of 0, 6, 12 were 0, 13.1% and 36.7% respectively. Pearson's correlation coefficient showed a strong correlation ($r=0.82$, $95\%$ CI $0.70$ to $0.89$) between the Jeopardy Score and volume of hypoperfusion on CMR ($p<0.0001$) (Abstract 099 figure 1).

**Conclusion** There is a strong correlation between risky indices and volume of inducible ischaemia by dynamic 3D CMR whole heart perfusion imaging. 3D CMR perfusion imaging offers a non-invasive alternative method of detecting ischaemic burden and myocardium at risk for the purpose of serial studies, guiding revascularisation and risk stratification.

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**101** INCORPORATION OF STRESS ECHOCARDIOGRAPHY INTO AN ACUTE CHEST PAIN SERVICE PROVIDES EXCELLENT FEASIBILITY, EARLY TRIAGING AND ACCURATE RISK STRATIFICATION OF PATIENTS WITH SUSPECTED ACUTE CORONARY SYNDROME BUT NON-DIAGNOSTIC ECG AND NORMAL 12-H TROPONIN

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**Background** Acute chest pain accounts for a substantial proportion of patients attending the Emergency Department (ED). Initial investigations are frequently inconclusive and many patients thus require admission for further risk stratification. We have previously demonstrated the clinical benefits and cost savings of stress echocardiography (SE) compared to stress ECG for risk stratification of patients admitted with suspected acute coronary syndrome (ACS) but normal ECG and negative 12-h troponin. However, the feasibility of SE in routine clinical practice and its ability to predict hard cardiac events in this patient population is unknown.

**Methods** Consecutive patients admitted via the ED with chest pain and who underwent SE within 24 h of admission via our acute chest pain service were assessed for feasibility of SE, time to test and were followed-up for hard cardiac events (cardiac death and acute myocardial infarction—AMI).

**Results** Of 719 consecutive patients, 674 (94.6%) had diagnostic images at SE and were followed-up over 26 months. The median time to test for all patients was 1 day and median in-hospital length of stay for those with normal SE was also 1 day. There were 17 hard events (14 cardiac deaths and 3 AMI). Annualised hard cardiac event rate in the normal SE group (n=517, 73.6%) was 0.58% compared with 5.5% in the abnormal SE group (p=0.002). Cox regression analysis revealed that among clinical, ECG and SE variables, only abnormal SE [p=0.001, HR 4.02, 95% CI 1.73 to 9.36] and advancing age (10-year increase) [p=0.005, HR 1.70, 95% CI 1.18 to 2.44] were independent predictors of hard events in the multivariate model. Similarly, abnormal SE was also the strongest predictor of cardiac death [p=0.001, HR 4.52, 95% CI 1.81 to 11.3]. At any stage during follow-up, an abnormal SE carried at least a fourfold increased risk of either cardiac death or any hard event over a normal SE result.

**Conclusion** This is the first study to show that the incorporation of SE into a clinical acute chest pain service has excellent feasibility, provides rapid assessment with early triaging and accurate risk stratification of patients with suspected ACS but non-diagnostic ECG and negative 12-h troponin.

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**Abstract 101 Figure 1** Kaplan–Meier survival estimate of time to death.
lesions was 70% whereas specificity 98%. Regarding moderate to severe valvular stenosis sensitivity was 85.7% whereas specificity was 100%. Auscultation for presence of valvular abnormality (without specifying which valve or what kind of abnormality) revealed a 93.8% specificity and a 45.9% sensitivity.

Conclusions In the current study use of PHHE after brief, bed-side training greatly improved the diagnostic accuracy of medical students and junior doctors, over and above history, physical examination and ECG interpretation.

103 AORTIC INFLAMMATION IS REDUCED, AND PARALELLES CHANGES IN AORTIC STIFFNESS BY ANTI-TNF α THERAPY IN RHEUMATOID ARTHRITIS
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Background Rheumatoid arthritis (RA) is a systemic inflammatory condition associated with increased cardiovascular risk which is not fully explained by traditional risk factors. Endothelial dysfunction and increased aortic stiffness may mediate some of the increased risk. Additionally, there may be direct vascular inflammation which could directly accelerate atherosclerosis. We hypothesised that patients with RA exhibit a subclinical aortic vasculitis which can be reversed with anti-tumour necrosis factor α (TNF) therapy.

Methods The aortas and carotid arteries of 15 patients with severe rheumatoid arthritis were imaged before and after anti-TNF therapy using 18F-fluorodeoxyglucose positron emission tomography (FDG-PET) with CT co-registration. Tracer uptake was analysed in various arterial segments by measuring maximum standard uptake values (SUV) and subsequently corrected for blood uptake to obtain a target to background ratio (TBR). Carotid to femoral pulse wave velocity (PWV) as a measure of aortic stiffness, disease activity and inflammatory biomarkers were also measured.

Results Mean baseline aortic TBR was 2.07±0.20. Following anti-TNF α therapy, there was a significant reduction in abdominal aortic TBR (−0.18±0.27; p<0.03) and in the most diseased segment in the whole aorta (−0.48±0.59; p<0.01). TBR was also reduced in all other aortic segments and the proportion of hot slices (defined as TBR>1.9) was reduced by 31%, but these did not reach statistical significance. There was no change in carotid TBR following treatment. Aortic PWV was reduced by 0.43±1.0 m/s (p=0.1) and there was a significant correlation between a reduction in aortic PWV and abdominal TBR (R=0.57; p=0.03) and between aortic PWV and proportion of “hot” slices (R=0.66; p=0.01). There was a concomitant reduction in serum CRP (−8±12 mg/l; p<0.02) and disease activity (ΔAS20 –1.41±1.51; p=0.002).

Conclusions This study demonstrates for the first time that patients with RA have high aortic and carotid FDG uptake, suggesting subclinical vasculitis. Moreover, they exhibit a reduction in FDG uptake following anti-TNF α therapy, which correlated with a reduction in aortic stiffness. These results suggest that subclinical vasculitis could be the mechanism behind the increased cardiovascular risk and that effective treatment of inflammation may help to reduce the cardiovascular risk in this patient population.