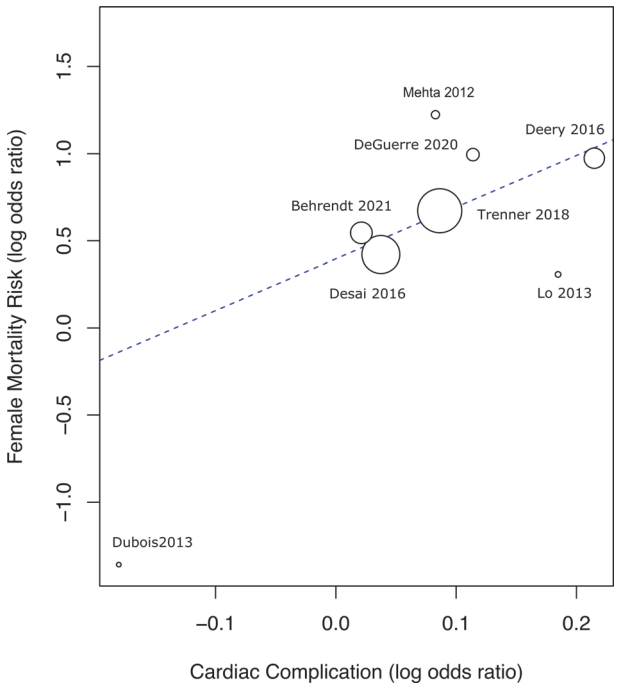

Abstract 35 Figure 2

of AAA (EVAR). (Cardiac: n= 8, βi =2.96 (se =1.27), p=0.02, tau2 = 0.00; renal: n=8, βi =2.50 (se =1.31), p=0.056, tau2 = 0.01).
Conflict of Interest None

36 TRANS-MYOCARDIAL EXTRACTION OF ENDOTHELIN-1 CORRELATES WITH INCREASED MICROVASCULAR RESISTANCE FOLLOWING PERCUTANEOUS CORONARY INTERVENTION

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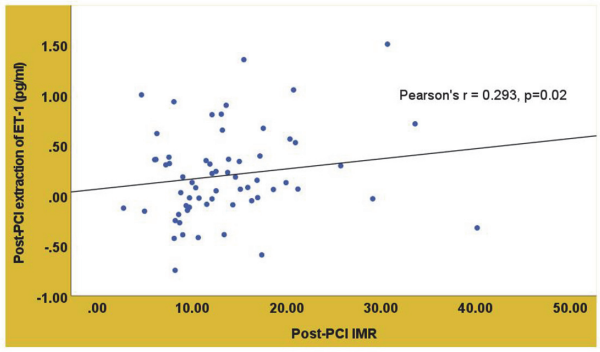
Introduction Coronary microvascular dysfunction (CMD) can persist following successful percutaneous coronary intervention (PCI). Endothelin-1 (ET-1) is a potent vasoconstrictor and may be an important mediator of CMD. We sought to assess the trans-myocardial gradient (TMG – coronary sinus minus coronary root levels) of ET-1 and its precursor - Big ET-1 and assess the correlation with pressure-wire indices of CMD: coronary flow reserve (CFR) and index of microvascular resistance (IMR).
Methods Paired blood samples from the aortic root and coronary sinus were collected before and after pressure wire guided PCI from patients with stable angina. Plasma was then analysed using specific enzyme linked immunosorbent assay (ELISA) for quantification of ET-1 and Big ET-1 and correlated with pressure-wire data.
Results Samples were analysed from 66 patients. Both mean ET-1 and Big ET-1 concentrations increased post-PCI in both the aorta (ET-1: 1.0±0.4 pg/ml to 1.4±0.4 pg/ml, p<0.0001 and Big ET-1: 2.8±1.3 pg/ml to 3.4±1.6 pg/ml, p<0.0001) and coronary sinus (ET-1: 1.0±0.3 pg/ml to 1.2±0.3 pg/ml, p=0.03 and Big ET-1: 3.2±1.7 pg/ml to 3.8±1.5 pg/ml,



Abstract 36 Figure 1

p=0.01). TMG extraction of ET-1 increased following PCI: 0.05±0.25 pg/ml vs.-0.20±0.41 pg/ml, p=0.01. In contrast, there was TMG release of Big ET-1 before and after PCI: 0.46±1.26 pg/ml vs. 0.38±1.03 pg/ml, p=0.52 **(figure 1)**. ET-1 extraction correlated with IMR post-PCI (Pearson's r = 0.293, p=0.02, **figure 2**). Patients with CFR<2 post-PCI demonstrated a numerical trend towards higher mean ET-1 extraction than those with preserved CFR post-PCI (0.30±0.51 pg/ml vs. 0.16±0.42 pg/ml, p=0.31) as did those with criteria for Type 4a Myocardial Infarction compared with those without (0.39±0.57 vs. 0.15±0.41, p=0.11).
Conclusions ET-1 and Big ET-1 significantly increase post-PCI. Trans-myocardial extraction of ET-1 increases post-PCI and correlates with post-PCI CMD.
Conflict of Interest none

37 INCIDENCE AND ONE YEAR OUTCOME OF PERIPROCEDURAL MYOCARDIAL INFARCTION FOLLOWING CARDIAC SURGERY: ARE THE UNIVERSAL DEFINITION AND SCAI CRITERIA FIT FOR PURPOSE?

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Introduction The diagnosis and clinical implication of periprocedural myocardial infarction (PPMI) following coronary artery bypass grafting (CABG) is contentious, especially given its importance in the interpretation of trial data. Two accepted definitions of PPMI yield discrepant results. Little is known about the association between the diagnosis of PPMI, using high sensitivity troponin (hs-cTn), and medium term mortality in patients who undergo CABG, either alone or in conjunction with another procedure.
Method Consecutive patients admitted to a cardiothoracic critical care unit (CCCU) over a six month period following open cardiac surgery had hs-cTnI assay performed on admission and every day for forty-eight hours, regardless of whether there was a clinical indication. Patients were categorised as PPMI using both the Universal Definition of MI (UDMI) and Society of Cardiovascular Angiography and Interventions (SCAI) criteria. Comorbidity data, surgical details and clinical progress in CCCU were recorded. One year mortality data were obtained from NHS Digital.