isas Leibniz-Institut für Analytische Wissenschaften – ISAS – e.V.

Dortmund & online

COLLOQUIUM

Right heart failure – insights from clinical and basic research

Speaker:

Prof Dr med Tanja Rudolph, Senior physician at the Clinic for General and Interventional Cardiology/Angiology at Herz- und Diabeteszentrum Nordrhein-Westfalen, Universitätsklinik der Ruhr-Universität Bochum, Bad Oeynhausen.

and

PD Dr Anna Klinke, Head of Agnes Wittenborg Institut für translationale Herz-Kreislaufforschung, Bad Oeynhausen.

Time:

Thursday, April 03, 2025 – 1 pm

Venue:
ISAS Campus, Lecture Hall
Otto-Hahn-Straße 6b
44227 Dortmund

&

WebEx: https://t1p.de/e0ovz
Meeting-ID: 2789 902 5055
Password: gHYCuppm987

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Abstract

Right heart failure (RHF) is a serious condition with complex pathophysiology, often linked to underlying pulmonary diseases and left heart failure leading to poor quality of life, increased risk of hospitalization and death.

Secondary right ventricular (RV) dysfunction due to RV pressure or volume overload is the most common reason for RHF. Left-sided heart failure as well as tricuspid regurgitation (TR) are the leading underlying causes in clinical practice. RV adaptions ("coupling") to maintain adequate flow leads to RV dilatation, reduced RV function and worsening of tricuspid regurgitation resulting in progression of RHF.

There is growing evidence that addressing tricuspid regurgitation with interventional and surgical strategies is able to reverse RV dysfunction translating into symptomatic improvement and better outcome. Also, we were able to show in a multicenter registry in a CT-based analysis that TR-induced RV enlargement affects primarily the apical part of the RV, whereas contraction is mainly reduced in the mid and basal part of the RV. Right ventricular–pulmonary arterial (PA) uncoupling was associated with increased mortality after one-year following catheter-based tricuspid valve interventions. Moreover, longitudinal and RV free wall contraction were associated with preserved RV-PA-coupling ratios.

Currently, there is no dedicated medical therapy which has been shown to have beneficial effects on symptoms, clinical course and prognosis of patients with RHF. Therefore, an advanced understanding of RHF mechanisms on systemic, hemodynamic, cellular and molecular level is urgently required to enhance therapeutic options. To meet this demand, we complement our clinical studies with experimental murine models of RV pressure or volume overload combining extensive functional phenotyping

with mechanistic investigations. We have observed that the transition from RV dysfunction to RHF can occur independently from structural myocardial remodeling but is driven by mitochondrial oxidative stress. Pharmacologic interventions targeting mitochondrial integrity show promising results in mice, which point towards an improvement of RV diastolic function and an alleviation of RHF symptoms. Investigation of patient RV myocardium facilitates the evaluation of experimental findings.

With these translational approaches we seek to advance individualized diagnostic and treatment options for RHF patients.