LVEF, LAVImin ≥18 mls/m² (HR 3.15 [95% CI 1.70–5.54], p < 0.001) showed a stronger association with outcome than LAVImax ≥34 mls/m² (HR 1.79 [95% CI 1.02-3.14], p = 0.041). Interactions and comparisons of the model x2 and Harrell’s C statistic confirmed superiority of LAVImin. In the invasive cohort, whilst LAVImin and LAVImax had a similar correlation at baseline (r = 0.41 [p < 0.001] versus r = 0.42 [p < 0.001]), LAVImin ≥18 mls/m² had a greater sensitivity for LVEDP ≥15 mmHg than LAVImin ≥34 mls/m² (sensitivity 59.4% versus 34.4%).

Conclusions: Using thresholds of ≥18 and ≥34 mls/m² respectively, LAVImin was a better predictor of survival than LAVImax, the pathophysiologic basis of which relates to LAVImin having improved sensitivity for detection of elevated LVFP. There could be incremental clinical value in measuring LAVImin alongside LAVImax.

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Who should be Screened for Subclinical Myocardial Dysfunction?
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Background: Detection of subclinical left ventricular (LV) dysfunction requires echocardiography, but it is unclear in whom and when screening should be performed.

Methods: This study assessed 617 young adults (aged 26–36 years) and followed for 13 years. Physical measurements, biochemistry and questionnaire were done at both baseline and follow-up. Echocardiogram was done at follow-up. This was compared against further definitive investigation within cardiology and intensive care groups of 0.82 and 0.85 respectively. Comparing Likert scores to definitive cardiac investigations, cardiology had a sensitivity of 83.3% and specificity of 55.9% for diagnosing type 1 MI, compared to 83.3% and 73.5% for intensivists. The ROC curve analysis showed no significant difference between the two groups (p = 0.4).

Conclusions: Asymptomatic myocardial disease may be detected in substantial numbers of apparently healthy middle-aged individuals. Prediction models derived from simple clinical and demographic variables are helpful in identifying individuals likely to have asymptomatic disease detectable with echocardiography.

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Clinical Cardiology/Clinical Trials (336–451)

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A Comparison of Cardiologist and Intensivist Clinical Assessment in Determining Type 1 Versus Type 2 Myocardial Infarction in a Critical Care Setting
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Introduction: High sensitivity troponin has been critical in detecting both thrombotic myocardial injury (type 1 MI), and non-thrombotic myocardial injury (type 2 MI) caused by demand ischaemia. However, non-specific troponin elevation in critical care settings creates a diagnostic dilemma in balancing the increased risks of invasive coronary assessment against misdiagnosis and incomplete revascularisation. Making this distinction relies on clinical assessment; however there is significant anecdotal discrepancy between cardiologists and intensivists. We sought to determine the accuracy of clinical assessment between these two groups in categorising type 1 versus type 2 MI.

Method: Forty clinical vignettes comprising cardiac history, electrocardiograms, and echocardiograms were presented to a group of blinded cardiologists (n = 9) and intensivists (n = 7). Participants were asked to predict likelihood of type 1 MI for each case on a 10-point Likert scale. This was compared against further definitive investigation including coronary angiograms and/or serial echocardiography.

Results: There was strong average inter-class correlation within cardiology and intensive care groups of 0.82 and 0.85 respectively. Comparing Likert scores to definitive cardiac investigations, cardiology had a sensitivity of 83.3% and specificity of 55.9% for diagnosing type 1 MI, compared to 83.3% and 73.5% for intensivists. The ROC curve analysis showed no significant difference between the two groups (p = 0.4).

Conclusion: This study highlights the limitation of reliance on clinical assessment alone in stratifying types of myocardial injury, and that additional non-invasive tests are required. While there was a higher pre-disposition for intensivists to score troponin elevation as type 1 MI, this was not statistically significant.

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