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Methods: TTEs performed for assessment of rhMS were included. Standard parameters of MS severity were measured (MVA [Planimetry/PHT]; PG/MG). MV and LVOT VTIs were recorded. Patients with >mild MR or aortic valve disease were excluded to avoid potential haemodynamic confounders.

Results: 76 patients (55 ± 14 years; 75% females) were included. MVA classified patients into mild (n=20; MVA $1.74\pm0.19\,\mathrm{cm}^2$), moderate (n=25; MVA $1.22\pm0.13\,\mathrm{cm}^2$) and severe (n=31; MVA $0.86\pm0.16\,\mathrm{cm}^2$) rhMS. MG and PG were significantly different between the rhMS grades but wide overlapping ranges resulted in loss of discriminatory power. MG:PG and MV VTI:LVOT VTI were able to differentiate severe from mild/moderate rhMS. Ideal cut-offs were MG:PG>0.5 (Sensitivity 90%; Specificity 87%) and MV VTI:LVOT VTI>3.5 (Sensitivity 84%; Specificity 91%) to identify severe rhMS.

Conclusions: MVA assessments are not always feasible and discordance in guideline recommended parameters are commonly encountered. MG:PG and MV VTI:LVOT VTI may be less flow/rate-dependent and offer incremental value in confirming severe rhMS.

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One Year Assessment of Left and Right Ventricular Function with Mid-Septal Pacing Using Strain Echocardiography



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Background: Right ventricular (RV) apical pacing is associated with left ventricular (LV) dysfunction. Furthermore, we have previously shown a decline in RV systolic function early after pacemaker insertion with mid-septal pacing. The aim of this study was to reevaluate biventricular function after one year of mid-septal pacing, including measurement of global longitudinal strain (GLS).

Methods: 23 patients requiring a permanent pacemaker, with normal baseline biventricular function, underwent echocardiography prior to implantation and at one year. We measured LV and RV GLS, LV ejection fraction (EF), tricuspid annular plane systolic excursion (TAPSE) and velocity (RV S'), as well as grade of tricuspid regurgitation (TR).

Results: Mean age was 75 ± 9.8 years. 16 (70%) patients had atrioventricular nodal dysfunction; 7 (30%) had sick sinus syndrome. 7 of 23 patients (30%) required $\geq 50\%$ ventricular pacing. At one year, there was a significant decline in LV GLS (-21.5% vs. 16.6%, p < 0.001), RV GLS (-25.8% vs. -21.0%, p < 0.001), LVEF (64% vs. 58%, p = 0.039) and RV S′ (13.9 vs. 11.6, p = 0.008) There was no correlation between any of these parameters and percentage of ventricular pacing. There was no change in TAPSE or TR grade between baseline and one year.

Conclusions: At one year, there was a decline in several parameters of biventricular systolic function that did not correlate with percentage of ventricular pacing. Further studies are required to evaluate the significance and aetiology of this

finding, and the longer-term effects of mid-septal pacing on LV and RV strain.

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Performance of a Novel Echocardiographic Marker Against Right Heart Catheterisation in Identifying Pulmonary Hypertension due to Left Heart Disease



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Introduction: Pulmonary hypertension (PH) requires separation into pulmonary vascular (pre-capillary) and left heart (post-capillary) causes. ePLAR, the echocardiographic Pulmonary to Left Atrial Ratio, is calculated as the ratio between peak tricuspid regurgitation (TR) velocity and the mitral E:E' ratio. We investigated whether ePLAR could differentiate precapillary from post–capillary disease in patients undergoing right heart catheterisation (RHC).

Methods: 887 patients undergoing RHC at Royal Perth Hospital between 1st January 2010 and 31st December 2015 were included. Echo studies performed within 2 months of the RHC were extracted from the National Echo Database Australia.

Results: 601 patients (68%) had PH (mPAP \geq 25 mmHg), mean age 68 \pm 19 years, 56% (n = 499) were males. ePLAR could be calculated in 184 patients of all patients who underwent RHC, of whom 59 patients (32%) did not have PH (mPAP < 25 mmHg, mean ePLAR 0.19 \pm 0.10 m/s).

123 patients with PH and measurable ePLAR were separated into pre-capillary (PCWP < 15 mmHg, n = 18) and post-capillary (PCWP \geq 15 mmHg, n = 105) causes. Cardiac Output was frequently not measured, so Pulmonary Vascular Resistance was not included. Despite similar mean PAP, pre-capillary PHT patients (age 67 ± 20 years, 39% males, mPAP 37 ± 9.6 mmHg, PCWP 11.7 ± 2.9 mmHg) had a mean ePLAR of 0.35 ± 0.2 m/s vs 0.19 ± 0.1 m/s (p < 0.001) for post-capillary patients (age = 67 ± 20 years, 56% males, mPAP 37.2 ± 9 mmHg, PCWP 24.2 ± 7 mmHg). Using multivariate regression (including ejection fraction, E:E′ ratio, left atrial volume, E-wave velocity and E:A ratio), ePLAR remained an independent predictor of post-capillary PH (standardised β-coefficient = -0.9, p-value = 0.001).

Conclusion: ePLAR is an independent predictor of PH due to left heart disease and can help to differentiate between pre and post-capillary PHT.

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