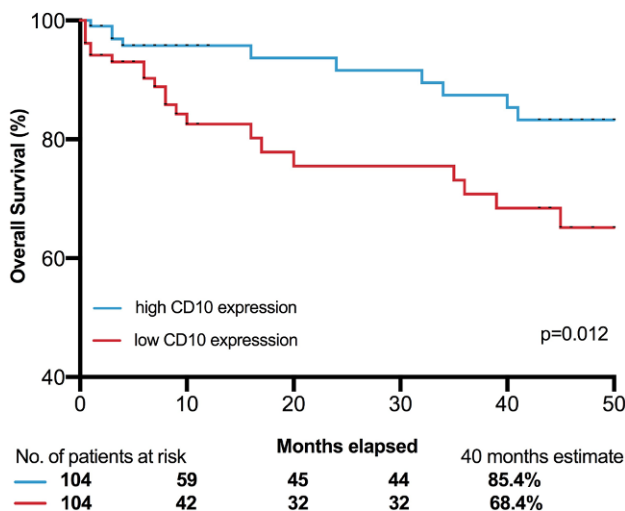
PS 26/26-6

Treatment with Tafamidis in patients with transthyretin amyloid cardiomyopathy

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Introduction: Transthyretin amyloid cardiomyopathy (ATTR-CA) is caused by deposition of amyloid fibrils in the myocardium. The deposition occurs when transthyretin (TTR) becomes unstable and misfolds. Tafamidis is a kinetic stabilizer

Fig. 2 | PS 26/26-5

	Baseline	FollowUp	Difference	p-Value
Cardiac Biomarkers	**n=31**	**n=31**		
NT-proBNP, pg/mL				
Median	2564.5	2024.0	- 540.5	**0.041**
Interquartile range	1618.5 – 5308.5.0	1070.0- 3400.3		
Troponin T, ng/L	58.08	62.38	+5.30	0.293
Functional Status	**n=26**	**n=26**		
6MWT, m	369.46	372.38	+2.92	0.276
NYHA Class, %				**0.001**
Class I	3.7	20.0	+16.3	
Class II	40.7	50.0	+ 9.3	
Class III	55.5	30.0	- 25.5	
Echocardiogram	**n=31**	**n=31**		
LV, mm	43.37	41.07	-2.30	0.098
RV, mm	35.50	34.35	-1.15	0.194
LA, mm	64.18	62.29	-1.89	0.173
RA, mm	62.07	60.46	-1.61	0.185
IVS, mm	20.27	21.78	+1.51	**0.013**
Longitudinal strain, %	-11.78	-10.98	-0.80	0.140
MRI	**n=20**	**n=20**		
IVS, mm	18.95	19.81	+0.86	0.289
Bulb aort	35.75	33.45	-2.30	**0.001**
PA diam	28.58	27.53	-1.05	**0.002**
LA, mm	70.30	67.70	-2.60	**0.031**
RA, mm	64.75	64.00	-0.75	0.087
LVEF, %	47.15	49.06	+1.91	0.732
LVEDV, ml	165.65	178.67	+13.02	**0.050**
LVSV, ml	84.75	89.19	+4.44	0.747
LVCO, l/min	5.25	5.54	+0.29	0.632
LVmass, g	204.11	204.58	+0.47	0.717
RVEF, %	41.48	43.22	+1.74	0.295
RVEDV, ml	180.95	196.67	+15.72	0.376
RVSV, ml	73.71	86.24	+12.53	0.085
RVCO, l/min	5.02	5.40	+0.38	0.604
ECV, %	47.64	48.30	+0.66	0.587

Fig. 1 | PS 26/26-6 Change from Baseline

of transthyretin that prevents tetramer dissociation and amyloidogenesis by wild-type and mutant TTR.

Methods: Thirty-one patients with diagnosis of transthyretin amyloid cardiomyopathy (wild-type) from our national amyloidosis registry were treated with tafamidis 61 mg for a period of six months. In our explorative analysis we aimed to evaluate the effects of tafamdis by changes from baseline of the serum *N*-terminal prohormone of brain natriuretic peptide (NT-proBNP) concentration, 6-minute walking distance, as well as cardiac structure and function.

Results: Main results are summarized in Table 1. In brief, we found a significant reduction from baseline in the serum NT-proBNP concentration in tafamidis-treated patients (median difference, –540.5 pg/mL, $p = 0.041$). Furthermore, a significant improvement in NYHA Functional Class after a six months treatment period was observed ($p = 0.001$). Tafamidis also improved the walking distance during 6 min at month six (mean difference, +2.92 m, $p = 0.276$). Echocardiographic findings revealed

decreases in left ventricular size (mean difference, –2.30 mm) and right ventricular size (mean difference, –1.15 mm), as well as left atrial size (mean difference, –1.89 mm) and right atrial size (mean difference, –1.61 mm). Improvements regarding the interventricular septum thickness (mean difference, +1.51 mm) and the global longitudinal strain (mean difference, –0.80 %) weren't observed. T1 mapping by cardiac MRI showed a small but insignificant increase in extracellular volume (mean, +0.66 %) in tafamdis-treated patients.

Conclusion: Treatment with tafamidis for a period of six months in patients with transthyretin amyloid cardiomyopathy results in a significant improvement in NT-proBNP levels, as well as NYHA functional class and may have positive effects on exercise capacity, cardiac structure and function.

PS 26/26-7

Myocardial work–new insights by deformation imaging in patients with advanced heart failure

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Introduction: The evaluation of myocardial contractility is essential for the prognosis of patients with advanced heart failure (HF), however currently used echocardiographic deformation imaging is controversial in the spectrum of end-stage HF. Non-invasive measurement of myocardial work is emerging as a new promising method for the assessment of myocardial contractility, as it additionally accounts for hemodynamic loading conditions of the ventricle. This study sought to assess the prognostic impact of myocardial work in patients with advanced heart failure and to compare it with routinely used deformation imaging parameters.

Methods: We included 234 patients with HFrEF under guideline directed therapy and comprehensively assessed myocardial work, including parameters of global work index (GWI), global constructive work (GCW), global wasted work (GWW), and global work efficiency (GWE), as well as global longitudinal strain (GLS) by speckle tracking echocardiography. The primary endpoint was all-cause mortality.

Results: Median age of the patients was 68 years (IQR 60–75) and 78 % were male. During a median follow up period of 60 months (IQR 20–60), 107 patients died. Median GWI was 526 mm Hg% (IQR 366–779) and median GCW was 730 mm Hg% (IQR 523–988). Parameters of myocardial work were inde-

Table 1 | PS 26/26-7 Univariable and multivariable cox regression analysis assessing the impact of parameters for myocardial contractility on long-term mortality

	Univariable model					Multivariable model		
	SD	HR	95 % CI	*P*-value	ROC	Adj. HR	95 % CI	*P*-value
Global Work Index, mmHg%	324	0.752	0.609–0.929	**0.008**	0.585	0.729	0.580–0.915	**0.006**
Global constructive work, mmHg%	369.7	0.679	0.524–0.880	**0.003**	0.625	0.658	0.495–0.876	**0.004**
Global longitudinal strain, %	3	1.325	1.079–1.627	**0.007**	0.596	1.368	1.083–1.728	**0.009**
Global wasted work, mmHg%	110.9	0.740	0.576–0.951	**0.019**	0.617	0.832	0.627–1.105	0.203
Global work efficiency, mmHg%	10.2	1.000	0.805–1.242	1.000	0.504	0.839	0.686–1.162	0.398
*adjusted for age, sex, ischemic etiology of heart failure, New York Heart Association class, creatinine, and left ventricular end-diastolic diameter								

pendently associated with long-term mortality, even after careful adjustment for clinical and echocardiographic confounders (Table 1). We further observed a significantly better calibration towards long-term mortality for GCW compared to GLS in the receiver operating characteristic curve (ROC) analysis ($P=0.007$).

Conclusion: This is the first study to comprehensively assess global myocardial work in patients with advanced heart failure. Important treatment decisions rely on the assessment of myocardial contractility and risk stratification, specifically in late stages of the disease where exact guiding of treatment success and timely allocation of more aggressive treatment strategies are warranted. By incorporating loading conditions, myocardial work seems to be able to sensitively detect changes in myocardial contractility that predict a dismal course of the disease. Our data suggests that global constructive work is a more precise parameter to predict long-term outcome than currently routinely used echocardiographic deformation imaging (i. e. GLS).

PS 26/26-8

Influence of different RAS-blockade modalities on granulocyte membrane-associated neprilysin (CD10) expression in HFrEF patients–comparison between ACEi, ARB and ARNi

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Introduction: The inhibition of the ubiquitous transmembrane enzyme neprilysin (NEP) by angiotensin receptor-neprilysin inhibitor (ARNi) therapy represents a novel mechanisms in the combat against heart failure with reduced ejection fraction (HFrEF). The soluble form of the enzyme, which can be detected in plasma, is discussed controversially as a potential biomarker in HFrEF. Plasma NEP activity is significantly reduced after therapy initiation with ARNi, whereas plasma NEP concentrations remain uninfluenced, and neither activity nor concentrations show a clear association with outcome. In contrast to soluble NEP, intact NEP (CD10) is expressed on the surface of leucocytes under physiological conditions. Alterations of NEP expression have been reported in sepsis, whereas CD10 was suggested to be indicative for neutrophil functional capacity. In a previous explorative study including patients on angiotensin-converting enzyme inhibitors (ACEi) or (angiotensin II receptor blockers) ARB we have shown that NEP expression on granulocytes is, interestingly, inversely correlated with HF severity and mortality. The aim of this study was to assess granulocyte membrane NEP expression in HFrEF patients under contemporary HF therapy including patients on angiotensin receptor-neprilysin inhibitor (ARNi) and to explore whether the different modalities of RAS-inhibition (RASi), i. e. ACEi/ARB/ARNi, have a significant influence NEP expression.

Methods: Consecutive patients with stable chronic HFrEF and optimal medical therapy, including ACEi, ARB as well as ARNi, have been enrolled prospectively. Healthy young adults without any RASi served as controls. CD10 expression expressed as mean fluorescence intensitiy (MFI) on peripheral leucocyte subsets was measured by flow cytometry in 100μL EDTA whole blood using a combination of six antibodies with fluorescence minus one sample as control [CD3(#555339), CD19(#555413), +/− CD10(#332777); BD Biosciences, San Jose, CA, USA].

Results: A total of 219 patients were included into the study. Median age was 64 (IQR 53–72) years, 161 (73 %) of the patients
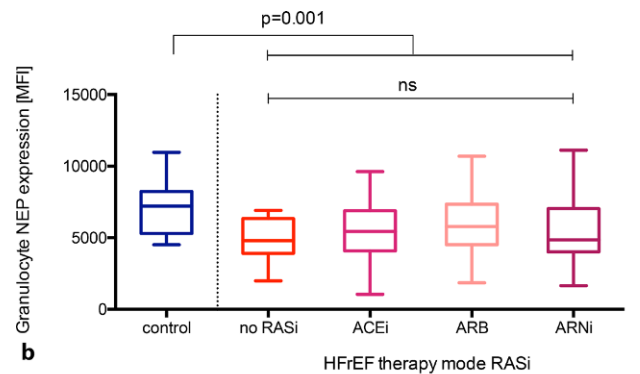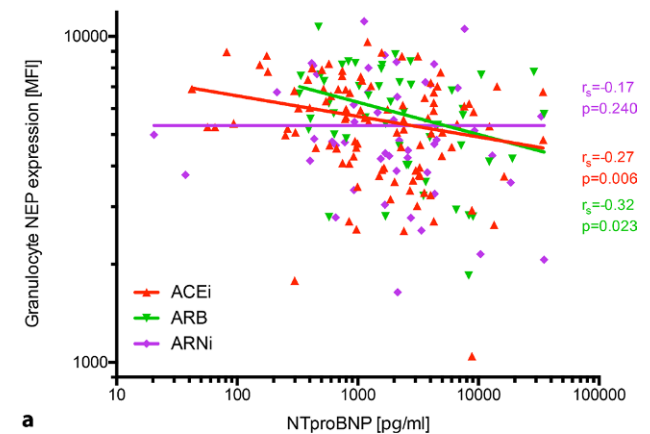
Abb. 1 | PS 26/26-8 Granulocyte NEP expression in stable HFrEF patients under contemporary guideline directed therapy according to different modalities of RAS-inhibition. a Mean fluorescence intensities of NEP (CD10) expression on granulocytes in chronic stable HFrEF patients under ACEi, ARB and ARNi or without any RASi therapy. Healthy controls are displayed for comparison. MFI of NEP expression is shown as Tukey-boxplots, *p*-values for the comparison of medians using the Kruskal-Wallis and the Mann-Whitney-U test are indicated. b Scatter plot for granulocyte NEP expression and NTproBNP levels according to ACEi, ARB or ARNi therapy. The linear log–log regression curve is shown, Spearmans correlations coefficients andsignificance of the association are indicated

were male, median NT-proBNP levels were 1861 (IQR 746–4264) pg/ml. 15 (7 %) patients were free from any RAS-blocker therapy, 101 (46 %) patients received an ACEi, 50 (23 %) patients an ARB and 53 (24 %) patients were on ARNi therapy. There was no significant difference regarding HF severity between the different RASi therapy groups ($p=0.143$ for NTproBNP and $p=0.974$ for NYHA class, comparison between all groups). Median MFI of CD10 on granulocytes was 5434 (IQR 4122–6901) for the total cohort. Fig. 1A shows granulocyte NEP expression for the different groups of RAS-blockade in HFrEF patients and controls. HFrEF was characterized by reduced granulocyte NEP expression [7203 (IQR 5337–8087) vs 5434 (IQR 4122–6901), $p=0.001$], whereas no difference in granulocyte NEP expression with regards to RASi modality ($p=0.390$) could be observed. Granulocyte NEP expression correlated significantly and inversely with NTproBNP for patients with ACEi and ARB [ACEi: rs = –0.27,

$p=0.006$ and ARB: rs $=-0.32$, $p=0.023$] however this association could not be proven for patients on ARNi [$p=0.240$] (Fig. 1B).

Conclusion: NEP inhibition by ARNi does not seem to influence the regulation of granulocyte NEP expression in chronic stable HFrEF patients. The inverse correlation between granulocyte NEP expression and NTproBNP levels is lost for patients on ARNi. Whether granulocyte NEP activity is reduced under ARNi compared to ACEi/ARB has to be investigated in future studies.

POSTERSITZUNG 27 – VITIEN 2

PS 27/27-1

Single center experience with 202 Inspiris bioprostheses

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Introduction: In the era of modern transcatheter aortic valve replacement, the use of bioprostheses versus mechanical valves is increasing, especially in younger patients with increasing wish for durable solutions without anticoagulant therapy. The Carpentier Edwards Inspiris Resilia (Inspiris; Edwards Lifesciences Inc., Irvine CA 92614, United States) bioprosthesis is presumed to outperform other available models in terms of hemodynamics and durability. However, real life data with consecutively enrolled patients have not yet been available. We aimed to investigate the hemodynamic and clinical performance of the Inspiris bioprosthesis by assessing perioperative as well as mid-term outcome.

Methods: This study was designed as a single-center retrospective observational trial. 202 patients (69.8 % male, with a mean age of 66 ± 9.8 years, range 22–84) underwent surgical aortic valve replacement with Inspiris bioprosthesis in our center between 7/2017 and 1/2020. Clinical and echocardiographic data were collected periprocedurally as well as at outpatient follow-ups.

Results: The most frequent indication for surgery was severe aortic valve stenosis (82 %) followed by aortic regurgitation (18 %). 24.3 % of the patients had a bicuspide valve, 3.5 % had a history of previous cardiac surgery. Concomitant procedures included coronary artery bypass grafting ($n=57$), replacement of the ascending aorta ($n=18$), Bentall-procedure and partial arch replacement ($n=3$), aortic root enlargement ($n=4$), aortic root reduction plastic ($n=4$) and mitral valve repair or replacement ($n=4$). 18 patients (8.9 %) were accepted for an upper hemisternotomy access. Mean aortic cross clamp and cardiopulmonary bypass time was 88 ± 34 min and 125 ± 49 min respectively. Mean implanted valve size was 23.5 ± 2.2 millimetres. Mean hospital length of stay was 13.4 ± 7.41 days. New onset of atrial fibrillation was detected in 12.4 %, atrial flutter in 1 % of the patients. Postoperative hemodynamics were as follows: mean/maximal transvalvular gradients of 13 ± 5.7 mm Hg/23.3 ± 9.9 mm Hg and a mean Vmax of 2.4 ± 0.9 m/s. 30-day mortality was 2.5 % ($n=5$). At a mean follow-up of 10.9 ± 8.5 (range 0.1–30) months overall survival was 97.5 %. Mean and maximal gradients at one year follow-up were 9 ± 3.6 and 17.3 ± 6 mm Hg, respectively, Vmax was measured to be 2.3 ± 0.8 m/

s. At two year follow-up mean and maximal gradients were 10 ± 2.2 mm Hg and 18 ± 3.2 mm Hg, while Vmax was 2 ± 06 m/s.

Conclusion: The Carpentier Edwards Inspiris Resilia bioprothesis has proven to be a safe option for aortic valve replacement with good postoperative hemodynamic performance, and has also shown excellent clinical and hemodynamic results at one and two year follow-up. Long-term studies are warranted to gain more evidence on durability as well as hemodynamic performance of this novel prosthesis.

PS 27/27-2

Tetralogy of Fallot: Is electrical dyssynchrony really a predictor for adverse events?

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Introduction: Right bundle branch block (RBBB) is frequent in patients with Tetralogy of Fallot (ToF). Prior studies could show an association of QRS width with mortality and ventricular arrhythmias, however the number of patients and especially the number of events were low in those studies. We sought to evaluate QRS width and its association with long-time cardiovascular mortality in ToF patients at our clinic for adults with congenital heart disease (ACHD).

Methods: Consecutive ToF patients presenting to our ACHD clinic were analyzed retrospectively. Only those patients with at least one documented ECG were included, QRS width was measured. Ten-year cardiovascular mortality was retrieved from the national death registry.

Results: A total of 151 patients (median age 20 years, IQR 18–32, 60 % male) were included in the final analysis. At total of 147 (96.7 %) patients were in sinus rhythm. Complete RBBB was present in 127 (84.1 %), incomplete RBBB in 12 (9 %) patients. Median QRS width was 150 ms (IQR 126–160). Four (2.6 %) patients died during 10-year follow-up. Neither QRS as a continuous variable, nor a QRS width of ≥160 ms, or ≥180 ms as categorical variables were associated with 10-year survival in a multivariate Cox regression model adjusting for age, sex, and NYHA class.

Conclusion: Late cardiovascular mortality in adult TOF patients is very low. In our patient cohort, there was no association of QRS width with outcome. Not pure electrical (ECG) but also mechanical (echocardiography) dyssynchrony should be studied in ToF patients. Electro-mechanical (un)coupling might be the real predictor for adverse outcomes and a target for future treatments.

PS 27/27-3

How low is „low-flow" in aortic stenosis? A retrospective analysis of patients with true low-flow/low-gradient aortic stenosis undergoing TAVI

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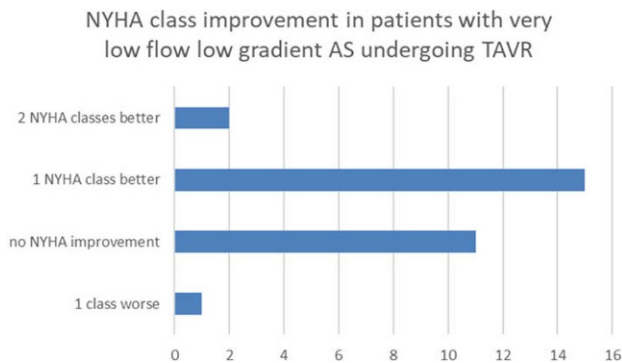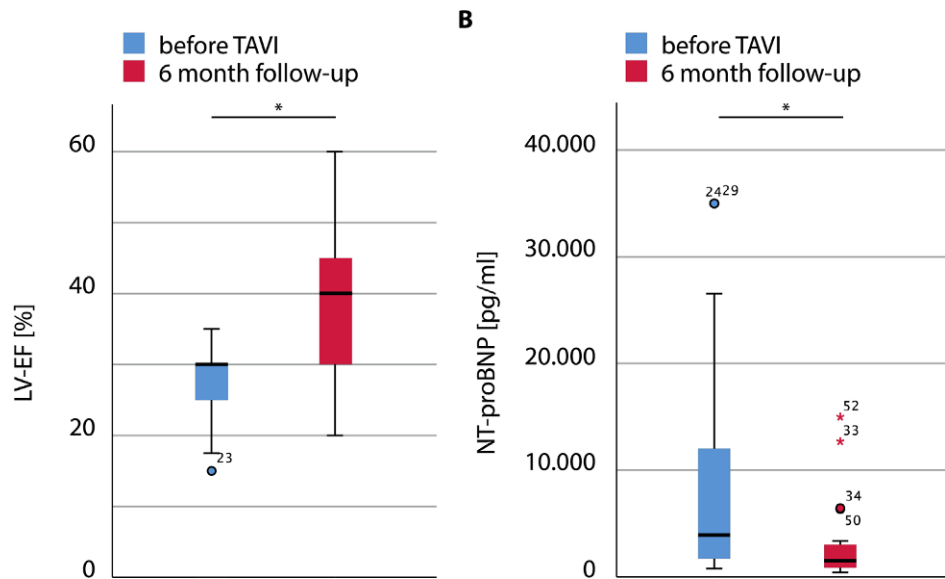
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Introduction: In recent years, transcatheter aortic valve implantation (TAVI) has become a valuable treatment option for patients with reduced left ventricular function (LVEF) and consecutive low-flow/low-gradient (LF/LG) aortic stenosis (AS). According to current literature, the presence of severe AS is unlikely in the case of severely reduced LVEF and a mean pressure gradient below 30 mm Hg. However, a considerable number of patients presenting with typical clinical symptoms and echocardiographic phenotype of severe AS, show gradients below 30 mm Hg. The aim of this study was to evaluate the clinical course and outcome in patients with LF/LG-AS, who underwent TAVI. We hypothesized, that even patients with mean gradients below 30 mm Hg would have a measurable clinical benefit and an improved LVEF as well as NYHA-class 6 month after TAVI.

Methods: Methods: In this single center retrospective study, data from patients undergoing TAVI between 2013 and 2017 was screened for individuals with characteristics of LF/LG-AS and an extraordinary low meanPG of <30 mm Hg. Transesophageal echocardiography was performed for planimetry of the aortic valve to confirm the diagnosis. A 6-month follow up was performed to assess changes in NT-proBNP levels, NYHA-Class and LVEF.

Results: 30 patients ($n=30$; female$=8$) diagnosed with LFLG-AS underwent TAVI within study period. As evidenced by preinterventional echocardiographic assessment, all patients had reduced LVEF <35 %, an aortic valve area (AVA <1 cm2), and a mean transvalvular pressure gradient (meanPG <30 mm Hg). Mean logistic EuroSCORE was 24.05 ±10.9. The majority of patients (56 %) witnessed a remarkable benefit in terms of NYHA class within the observed period, while NT-proBNP-levels decreased from 9103 ±10.151 pg/ml to 2747 ±3334 pg/ml ($p=0.002$). Moreover, LVEF increased from 27.3 %±5.6 to 38.5 %±9.9; $p=0.001$ within the observed follow-up of 6 month.

Conclusion: Based on our data we postulate that TAVI might be a valuable treatment option even for patients in the high-risk constellation of severely reduced LVEF and mean gradients far below 30 mm Hg.

PS 27/27-4

Atrial flutter caused by an uncommon etiology of tricuspid regurgitation

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Introduction: Valvular disease and atrial fibrillation/flutter (AF/AFL) interact with and sustain each other through volume and pressure overload, tachycardiomyopathy, and neurohumoral factors. In mitral regurgitation, it is well acknowledged that in cases with sever valve dysfunction, AF/AFL can be regarded as a marker for progressive disease. Tricuspid regurgitation, however, is regarded to be rather a result than a cause of AF/AFL. We present a patient in whom primary tricuspid regurgitation was identified as the cause for typical atrial flutter.

Methods: A 52-year-old man was referred because of typical atrial flutter. Echocardiography showed right atrial and ventricular enlargement with moderate tricuspid regurgitation. Despite transesophageal echocardiography and magnetic resonance imaging, the etiology of tricuspid regurgitation remained unclear. The patient underwent cavotricuspid isthmus ablation and a reablation after 6 months because of recurrence. Six months later he developed atypical AF/AFL, which was treated by pulmonary vein isolation. Eight months later he developed

recurrent atrial fibrillation and right heart failure NYHA III. 3D-transesophageal echocardiography showed a restriction of the septal leaflet due to short chordae tendineae pulling the leaflet towards the septum. The regurgitation was aggravated by annular dilatation leading to a pseudoprolapse of the other two leaflets. The restriction of the septal leaflet was assumed due to a congenital dysplasia.

Results: During surgery, a cleft between the septal and posterior leaflet was found additionally to the restriction of the septal leaflet. The cleft was sutured by Prolene 5-0 and a 34 mm Contour 3D® annuloplasty ring was implanted. Follow-up investigation after three months showed that the patient had recovered well and was asymptomatic. The right ventricle is still dilated but smaller than before. The electrocardiogram still showed atrial fibrillation.

Conclusion: Usually typical atrial flutter can be well treated by cavotricuspid isthmus ablation but if this is not the case, a structural cause in the right heart should be suspected. For the assessment of the underlying disease, multimodality imaging is necessary. It is often difficult especially to visualize tricuspid valve because of the non-planar structure with an elliptical saddle-shaped annulus [1]. If tricuspid valve dysplasia is suspected, the ideal imaging method is 3D-transesophageal-echocardiography (TEE), which allows simultaneous visualization of the leaflets, their movements and the coaptation [2]. Repair of the tricuspid valve is indicated when primary tricuspid regurgitation leads to progression of right ventricular dilatation or dete-

Abb. 1 | PS 27/27-4 3D-transesophageal-echocardiography of the tricuspid valve

Abb. 2 | PS 27/27-4 The tricuspid valve

rioration of the right ventricular function. A new-onset atrial flutter might suggest disease progression and thus indicate an early intervention [3]. In our case, tricuspid valve dysplasia could be identified with 3D-TEE as the cause of the tricuspid regurgitation and the consequent volume overload leading to typical atrial flutter. The decision for a surgical intervention was made because of right heart failure but an early reconstruction of the tricuspid valve might have been already indicated, when the patient developed atrial flutter. In the postoperative follow-up after three months, the patient still had atrial fibrillation but we are hoping, that he might be able to restore sinus rhythm, if reverse remodeling progresses.

PS 27/27-5

ECG changes and right ventricular dilatation leads to diagnosis and therapy of rare congenital heart disease–Case presentation

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Introduction: Atrial septal defect (ASD) is a congenital heart disease which can result from different anatomic defects including secundum ASD, primum ASD, sinus venosus defect and coronary sinus septal defect. As a result of left to right shunting and volume overload right heart enlargement and dysfunction can be observed. The development of irreversible PAH is a well-known long term complication [1].

Methods: Patient 1: An 18-year-old asymptomatic male was referred to our cardiology department because of incomplete right bundle branch block (RBBB) in routine ECG and suspicious right ventricular dilatation. Echocardiography showed a mildly dilated right ventricle with preserved ejection fraction. Furthermore, an unroofed coronary sinus ASD with persistent left vena cava superior and bidirectional shunt could be identified as the cause of right ventricular load. Pro-BNP levels were in normal ranges. The cardiac defect was completely repaired by open heart surgery. The unroofed coronary sinus was closed with a pericardial patch Patient 2: A 26-year-old female was referred from a peripheral hospital due to increasing dyspnea. ECG was abnormal with incomplete RBBB and right axis deviation. Echocardiography showed a dilated right ventricle with suspicion of ASD in the superior interatrial septum. Elevated systolic pulmonary artery pressure of 50 mm Hg was estimated. Pro-BNP was elevated with 386 pg/ml. CT proofed the sinus venosus atrial defect with partial anomalous pulmonary venous connection (PAPVC, right upper pulmonary vein to vena cava superior). Right heart catheterization showed normal pulmonary pressures. The patient underwent Warden Repair without any complications. Postoperative echocardiography reveals normalization of right ventricular dimensions

Results: Both defects could be repaired by open heart surgery. The unroofed coronary sinus was closed with a pericardial patch. The patient with sinus venosus atrial defect and PAPVC underwent Warden Repair.

Conclusion: In patients with unexplained right ventricular overload, an atrial connection should be ruled out. Echocardiography is the first method of choice, despite normal echocardiographic findings doesn't definitely exclude a possible connection. Imaging techniques like TEE, MRI, CT, and nuclear imaging can provide additional information about the presence and direction of shunting as well as the evaluation of associated abnormalities like partial anomalous venous connection or
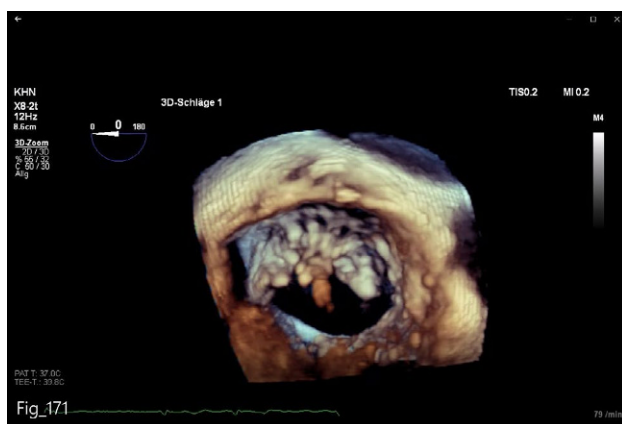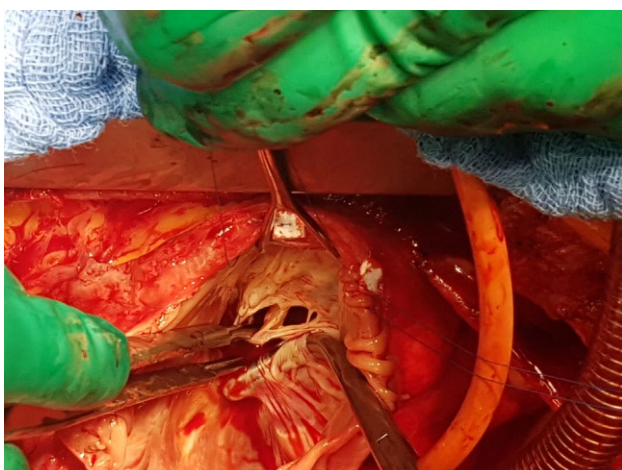
unroofed coronary sinus ASD. Early repair is essential to avoid the development of pulmonary hypertension.

PS 27/27-6

Myocardial work efficiency: A new echocardiographic parameter in the follow-up of patients with repaired Tetralogy of Fallot

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Introduction: As many as 21 % of patients with Tetralogy of Fallot (TOF) have previously been described to suffer from reduced left ventricular function (LVF). In these patients, besides low preload in the presence of severe pulmonary regurgitation, especially dyssynchronous contraction due to right bundle branch block can lead to systolic lengthening during LV ejection, resulting in "wasted work" and thereby reduced LVF. The new echocardiographic parameter myocardial work efficiency (MWE) can be calculated as ratio between constructive work and the sum of wasted and constructive work. We sought to evaluate the feasibility of MWE in our TOF cohort both as a single parameter and as a follow-up parameter measured at two time points.

Methods: All patients presenting at our outpatient clinic for adults with congenital heart disease with the diagnosis repaired TOF were included. For final analysis, only those patients who were in sinus rhythm and had sufficient 2D image quality of an apical-4-chamber, an apical-3-chamber, and an apical-2-chamber view in the index transthoracic echocardiography examination (TTE) and in one additional examination fulfilling the same criteria were included. Left ventricular ejection fraction (LVEF), global longitudinal strain (GLS), myocardial work load (MWL), and MWE were calculated.

Results: Eight patients were included in the final analysis, median age was 31.5 years (21–47), five (31 %) were male, and median time between the two TTE was 15.5 months (12–21.5). With 58 % vs 57 %, –18 % vs. –18 %, 1948 mm Hg% vs. 1960 mm Hg%, and 94 % vs. 94 % there was no difference between the two time points in LVEF, GLS, MWL, and MWE. During this short-time follow-up none of the patients died, and none of them suffered clinical deterioration.

Conclusion: In this small cohort of TOF patients, LVF was normal in all participants. MWE with 94 % was only slightly reduced. Measures did not differ between the two TTE and might therefore be feasible parameters which can be included into the longtime echocardiographic follow-up of these patients.

POSTERSITZUNG 28 – RISIKOFAKTOREN/STOFFWECHSEL/LIPIDE 3

PS 28/28-1

Chronic kidney disease is a type 2 diabetes risk equivalent in patients with established coronary artery disease

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Introduction: Both type 2 diabetes (T2 DM) and chronic kidney disease (CKD) confer a high risk of cardiovascular disease (CVD). We aimed at investigating the single and joint effects of T2 DM and of CKD on cardiovascular event risk in high-risk patients with established coronary artery disease (CAD).

Methods: We prospectively investigated 1460 patients with angiographically proven CAD over 10.3 ± 4.8 years.

Results: Cardiovascular events occurred more frequently in T2 DM patients ($n = 449$) than in non-diabetic subjects (56.2 % vs. 44.5 %; $p < 0.001$) and in patients with CKD (eGFR <60 ml/min/1.73 ^{m2}; $n = 264$) than in those with an eGFR ≥60 ml/min/1.73 ^{m2} (61.7 % vs. 45.1 %; $p < 0.001$). When both, T2 DM and CKD were considered, 856 subjects had neither T2 DM nor CKD, 340 had T2 DM but not CKD, 155 did not have diabetes but had CKD, and 109 had both T2 DM and CKD. When compared with the cardiovascular event rate among patients with neither T2 DM nor CKD (42.5 %), cardiovascular risk was significantly higher in patients with T2 DM who did not have CKD (51.5 %; $p = 0.002$) as well as in non-diabetic patients with CKD (55.2 %; $p = 0.009$) and was highest in patients with both, T2 DM and CKD (71.0 %; $p < 0.001$), in whom the event risk was higher than in those with T2 DM but no CKD ($p < 0.001$) or those without T2 DM but with CKD ($p = 0.005$); event risk however was did not differ significantly between non-diabetic CKD patients and T2 DM patients who did not have CKD ($p = 0.692$).

Conclusion: We conclude that CKD is a T2 DM risk equivalent in patients with established CAD.

PS 28/28-2

Early onset of menopause is associated with increased peripheral atherosclerotic plaque volume and progression–a risk factor unique to women

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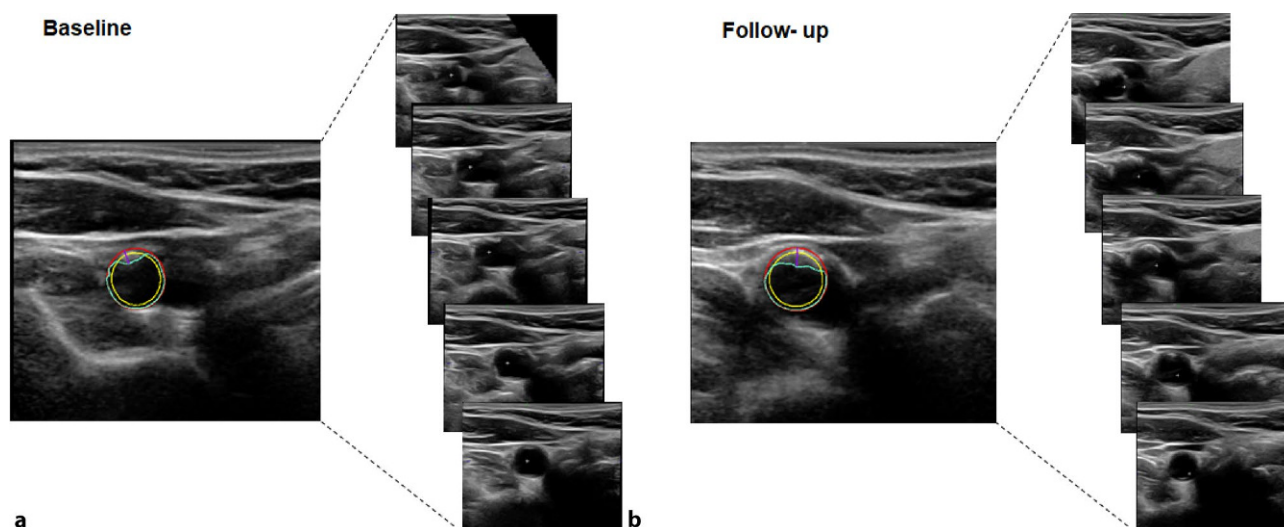
Fig. 1 | PS 28/28-2 Quantification of plaque volume of the right carotid artery using images obtained from 3D ultrasound at (a) Baseline and (b) Follow-up, showing atherosclerotic progression in a female with early onset of menopause
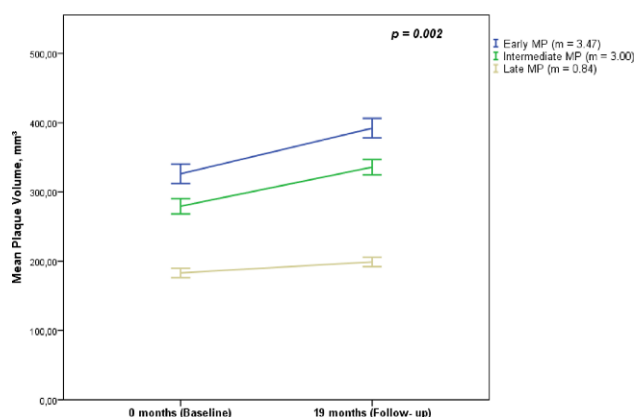
Abb. 2 | PS 28/28-2 Line diagram representing the total plaque volume progression for early (< 45 years), intermediate (45 to 52 years) and late (> 52 years) onset of menopause. Baseline and follow-up examinations are 19 (± 8) months apart. Slope of the lines are displayed in the figure

Introduction: Cardiovascular disease (CVD) is the leading cause of death in western countries. One risk factor unique to women is the menopausal status. The aim of this study was to analyse the influence of the onset of menopause (MP) on the extent and progression of atherosclerotic plaque volume (PV).

Methods: Postmenopausal women with at least one cardiovascular risk factor (CVRF) but without established CVD were included. Quantification of PV was performed in peripheral arteries using a three-dimensional (3D) ultrasound (US) technique (see Fig. 1). Follow-up examination to assess PV progression was performed after 19 (± 8) months.

Results: 110 consecutive postmenopausal women (mean age 65.5) were included. Females with an earlier onset of MP (< 45 years) had a significantly higher PV than those with an intermediate (45–52 years) or later onset of menopause (> 52 years), irrespective of other CVRF (244 mm³ vs. 193 mm³ vs. 73 mm³, respectively, $p = 0.023$). In addition, women with an earlier onset of MP had a higher PV progression compared to women with an intermediate or late onset (40 mm³ vs. 35 mm³ vs. 8.5 mm³; $p = 0.002$, respectively; see Fig. 2). Moreover, these results were confirmed in multivariate regression, where only onset of MP (OR 0.88; 95 %CI 0.81–0.96; $p = 0.004$) and age (OR 1.06; 95 %CI 1.08–1.13; $p = 0.025$) were significant predictors for a higher atherosclerotic progression.

Conclusion: An earlier onset of MP was associated with an increase in atherosclerotic PV and accelerated progression, independent of other CVRF. Our results support the argument that risk estimation based solely on conventional cardiovascular risk classification systems may underestimate the risk in a particular individual. Particularly in women, risk stratification should include the evaluation of menopausal status and its onset as these parameters may also influence atherosclerotic disease burden and progression beyond conventional risk scores. The strengths of our study are the prospective study design and exact quantification of atherosclerotic PV. To our knowledge there is no prior study that assessed carotid and femoral atherosclerotic progression in postmenopausal women.

PS 28/28-3

The body mass index during adolescence as an early predictor for future cardiovascular and metabolic health

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Introduction: The body mass index (BMI) is regarded a major risk factor for metabolic disorders, cardiovascular diseases and cancer. Despite several studies aimed to investigate the relationship between BMI early in life and future metabolic risk, results are controversial, and data are often incomplete. Thus, we aimed to investigate the influence of BMI during ado-

lescence on health during senescence in a large cohort of comprehensively characterized patients.

Methods: 1638 patients were included as part of a colorectal carcinoma colonoscopy screening program between 03/2007 and 12/2011. Patients were characterized using biochemical and metabolic parameters, as well as a detailed questionnaire on dietary and lifestyle habits. Only patients with stable weight within the last 6 months were included. Data on each individual's weight at 10 and 20 years were extracted from health questionnaires. Finally, the diagnosis of hepatic steatosis was established using ultrasound.

Results: Patients with a mean age of 58.4 ± 10.0 years were analyzed, including 820 (50.1%) patients with hypertension and 227 (13.9%) with type 2 diabetes mellitus (T2 DM). When stratifying patients according to their BMI at 20 years (<20 kg/mg^2: $n = 337$; 20–25 kg/mg^2: $n = 1072$; 25–30 kg/mg^2: $n = 203$; >30 kg/mg^2: $n = 26$), patients had a higher BMI at later study timepoint (24.8 ± 4.4 vs. 27.2 ± 4.3 vs. 30.5 ± 5.7 vs. 34.1 ± 7.0 kg/m^2, $p < 0.001$) and a higher prevalence of hypertension (43.0% vs. 49.7% vs. 61.6% vs. 65.4%, $p < 0.001$), T2 DM (11.6% vs. 13.0% vs. 20.7% vs. 26.9%, $p = 0.003$) and hepatic steatosis (39.5% vs. 49.0% vs. 59.1% vs. 65.4%, $p < 0.001$). On multivariate analyses correcting for age, gender, alcohol consumption, smoking status, dyslipidemia, hypertension and T2 DM, BMI at 20 years was associated with a higher risk for hypertension (adjusted hazard ratio [aHR]: 1.068 [95%CI: 1.027–1.111], $p = 0.001$), T2 DM (aHR: 1.051 [95%CI: 1.002–1.104], $p = 0.043$) and hepatic steatosis (aHR: 1.076 [95%CI: 1.036–1.117], $p < 0.001$) despite BMI at study timepoint was a stronger risk factor (aHR for hypertension: 1.132 [95%CI: 1.103–1.161], $p < 0.001$); (aHR for T2 DM: 1.138 [95%CI: 1.103–1.174], $p < 0.001$); (aHR for hepatic steatosis: 1.296 [95%CI: 1.253–1.339], $p < 0.001$).

Conclusion: BMI during adolescence could serve as a very early marker for future metabolic disorders or cardiovascular diseases. Therefore, lifestyle interventions could be useful to change the course of disease at an early time.

PS 28/28-4

Influence of alcohol consumption on cardiovascular and metabolic risk

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Introduction: Alcohol has been recognized as a significant contributor to global morbidity and mortality. Despite the established believe of the deteriorating effect of significant alcohol consumption (≥ 20 g/day for female and ≥ 30/g for male) on liver health, studies report controversial results about the effect lower amounts on cardiovascular health. Here, we investigate their effect on cardiovascular and metabolic risk.

Methods: 4809 individuals were included as part of a colorectal carcinoma colonoscopy screening program between 03/2007 and 07/2019 from a single center. Patients were char-

acterized using biochemical and metabolic parameters. Data on alcohol consumption were extracted from food frequency questionnaires. For group comparison, patients were stratified according to their alcohol consumption into insignificant (none or <10 g/week), moderate (10–130 g/week) or significant (≥ 140 g/week) consumption. Factors of the metabolic syndrome were defined according to the International Diabetes Federation (2005).

Results: Prevalence of male gender (41.1% vs. 74.5% vs. 92.8%), hypertension (67.0% vs. 71.0% vs. 83.0%) and metabolic syndrome (43.7% vs. 45.4% vs. 68.6%, all $p < 0.001$) were significantly different between groups with the highest prevalence in patients with significant alcohol consumption. Notably, the prevalence of prediabetes (34.1% vs. 38.1% vs. 44.4%, $p = 0.001$) increased in parallel with alcohol consumption. On multivariate binary regression analysis adjusted for age, BMI gender, smoking status and physical activity, alcohol consumption was associated with hypertension (adjusted hazard ratio [aHR] per 10 g: 1029, 95% confidence interval [CI]: 1.006–1.052, $p = 0.012$), metabolic syndrome (aHR: 1.031, 95%CI: 1.011–1.051, $p = 0.002$) and prediabetes (aHR: 1.022, 95%CI: 1.001–1.043, $p = 0.036$). Finally, linear regression analysis revealed an independent association between alcohol consumption and Framingham risk score (regression coefficient B: 0.056, 95%CI: 0.003–0.108, $p = 0.040$) indicating higher cardiovascular risk.

Conclusion: Metabolic and cardiovascular risk might be significantly influenced by alcohol consumption, starting already at low or moderate levels.

PS 28/28-5

Kardiovaskuläre Risikofaktoren bei akutem Myokardinfarkt

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Einleitung: Klassische Risikofaktoren wie Rauchen, Diabetes mellitus, Hyperlipidämie und arterielle Hypertonie spielen bei der Entstehung der koronaren Herzkrankheit (KHK) eine ausgeprägte Rolle. So ist bekannt, dass über 80% der an KHK erkrankten Menschen mindestens einen dieser Risikofaktoren aufweisen. Ein Auftreten von KHK im frühen Alter kann bei Männern in Zusammenhang mit Rauchen gebracht werden. Bei Frauen sind es Rauchen und Diabetes, welche mit einem frühen Auftreten von KHK assoziiert sind. Bei Rauchern führt der Nikotinkonsum, im Vergleich zu Nichtrauchern, zu einer früheren Manifestation der KHK um beinahe 10 Jahre.

Methoden: Im Rahmen der Akutkoronarangiographien bei akutem MCI (STEMI, NSTEMI) wurden bei 50 Patienten Faktoren (Alter, STEMI/NSTEMI, Größe, Gewicht, BMI, Blutzucker, LDL, TG, AHT, HL, DM, HU, Nikotin, KHK, PAVK, CAVK, Sport, Familie) erhoben, die das kardiovaskuläre Risikoprofil beeinflussen.

Resultate: Auffällige ist, dass 71,4% Nikotin konsumieren. Im Vergleich liegt der Raucheranteil in der Gesamtbevölkerung nur bei 24,3% und nimmt mit dem Alter ab. Dem Gegenüber geben 60,9% an regelmäßig Sport zu betreiben, wobei in der vergleichbaren Gesamtbevölkerung 56,1% mindestens 150 min sportlich aktiv sind.

Schlussfolgerungen: Bei Patienten mit einem MCI scheinen Raucher erheblich überrepräsentiert zu sein. Weiters dürften sich die Patienten hinsichtlich des Ausmaßes ihrer körper-

lichen Aktivität als protektiven Faktor überschätzen. Die in der Vergangenheit gesundheitspolitischen und aufklärenden Maßnahmen bezüglich Gesundheitsbewusstsein scheinen noch nicht zu einem ausreichenden Erfolg geführt zu haben und bedürfen einer weiteren Intensivierung.

PS 28/28-6

Type 2 diabetes and risk of cardiovascular events in peripheral artery disease versus coronary artery disease patients

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Introduction: The prevalence of type 2 diabetes (T2 DM) is higher in peripheral artery disease (PAD) than in coronary artery disease (CAD) patients, and PAD overall confers higher cardiovascular risk than CAD. How cardiovascular risk compares between PAD and CAD patients when analyses are stratified by the presence of T2 DM is unclear and is addressed in the present study.

Methods: We prospectively recorded cardiovascular events over 7.6 ± 5.0 years in 253 patients with PAD, of whom 41.9 % had T2 DM and in 923 patients with stable CAD, of whom 26.7 % had T2 DM. Four groups were analyzed: CAD patients without diabetes (CAD/T2 DM–; $n = 677$), CAD patients with T2 DM (CAD/T2 DM+; $n = 246$), PAD patients without diabetes (PAD/T2 DM–; $n = 147$) and PAD patients with T2 DM (PAD/T2 DM+; $n = 106$).

Results: The cardiovascular event rate was lowest in CAD/T2 DM- patients (40.5 %). It was significantly higher in CAD/T2 DM+ patients (50.2 %; $p = 0.007$), in PAD/T2 DM- patients (55.2 %; $p < 0.001$), and in PAD/T2 DM+ patients (67.6 %; $p < 0.001$), who in turn were at a higher risk than CAD/T2 DM+ or PAD/T2 DM- patients ($p < 0.001$ and $p = 0.013$, respectively). Further, cardiovascular risk was significantly higher in PAD/T2 DM- than in CAD/T2 DM+ patients ($p < 0.001$). Cox regression analysis after multivariate adjustment confirmed that PAD versus CAD (HR = 2.58 [2.12–3.15]; $p < 0.001$) more strongly than the presence of T2 DM (HR = 1.55 [1.30–1.85]; $p < 0.001$) predicted cardiovascular events.

Conclusion: We conclude that even when compared to CAD PAD confers a higher risk than T2 DM. PAD patients without diabetes are at a higher risk than the extremely high risk patients with the combination of CAD and T2 DM; risk is exceedingly high in PAD patients with T2 DM.

PS 28/28-7

The A body shape index and type 2 diabetes are mutually independent predictors of cardiovascular events and mortality 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 cardiovascular events and mortality 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 1546 patients with established CVD (1299 patients with angiographically proven stable coronary artery disease and 247 patients with sonographically verified PAD) over a mean follow-up time of 9.7 ± 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 ± 0.5; $p < 0.001$). Prospectively, the ABSI significantly predicted the incidence of cardiovascular events ($n = 749$) and death ($n = 619$) after adjustment for age, gender, smoking, hypertension, LDL cholesterol, HDL cholesterol, and T2 DM (standardized adjusted HRs 1.11 [1.03–1.19]; $p = 0.006$ and 1.18 [1.09–1.27]; $p < 0.001$, respectively). T2 DM in turn also significantly predicted cardiovascular events and death in this fully adjusted model independently from the ABSI, with adjusted HRs of 1.37 [1.17–1.0]; $p < 0.001$ and 1.40 [1.18–1.6]; $p < 0.001$, respectively.

Conclusion: We conclude that the ABSI and T2 DM are mutually independent predictors of cardiovascular events and mortality in patients with PAD.

BEST ABSTRACTS CHIRURGIE

BA 1/1-2

Simultaneous carotid endarterectomy and coronary artery bypass grafting–a single center experience

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Introduction: Patients requiring coronary artery bypass grafting (CABG) and carotid endarterectomy (CEA) receive staged (CABG before CEA), reversed-staged (CEA before CABG) or simultaneous treatment. The most efficient operative strat-

egy remains unknown. This retrospective study evaluated the outcome in patients undergoing simultaneous CEA and CABG.

Methods: Between 2000 and 2019 a total of 160 patients (70.1 ±8.3 years; 72.5 % male) with triple-vessel or left main trunk symptomatic coronary artery disease associated with asymptomatic uni- or bilateral carotid artery stenosis ≥70 % underwent simultaneous CEA and CABG at our institution. CEA was performed before cardiopulmonary bypass followed by CABG. Primary study endpoints were defined as mortality, stroke and myocardial infarction at 30 and 90 days. Also, the composite endpoint of these events was investigated.

Results: Mean logarithmic EuroSCORE was 9.0 ±7.3 %. Bilateral carotid artery stenosis ≥70 % was present in 18.8 % of patients and peripheral arterial disease in 28.7 % of patients. In 96.3 % of cases eversion carotid endarterectomy was performed. Mean aortic cross-clamp time was 39 ±14 min, and cardiopulmonary bypass time was 83 ±30 min. Median stay on the intensive care unit was 2 days. Mortality, stroke and myocardial infarction rates were 5 %, 7.5 % and 5 % at 30 days and 7.5 %, 7.5 % and 5 % at 90 days, respectively. Combined adverse events occurred in 13.1 % ($n=21$) at 30 days and in 13.8 % ($n=22$) of patients at 90 days. All 12 patients suffering from postoperative stroke had severe cerebrovascular occlusive disease, including contralateral carotid artery stenosis ≥50 % in 8 patients and occlusion in 2 patients.

Conclusion: Patients with coronary and cerebrovascular occlusive disease undergoing simultaneous CEA and CABG are at increased risk of perioperative cardiovascular events, including stroke. Results of this study are comparable to data from a limited number of randomized controlled trials.

BA 1/1-3

Pediatric ross operation–over 25 years of single centre experience

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Introduction: If aortic valve replacement becomes inevitable, the Ross procedure is an attractive option for the management of aortic valve disease in children. In patients with multi-level left ventricular outflow tract obstruction a Ross-Konno procedure is performed to relieve subvalvular tunnel-like obstruction. Data on long-term outcomes over 20 years is limited in pediatric patients following the Ross procedure. Pulmonary autograft and homograft durability remain a concern regarding re-intervention rates. We reviewed our experience with the pediatric Ross procedure, which is performed since 1991 at our center to report on survival and freedom from re-operation rates in the third decade after intervention.

Methods: A retrospective analysis of all patients aged younger than 18 years who underwent a Ross or Ross-Konno procedure at our institution since 1991 was conducted. Mortality was cross-checked with the Austrian Federal Statistical Agency providing a mortality follow-up until February 2020. Long-term survival status is known for 97.1 % of patients. 3 patients are lost to follow-up. These patients were only trans-

ferred to our center for surgery and postoperatively followed at non-Austrian centers. Time-related events were assessed using Kaplan–Meier estimator.

Results: From April 1991 to January 2020, 102 children aged less than 18 years underwent a Ross respectively Ross-Konno ($n=14$) procedure at our institution. The patients (70.6 % male) had a median age of 10.1 (IQR 5.7–14.4) years. 11.8 % percent were younger than one year of age at time of surgery and 4 procedures were performed in neonates. 68.6 % of the patients had undergone a previous aortic valve intervention. 51 % of the patients had undergone a previous sternotomy. The patients had short ICU stays with a median ICU stay of 2 (IQR 1–5) days and a median hospital stay of 13 (IQR 10–16) days. Median follow-up time was 11.3 (IQR 2–18) years with a maximum follow up of 27.6 years. Early-mortality was 0.98 % and there were 6 late deaths. Survival was 90.9 % and freedom from any Ross-related reoperation was 58.4 % at 25 years. Freedom from pulmonary re-operation was 68.2 % and freedom from autograft re-operation was 88.8 % at 25 years. Root reinforcement of the autograft was performed in 12.7 % of the patients either with the autograft being sutured into a Valsalva prothesis or as formerly performed being wrapped with a vicrly mesh or the remnant aortic wall. 17 patients had 22 re-operations in pulmonary position. 8 patients underwent transcatheter pulmonary valve replacement with a mean time to implantation of 11 ±3.6 years after Ross operation. Three patients underwent re-operation after percutaneous pulmonary valve replacement.

Conclusion: Pediatric Ross procedure shows excellent long-term survival. Complication- and mortality-rate were higher in the more complex Ross-Konno than in the Ross cohort. As expected, re-operation was more common in the pulmonary position than on the autograft and was associated with younger age. Postponing re-operation in pulmonary position with the option of a transcatheter valve is an attractive option to reduce re-operation rate over lifetime.

BA 1/1-4

External prosthetic reinforcement of the pulmonary autograft

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Introduction: Following the Ross procedure with implantation of the autograft (AG) as free-standing root replacement, dilation of the neo-aortic root occurs in a substantial proportion of patients, especially during the second postoperative decade. This remains a major drawback of the procedure, even if a valve-sparing reoperation can be performed in the majority of cases. This retrospective study evaluates early results of prosthetic reinforcement of the pulmonary AG in order to prevent aneurysmal degeneration.

Methods: Between 1991 and 2017 a total of 191 patients underwent a Ross procedure at our institution using different operative techniques. Since 2015, in 16 adolescent and adult patients (mean age of 27.1 ±16.1 years) the AG was externally reinforced with a straight Dacron graft. Clinical and echocardiographic follow-up is 100 % complete with a mean of 19.7 ±5.8 months.

Results: The mean aortic cross-clamping time was 102 ±39 min, and the mean cardiopulmonary bypass time was 241 ±64 min. Mean total operative time was 6.9 ±2.1 h. The median stay

on the intensive care unit was 2 days. No bleeding complication requiring a re-exploration occurred. In-hospital mortality was 0 %. Patients were discharged with grade 0 (14 patients) or grade I aortic regurgitation (2 patients). In one patients both the AG and homograft were replaced 3 months after the Ross procedure because of endocarditis, leading to 93.8 % freedom from reoperation at 2 years. The mean length of hospital stay was 14.3 ± 6.2 days. There were no late deaths during the study period. Follow-up echocardiography conformed stable neo-aortic root diameters and stable AG valve function in all patients.

Conclusion: Prosthetic reinforcement of the pulmonary AG can be performed with low morbidity and mortality and provides an excellent early valve function.

BA 1/1-5

Klappenerhaltender Neoaortenwurzelersatz nach Norwood-Rekonstruktion der Aorta

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Einleitung: Einleitung Chronischer Systemdruck kann zu aneurysmatischer Ausweitung der Pulmonalarterie führen, wie dies Erfahrungen nach arterieller Switch-Operation und der Ross-Operation als freiem Wurzelersatz zeigten. In gleicher Weise kann dies nach einer Norwood Operation passieren, bei der im Neugeborenenalter der Pulmonalarterienhauptstamm zur Neoaortenrekonstruktion genutzt wird. Die Aneurysmenbildung ist ein über Jahre langsam fortschreitender Prozess, der auch zu einer Klappeninsuffizienz führen kann.

Methoden: Methoden Wir führten bislang bei 3 Patienten nach einer Norwood Rekonstruktion einen Prothesenersatz der Neoaorta aszendens unter Erhaltung beider Semilunarklappen durch. 2 Patienten waren zum Zeitpunkt der Operation bei hypoplastischem Linksherzsyndrom bzw. Trikuspidalatresie, TGA und hypoplastischer Aorta im Fontan Status (18 Jahre und 8 Jahre alt) mit Neoaszendensaneurysma. Der dritte Patient mit Dextrokardie, Aortenatresie, hypoplastischem Aortenbogen und Ventrikelseptumdefekt erhielt im Neugeborenenalter eine biventrikuläre Vollkorrektur in Form einer Norwood-Rastelli Operation. Im Alter von 2 Jahren führten wir einen Aszendensersatz bei Aneurysma und signifikanter Neoaortenklappeninsuffizienz durch.

Resultate: Resultate Die klappenerhaltende Rekonstruktion erfolgte unter milder Hypothermie an der extrakorporalen Zirkulation. Aortenklemmzeiten waren 99 min, 130 min, und 148 min. Die ursprüngliche hypoplastische Aortenwurzel wurde aus der Norwood Rekonstruktion herausgelöst, die aneurysmatisch erweiterte Pulmonalarterienwand bis zur Klappenebene vollständig entfernt und durch eine Dacronprothese ersetzt (20, 22 und 24 mm Durchmesser). Dabei kam zweimal eine Yacoub-Technik und einmal eine David-Technik zur Anwendung. Die originäre Aorta wurde mit Hilfe einer End-zu-Seit Anastomose mit der Prothese verbunden. In einem Fall wurde auch die originäre Aortenwand durch eine zweite Dacronprothese ersetzt, da sie ebenfalls aneurysmatisch erweitert war. Postoperativ zeigten sich eine gute Ventrikelfunktion und dichte Neoaortenklappen ohne Gradient über der Aortenrekonstruktion.

Schlussfolgerungen: Schlussfolgerungen Die Yacoub oder David Methode des klappenerhaltenden Neoaortenwurzeler-

satzes mit End-zu-Seit Anastomose der originären Aorta ist eine gute Behandlungsoption für späte Neoaortenaneurysmen nach einer Norwood-Operation.

BA 1/1-6

Shockwave therapy facilitates reprogramming of somatic cells towards endothelial cells via TLR3-dependent epigenetic modifications

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Introduction: Reprogramming of resident somatic cells towards functional endothelial cells is a promising strategy for the regeneration of ischemic tissue. Thereby, stimulation of the innate pattern recognition receptor Toll-Like receptor 3 (TLR3) is crucial for effective chromatin remodeling and nuclear reprogramming. Mechanical conditioning of ischemic tissue via shock wave therapy (SWT) has been shown to have distinct angiogenic effects in a TLR3-dependent manner. We hypothesized, that the observed angiogenic effects of SWT may be due to transdifferentiation of somatic cells towards endothelial cells.

Methods: Human cardiac Fibroblasts (Fb) were treated with SWT or TLR3 agonist polyI:C, transferred to Endothelial Differentiation Medium and analyzed for endothelial-specific markers via rt-PCR and FACS analysis after 7 and 14 days. Reprogrammed cells positive for CD31 were sorted, assessed for endothelial gene expression and subjected to functional endothelial cell assays. Myocardial infarction was induced in C57BL/6 with subsequent SW treatment. Cardiac function was assessed via echocardiography and CD31 positive endothelial cells were quantified to assess angiogenic effects in vivo.

Results: Human cardiac fibroblasts show abundant expression of TLR3. Mechanical stimulation activated the TRIF-dependent TLR3 signaling. TLR3 stimulation resulted in altered expression of chromatin-modifying enzymes. Furthermore, increased expression of PRDM14 and methylation of histone 3 could be observed, clearly indicating epigenetic modifications. Upon treatment, fibroblasts showed significantly increased expression of endothelial markers CD31, VEGFR2 and VE-Cadherin. Effects were reversible using a TLR3/dsRNA complex inhibitor. In line, we found an increased population of endothelial cells in FACS analysis, indicating a phenotype-switch of fibroblasts upon treatment. Reprogrammed sorted cells positive for CD31 showed indeed an endothelial gene expression profile with endothelial cell properties including NO-production and tube formation. In vivo, we found increased numbers of CD31-positive endothelial cells concomitant with improved cardiac function after treatment. These effects were absent in mice additionally treated with a TLR3 inhibitor.

Conclusion: TLR3 activation shows clearly an angiogenic and regenerative potential in ischemic heart disease, as it facilitates transdifferentiation of somatic fibroblasts towards an endothelial cell-type via chromatin remodeling. Therefore, mechanical TLR3 stimulation displays an efficient treatment strategy for myocardial ischemia.

BA 2/2-2

Outcome of rapid deployment aortic valves: Single-center long-term experience after 750 implants

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Introduction: The Edwards INTUITY Valve System is an aortic bioprosthesis with a balloon-expandable stent frame that enables rapid-deployment valve replacement. We analyzed our single-center long-term experience with a follow-up of up to ten years.

Methods: Between May 2010 and February 2020, 750 consecutive patients with severe aortic stenosis or combined aortic valve disease implanted with a rapid-deployment valve at our institution were included in a prospective and ongoing database with a median follow-up of 19 months and a total accumulated follow-up of 2140 patient-years. Preoperative characteristics, operative parameters, survival, valve-related adverse events, and valve hemodynamics were assessed.

Results: The mean age was 74 ±8 years, 46 % female. Concomitant procedures were performed in 362 (48.3 %) patients. In the case of isolated AVR (388/700), a minimally invasive surgical (MIS) approach was conducted in 297 patients (76.5 %). Cross-clamp and cardiopulmonary bypass times for isolated aortic valve replacement were 107.7 ±29.1 and 73.9 ±21.8 min for MIS approaches and 93.0 ±28.7 and 57.6 ±20.4 min for full sternotomy ($p < 0.001$). Mean gradients at discharge, one year, three and five years were 13 ±5, 11 ±4, 12 ±5 and 13 ±8 mm Hg. New early pacemaker implantation was required in 9.1 %. Reintervention or reoperation with valve explantation for structural degeneration, non-structural dysfunction or endocarditis occurred in 22 cases (2.9 %); 2 (0.3 %) cases were Valve-in-Valve procedures for structural valve degeneration. Thirty-day mortality was 0.7 % (5/750) and overall survival at one, three and five years was 95 %, 88 % and 80 %.

Conclusion: This RD-AV has shown excellent long-term results concerning hemodynamic performance, durability and safety.

BA 2/2-3

Reality show–TAVI and SAVR in a real world scenario

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Introduction: 1.) Evaluation of TAVI and SAVR in a real world scenario 2.) all-comers base 3.) stratified in low, intermediate and high STS risk groups

Methods: 376 TAVI and 1130 SAVR were performed (2007–2017) Multivariate Cox-regression model were calculated.
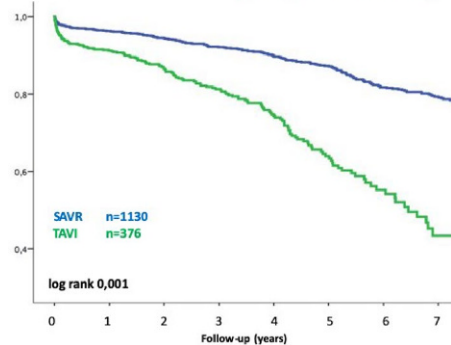
Fig. 1 | BA 2/2-3
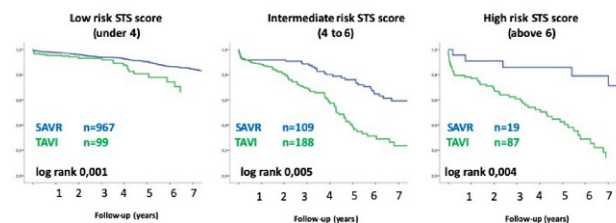
Fig. 2 | BA 2/2-3

Standardized Covariates of Cox-Regression: age gender LV-function creatinine clearance diabetes atrial fibrillation NYHA III–IV COPD redo-surgery

Results: see graphs

Conclusion: In a real world scenario, results after AVR show dramatically different survival of SAVR compared to TAVI Patient selection is of up-most importance.

BA 2/2-4

Der Aortenklappenstenose-Patient mit niedrigem und intermediärem EuroSCORE II – Langzeitergebnisse aus den Heart Team-Besprechungen des universitären Herzzentrum

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Einleitung: Die Ausweitung der Indikationen auf niedrigere Risikoklassen wird aufgrund der guten Transkatheter-Aortenklappen-Implantation (TAVI)-Resultate der Hochrisiko-PatientInnen angestrebt. Ziel dieser Studie war es, die 30-Tages- und die Langzeit-Mortalität des konventionellen Aortenklappenersatzes (AKE) im Vergleich zur TAVI im Universitären Herzzentrum zu evaluieren.

Methoden: Daten von 418 PatientInnen aus den interdisziplinären Heart Team-Besprechungen aus den Jahren 2011–2014 wurden retrospektiv untersucht und in Untergruppen ± CABG/PCI unterteilt. Die Ergebnisse des Kollektivs mit EuroScore II

(ES II) ≤4 % (Gruppe I) wurden mit denen aus dem Kollektiv mit ES II von 4–8 % (Gruppe II) verglichen. Für 116 PatientenInnen fand sich ein rezenter Eintrag im Krankenhausinformations-System, die restlichen 302 wurden telefonisch kontaktiert.

Resultate: Das mittlere Alter zum Zeitpunkt des Eingriffes lag in der AKE-Gruppe bei 77 ± 6,4 Jahren, in der TAVI-Gruppe bei 82 ± 6,2 Jahre. Lost-to-Follow-up waren 3,3 % der PatientInnen. Die mittlere Beobachtungszeit lag bei 5,5 ± 1,2 Jahren. Die 30-Tages-Mortalität zeigte keinen signifikanten Unterschied zwischen beiden Verfahren in Gruppe I (AKE ($n=67$) 4,5 %, AKE + CABG ($n=19$) 0 % versus TAVI ($n=157$) 5,7 %, TAVI + PCI ($n=27$) 7,4 %) und Gruppe II (AKE ($n=15$) 6,7 %, AKE + CABG ($n=18$) 16,7 % versus TAVI ($n=82$) 8,5 %, TAVI + PCI ($n=19$) 0 %). Ähnlich verhält es sich mit der 1-a-Mortalität in beiden Kohorten (Gruppe I: AKE ($n=67$) 10,4 %, AKE + CABG ($n=19$) 5,3 % versus TAVI ($n=157$) 13,4 %, TAVI + PCI ($n=27$) 29,6 %; Gruppe II: AKE ($n=15$) 6,7 %, AKE + CABG ($n=18$) 16,7 %, TAVI gesamt ($n=82$) 18,3 %, TAVI + PCI ($n=19$) 10,5 %). Die Langzeitergebnisse zeigen, dass das Kollektiv in der Gruppe I nach AKE im Mittel 55 ± 25,5 Monate lebt, das Kollektiv AKE + CABG 50 ± 21,4 Monate, respektive das TAVI-Kollektiv 48 ± 23,7 sowie TAVI+PCI 38,9 ± 30,2 Monate. Gruppe II zeigt ein Überleben nach AKE im Mittel von 60 ± 21,2 Monaten, AKE + CABG 48 ± 23,3 Monaten, nach TAVI 42 ± 25,4 Monaten und nach TAVI+PCI 43 ± 19,5 Monaten.

Schlussfolgerungen: Die Resultate dieser retrospektiven Analyse einer „wahren Welt"-Population zeigen im Vergleich zu den randomisierten Studien mit zahlreichen Ausschlusskriterien, dass die AKE-Gruppe ein besseres Langzeitergebnis aufweist. Die AKE-Gruppe war durchschnittlich um fünf Jahre jünger, das TAVI-Kollektiv wies Risikofaktoren auf, die im ES II nicht abgebildet werden. Dies bestätigt, dass der EuroSCORE II allein für die Festlegung des optimalen Therapiekonzeptes nicht ausreichend ist. Die präoperative Risikostratifizierung durch ein multidisziplinäres Herz-Team ist essentiell für die optimale PatientInnenversorgung.

BA 2/2-5

Aortic valve replacement with decellularized homografts: a single-center experience

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Introduction: Aortic valve replacement with homografts obtained from human donors is an excellent alternative to biological or mechanical valve substitutes. Compared to cryopreserved homografts, in case of decellularized homografts (DH) all donor cells are removed using different enzymatic and detergent schemes, but the extracellular matrix is preserved, which reduces immunogenicity and allows spontaneous recellularization of the graft with the recipient's cells, with the hemodynamic benefits of a human native valve. The aim of this work was to analyze our single-center experience after the implantation of decellularized aortic homografts.

Methods: Vienna General Hospital is one of the centers included in the multicentric European ARISE clinical trial (Aortic Replacement Using Individualised Regenerative Allografts). The aim is to evaluate decellularized aortic homografts (DAH) regarding safety, durability and hemodynamic performance. The DAH were evaluated considering the presence/absence of the adverse reactions and freedom of valve dysfunction. Each patient received a non-seeded decellularized aortic homograft and and has been followed-up in our center after the procedure.

Results: Since 2016, 36 patients, mean age 42.9 ± 17.5 (2 to 62 years), 11 (31 %) female received a DAH in our center. One patient previously underwent an aortic valve replacement, two patients previously underwent a valvuloplasty. Mean diameter of the implanted DAH was 21.4 ± 6.7 mm. Mean bypass time and mean cross-clamp time were 159.6 ± 49.8 min and 124.8 ± 35.3 min. The mean follow-up was 13.7 months, up to 41.7 months. Operative complications occurred in 5.5 % patients: in one case, bleeding of the distal anastomosis which required a 2nd cross-clamp time and a pericardial patch extension and another case of coronary kinking after reimplantation of the the left main, with subsequent coronary bypass need. Survival is 100 % in this small cohort of patients. No cases of non-structural dysfunction, structural degeneration, valve endocarditis or thrombosis have been observed. Reoperation with valve explanation was also absent. The mean gradient at follow-up was 6.0 mm Hg.

Conclusion: Early results showed a very low rate of postoperative complications and excellent hemodynamic features of DAH at our center.

BA 2/2-6

On-X vs. St Jude–comparison of two mechanical heart valves with low-thrombogenic profile

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Introduction: Mechanical prostheses are characterized by their exceptional longevity. Throughout the evolution, bileaflet heart valves remained comparable in terms of basic design structures: occluder, housing and sewing ring. It is thus assumed that long-term results will not vary much. Early haemodynamic performances have already been compared between the On-X and the St Jude mechanical prosthesis in previous studies. Whether mid- or long-term outcomes differ is yet to be investigated.

Methods: 459 consecutive patients have undergone aortic valve replacement with either of these two prostheses from first implementation until December 2016 at the General Hospital of Vienna. Data on survival, complication rates and surgical specifics were acquired within a retrospective analysis from the Viennese documentation system and through a cross-sectional telephone follow up.

Results: The On-X prosthesis was implanted in 227 patients and compared to 232 subjects with the St Jude mechanical heart valve. Mean age was 56 +/- 11 years for the On-X group vs 51 +/- 12 years for the St Jude group. On-X patients were followed for a mean of 9 +/- 6 years for survival and 7 +/- 6 years for adverse events, a mean follow up of 7 +/- 5 years was observed for survival and 5 +/- 4 years for adverse events in the St Jude group. Apart from minor differences in the presence of pulmonary hypertension (On-X < St Jude) and chronic kidney disease (On-X > St Jude) baseline characteristics were equally distributed. Concomitant procedures were performed in 49 % ($N=110$) with the On-X prosthesis and 60 % ($N=140$) with the St Jude prosthesis. Over-all mortality was slightly higher within the On-X group (36 % $N=82$ vs 26 % $N=60$, $p=0.02$), however, early mortality was comparable between both groups (13 % $N=11$ vs 17 % $N=10$, $p=0.637$). Valve-related deaths occurred in 8 % ($N=19$) in the On-X group and 10 % ($N=22$) in the St Jude

group ($p < 0.05$). The incidence of valve-related complications was equal in both groups in terms of valve thrombosis, non-cerebral embolism, arrhythmias, reinterventions and bleedings. Interestingly, the incidence of endocarditis was likelier with the St Jude whereas strokes were more common in the On-X group (3 % $N=6$ vs 5 % $N=12$, $p=0.008$ and 11 % $N=24$ vs 5 % $N=12$, $p=0.037$).

Conclusion: We conclude that both prostheses offer similar long-term outcomes despite relevant differences in over-all mortality due to the higher number of high-risk patient population in the On-X group. It should be considered whether the St Jude prosthesis can be treated with lower target INR as well. Further investigations are required in the future.

BA 2/2-7

15 years of frozen elephant trunc: long-term results in the therapy of aortic syndromes

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Introduction: Frozen Elephant Trunc (FET) Prosthesis is an established therapeutic option in the treatment of complex aortic diseases with involvement of the arch and proximal descending aorta. We aimed to investigate the long-term outcomes after FET repair.

Methods: 100 consecutive patients (57 % male, with a mean age of 61.2 years, range 29–77) received a FET repair with Jotec Evita Open hybrid graft (Hechingen, Germany) in our department between 8/2005 and 3/2017. Indications included acute and chronic dissections and thoracic aortic aneurysms. In-hospital data were collected retrospectively. Outpatient follow-up with CT-scan was carried out on an annual basis. Follow-up was 96 % complete, at 5.5 ±3.6 years (range 0.2–14.5 years). Endpoints included operative morbiditiy and mortality, long-term overall survival as well as the need for secondary interventions.

Results: Concomitant procedures included aortic root replacement $n=14$, aortic valve replacement $n=8$, CABG $n=13$, mitral valve surgery $n=3$ and intraprocedural thoracic endovascular aortic repair (TEVAR) $n=2$. In-hospital mortality, spinal

Freedom from reinterventions

Fig. 1 | BA 2/2-7

Table 1 | BA 2/2-7

Freedom from secondary interventions (Kaplain-Meier estimates)	Mean (in years ± SE)	At 5 years (% ± SE)	At 10 years (% ± SE)
Acute dissections	11.5 ± 0.9	89.8 ± 5.6	70.4 ± 11.3
Chronic dissections	8.3 ± 1.4	63.3 ± 13.3	47.5 ± 16.9
Aneurysms	7.2 ± 0.7	75.4 ± 7.7	11.7 ± 10.5
Overall	9.1 ± 0.6	78.9 ± 4.7	43.8 ± 8.2

cord injury and permanent stroke rates were: 8 %, 1 % and 7 % respectively. At 5 years, Kaplan-Meier overall survival was 74.5 ±5.0 % and freedom from aortic death was 86.5 ±3.7 %, whereas at 10 years, overall survival was 57.4 ±7.1 % and freedom from aortic death was 78.5 ±6.6 %. Causes of late aortic mortality included aortic rupture ($n=3$), aortic-oesophageal fistule ($n=1$), aorto-bronchial fistule ($n=1$) and complications following open thoracoabdominal aortic repair ($n=2$). 32 patients needed a reintervention of the thoracic aorta, 3 of those were performed as an emergency (one open thoracoabdominal aortic aneurysm [TAAA] repair and one TEVAR for acute rupture as well as one Bentall-procedure due to re-dissection of the aortic root), whilst 29 cases (11 TAAA repairs and 18 TEVARs) were planned and indicated by large or growing diameter of the descending aorta. The Kaplan-Meier estimated freedom from secondary interventions according to initial FET indication is shown in the table and diagram.

Conclusion: Frozen elephant trunc became the primary therapeutic option in extensive thoracic aortic diseases involving the arch and/or the proximal descending aorta. The high reintervention rate for chronic and especially for type B dissections as well as for thoracic aneurysms is related to already existing pathologies. Late expansion of the remaining untreated aorta with possible fatal outcome occurs even after 10 years, therefore careful follow-up and timely planning of reinterventions are mandatory in this patient cohort.

BA 2/2-8

Antegrade stentgraft delivery in acute type A dissection–the good, the bad and the ugly

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Introduction: We analyzed the mid-term results of antegrade stentgraft delivery in the descending thoracic aorta in acute type A dissection (AAD).

Methods: Outcomes were evaluated for 375 patients who underwent surgery for AAD between February 2000 and August 2019 (standard repair, SR, $n=337$, 90 %; standard repair+ antegrade stentgraft delivery, SR+TEVAR, $n=38$; 10 %). 89 % ($n=33$) of patients who received antegrade TEVAR had computed tomography post-surgery. Median follow-up time in the whole cohort was 21 months.

Results: Mean age at time point of AAD 59.5 years. 76 % of patients were male. Indications for antegrade TEVAR were: entry/re-entry of the dissection in the descending aorta in 50.0 % ($n=19$), true lumen compromise in 16 % ($n=6$) and malperfusion syndrome of lower extremities and/or abdominal organs in 13 % ($n=5$). Chronic dissection or aneurysm of the descending aorta was present in 11 % of patients ($n=4$)
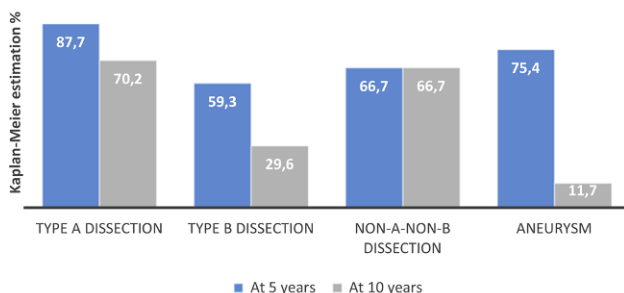
and intramural haematoma in 5 % ($n=2$). The remaining 5 % of patients ($n=2$) had more than one indication for TEVAR of the descending aorta. Technical success of intraoperative TEVAR could be achieved in 95 % ($n=36$). Perforation of the descending aorta during stentgraft deployment occurred in one patient. In one case additional placement of a second antegrade TEVAR due to inaccurate placement was necessary. Evaluation of hospital outcome did not reveal any significant differences between the two treatment groups. Out of 15 patients who suffered from preoperative malperfusion of abdominal organs or lower limbs, 13 were free from signs of postoperative malperfusion after TEVAR. Mid-term survival was superior the SR group ($p=0.05$). While 22 patients (67 %) showed a good result in follow-up imaging. 9 patients (27 %) suffered from a postoperative endoleaks (5 type Ia, 4 type II), 1 patient (3 %) from pseudoaneurysm and 3 patients (9 %) from type B dissections during follow up and reintervention rate was of 8 % (3 patients).

Conclusion: Antegrade TEVAR in AAD is an easy applicable and helpful tool to improve antegrade distal downstream flow in order to regain/stabilize organ perfusion and or to secure distal open anastomosis against bleeding from retrograde expansion of the dissection. However, careful indication of treatment or modifications of proximal sealing of the stentgraft are necessary due to a non-negligible number of endoleaks. In order to evaluate impact of antegrade TEVAR on remodelling of the distal aorta longer follow-up times are needed for validation.

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