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The Echocardiographic Pulmonary to Left Atrial Ratio (ePLAR): Identifying Cause of Pulmonary Hypertension and Subsequent Mortality Risk

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Background: ePLAR has been proposed as a simple, noninvasive transthoracic echocardiographic (echo) marker to help identify cause of pulmonary hypertension (PH). ePLAR is the ratio of peak tricuspid regurgitation velocity (TRV, indicates pulmonary artery pressure (PAP)) to the E:E' ratio (indicates left ventricular filling pressure).

Methods: ePLAR was applied to the National Echo Database Australia (NEDA) to assess broad applicability, then to the Armadale Echo Study, in which both the cause of PH and mortality status was known. Left heart diseases (LHD) were pooled together.

Results: In NEDA (302,746 echos on 174,229 patients), TRV was not measurable in 40% of patients and E:E' ratio in 121,019 patients (39%). ePLAR was calculated in 126,031 patients (41%).

In the Armadale study, 898 patients with PH were identified, and 256 had sufficient TR and measured E:E' ratios to allow a valid ePLAR calculation (29%). ePLAR was lower in PH due to LHD (0.17 ± 0.07, n = 174, age 76 years, PAP 55.7+/−10.0 mmHg) than other causes for PH (0.27 ± 0.12, n = 82, mean age 72.9 years, PAP 58.5+/−13.9 mmHg). In LHD, below-median ePLAR of 0.17 was associated with increased mortality (mean survival times 1180 vs 1605 days, p = 0.007). ROC curves demonstrated AUC = 0.796, P < 0.001 for PH from left heart disease.

Discussion: ePLAR can be calculated in 41% of echos, and when PH is identified, is a simple discriminator of LHD from other causes. In PH due to LHD, a lower ePLAR predicts a higher mortality, and may be useful as a prognostic tool.

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The Outcome of Applying the 2016 ASE/EACVI Guidelines for the Evaluation of Left Ventricular Diastolic Function to 50,000 Echocardiograms

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The 2016 ASE/EACVI diastology guidelines focus on four core parameters (septal/lateral e'vel, E/e', TRvel and LAVi), arranged in two algorithms.

Methods: 50,000 echocardiograms were retrospectively recruited and the algorithms were applied electronically using logical operators. The first algorithm was applied if EF≥50% and the second if diastolic dysfunction (DD) was found present, or if EF < 50%.

Results: With EF≥50%, DD was not present (69%), present (12.5%) or indeterminate (12.9%). In those with DD, a further 42% were considered indeterminate using the second algorithm. An indeterminate result occurred most frequently due to abnormal e'vel and LAVi, and normal TRvel and E/e', possibly because the guideline cut-off is near the mean e'vel(septal8 ± 3 cm/s, lateral10 ± 4 cm/s) and LAVi(33 ± 11 ml/m2), but closer to 1SD from the mean for TRvel(2.5 ± 0.4 m/s) and E/e'(10.0 ± 4.5). This is reflected in the high-sensitivity(>80%), low-specificity(<80%) of e'vel and LAVi, and low-sensitivity(<60%), high-specificity(>95%) of E/e' and TRvel for DD. In EF≥50%, DD was grade 1 in 7.3%, 2 in 41.4%, 3 in 5.2%. When EF < 50%, DD was grade 1 in 45%, 2 in 17%, 3 in 10%, indeterminate in 15%. Increasing DD grade was significantly associated with decreasing e'vel and increasing E/e', TRvel and LAVi(p < 0.00001). E/e' > 14 had the highest PPV(99%) and NPV(93%) for DD.

Conclusion: The new guidelines frequently diagnosed "indeterminate" DD. Fewer patients with EF≥50% were found to have grade 1 than grade 2 DD, probably due to many being misclassified as normal or indeterminate. Both outcomes may be due to the cut-off values chosen and the use of the same cut-offs in both algorithms.

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The Relationship Between Lipoprotein Particle and Attenuated Coronary Atherosclerotic Plaques in Patients Treated with Apabetalone — Insights from ASSURE Trial

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Background: Apabetalone (RVX-208), the first selective bromodomain and extra-terminal (BET) inhibitor, modulates lipid and inflammatory pathways implicated in atherosclerosis. The impact of apabetalone on attenuated plaque (AP), a measure of vulnerability, is unknown.

Methods: The ApoA-1 Synthesis Stimulation and intravascular Ultrasound for coronary atheroma Regression Evaluation (ASSURE) study employed serial IVUS measures of coronary atheroma in 281 patients treated with RVX-208 or placebo for 26 weeks. Attenuated plaque (AP) was measured at baseline and follow-up. Factors associated with changes in AP were investigated.

Results: AP was observed in 31 (11%) patients. The apabetalone group demonstrated reductions in AP length by 1 mm (p = 0.03), AP arc by 37.0° (p = 0.004) and the AP index (API) by 34.6 mm° (p = 0.02). The change in API correlated with the on-treatment HDL particle concentration (r = -0.50, p = 0.007), but not HDL-C (r = -0.20, p = 0.36) or