Right ventricular function in moderate-to-severe ventricular tricuspid regurgitation: Is there a pseudo-normalization of conventional echocardiographic parameters?

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Background: A reliable evaluation of right ventricular (RV) function in the presence of moderate-to-severe tricuspid regurgitation (m/s-TR) is of paramount importance for surgical or interventional risk stratification. Due to the significant systolic regurgitation of blood from the RV to the low-pressure right atrium, conventional echocardiographic parameters could appear normal and thus overestimate RV function (pseudo-normal). The aim of this study was to analyse and compare RV function in trace TR versus m/s-TR by the invasive pressure-volume-loop (PV-loop) technique, the gold standard of RV function analysis, with conventional echocardiographic indices in patients with the ventricular form of mild/no vs. m/s-TR.

Methods: The study was conducted in 134 patients with HFrEF in a post-hoc analysis (110 from the Magdeburg CRT Responder Trial, and 24 from a local CRT-optimization trial).

Results: The logistic regression analysis demonstrated that RV size (RV-end-diastolic volume, RVEDV) and RV function (measured as RV fractional area change, FAC), TAPSE, and lower fractional shortening (FS) of proximal the RV-outflow tract (RVOTprox) was closely associated with PV-loop-derived RV total afterload (pulmonary arterial elastance, Ea) and coupling of RV contractility (end-systolic RV elastance, Ees) to Ea (Ees/Ea) (all, p<0.001). Compared to trace TR (n=98, 73%), m/s-TR (n=36, 27%) was accompanied with significant higher total afterloads (Ea: 0.75 vs. 0.41, p<0.001) and lower RV-PA coupling ratios (Ees/Ea, 0.4 vs. 0.79, p<0.001). This was associated with enlarged RVs (RVEDV: 210ml vs. 166ml), lower FAC (31% vs. 50%), TAPSE (13mm vs. 18mm), and RVOTprox (22% vs. 39%) (all p<0.001). For better comparability between trace TR and m/s-TR tertiles (T1-T3) of similar afterloads (Ea) were formed. The m/s-TR patients showed significantly lower RV-PA coupling ratios than trace TR at the lower two Ea tertiles T1 (p=0.034) and T2 (p<0.001). At Ea T3 the difference was no longer significant (p=0.055). The hemodynamic data are mirrored by significant lower FACs, FS RVOTprox, and TAPSE at T1 and T2 (all p<0.05), and a nonsignificant difference at T3 in m/s-TR.

In conclusion: In our cohort of HFrEF patients, echocardiographic RV function reflects very closely the hemodynamic coupling efficiency of RV contractility (Ees) to afterload (Ea) and seems independent from the extent of TR. We found no evidence for pseudo-normal or overestimated echocardiographic RV function in m/s-TR.