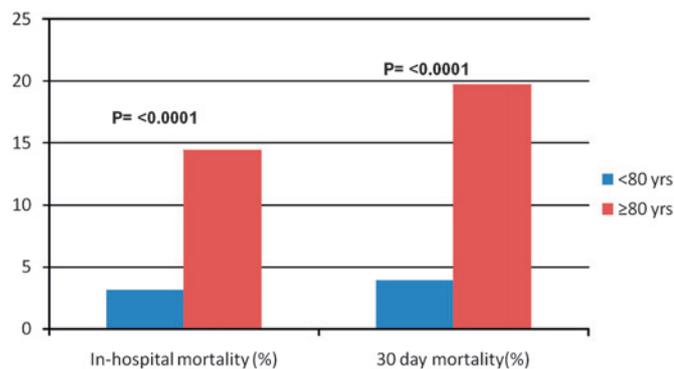


at PPCI in elderly patients such as SENIOR PAMI (Grines, 2005) and TRIANA (Bueno, 2009) the minimum age for inclusion was 70 yrs and 75 yrs respectively. With an ageing population in the western world, about 20% of patients admitted for suspected STEMI are ≥ 80 yrs. We evaluated the outcome of PPCI in patients ≥ 80 yrs who were admitted to our unit with STEMI.

Methods Our PPCI service was started in September 2009 and we analysed all the patients who were ≥ 80 yrs presenting to the PPCI service between September 2009 and September 2010 (13 months). Prospectively entered data were obtained from our dedicated cardiac service database system (Philips CVIS). Mortality data were obtained from the summary care record (SCR) database. Follow-up data were obtained from patients' respective district general hospitals and general practitioners medical records.

Results Of the 998 patients who were admitted to our unit for primary PCI for suspected STEMI during the study period, 183 (18.3%) were ≥ 80 yrs of age. After excluding 51 patients (27.9%) who did not undergo PPCI, we included 132 (70.1%) patients for analysis. Of those who were included in the study ($n=132$, 63 female), the mean age was 85 ± 3.95 yrs (range 80–99 yrs, median 85 yrs). There were 20 diabetics (15.2%) and 39 (29.5%) had previous myocardial infarction. Ten patients (7.6%) were in cardiogenic shock on arrival of which 9 (90%) had an Intra aortic balloon pump (IABP). The infarct related vessel was the right coronary in 42.4% and left anterior descending in 37.1%. Drug eluting stents were used in 40.2% of patients. In-hospital and 30-day mortality was 14.4% and 19.7% respectively. There was a significant difference in the mortality between patients age < 80 yrs and those ≥ 80 yrs (Abstract 45 figure 1). In patients ≥ 80 yrs, mortality and bleeding risk increased markedly with advancing age (Abstract 45 table 1).



Abstract 45 Figure 1

Abstract 45 Table 1

%	80–84 yrs (N=62)	85–89 yrs (N=51)	≥ 90 yrs (N=19)
Inhospital mortality	9.7	15.7	26.3
30-day mortality	14.5	23.5	26.3
30-day MI	3.2	2.0	0
30-day CVA	1.6	0	0
Major bleeding requiring blood transfusion	1.6	3.9	10.5

Conclusion This study clearly demonstrates a significant mortality difference between patients aged < 80 yrs and those ≥ 80 yrs treated with PPCI. Our 30-day mortality outcome in patients ≥ 80 yrs (19.7%) was similar to the subgroup analysis of the PPCI arm in similar SENIOR-PAMI patients (19%). In the same analysis, the thrombolytic group had a lower (16%) mortality. Further studies are required to determine whether PPCI should be routinely used in very elderly patients presenting with STEMI.

46 PROGNOSTIC VALUE OF BASELINE RENAL FUNCTION ON LONG TERM OUTCOME IN PATIENTS UNDERGOING PRIMARY PERCUTANEOUS CORONARY INTERVENTION FOR ST-ELEVATION MYOCARDIAL INFARCTION

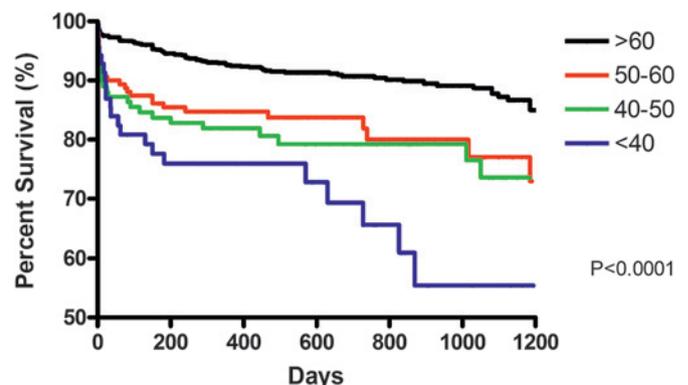
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Background Renal impairment is associated with increased cardiovascular mortality following acute coronary syndromes (ACS), however there is limited data assessing this relationship in the context of primary PCI and whether it exists with other major adverse cardiovascular events.

Methods Clinical information was analysed from a prospective data base on 2310 STEMI patients who underwent primary PCI between January 2004 and May 2010 at a London centre. Information was entered at the time of procedure and outcome assessed by all-cause mortality information provided by the Office of National Statistics via the BCIS/CCAD national audit. Estimated glomerular filtration rate (eGFR) was calculated using the modified diet in renal disease equation and patients were divided into groups based on eGFR (< 40 , 40–50, 50–60, > 60 ml/min/1.73 m²). 3-year composite of MACE (death, reinfarction, stroke and target vessel revascularisation) were compared between groups.

Results The average eGFR in all patients was 73.40 ± 23.37 (95% CI 72.25 to 74.56) ml/min/1.73 m². The prevalence of coexisting risk factors (hypertension, diabetes mellitus, hypercholesterolaemia), previous MI, previous CABG and cardiogenic shock were higher among patients with reduced eGFR. There was a progressive increase in MACE with declining eGFR (OR=4.84, 95% CI 2.94 to 7.96, for comparison between the highest and lowest eGFR groups). See Abstract 46 figure 1. After adjustment for baseline characteristics including age, diabetes and cardiogenic shock renal function based on the GFR at admission remained a strong independent predictor of outcome.



Abstract 46 Figure 1 All MACE after PCI for STEMI.

Conclusion Baseline renal dysfunction in patients undergoing primary PCI is associated with an increased risk for combined death, re-infarction and recurrent angina. This risk increases linearly with declining eGFR.

47 CARDIOVASCULAR EVALUATION OF ENGLISH PREMIERSHIP RUGBY PLAYERS

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Introduction Recent experience of pre-participation cardiovascular evaluation (PPCE) in Italian athletes demonstrates a significant

reduction in mortality from cardiomyopathies and cardiac conduction disorders. Although PPCE is endorsed by large medical and sporting organisations, including the European Society of Sports Cardiology, the International Olympic Committee and FIFA, the state health system in the UK (and many other Western countries) does not support cardiovascular evaluation of athletes. Certain elite sporting organisations in the UK mandate PPCE in all athletes prior to competition. In 2010 the English Premier Rugby league introduced formal PPCE in all competing players.

Methods Athletes participating in the English Premiership Rugby underwent PPCE with a structured clinical questionnaire and 12-lead ECG. Trans-thoracic echocardiogram (TTE) and additional investigations were performed where indicated.

Results A total of 606 players were assessed (mean age 22.9 years; range 15–37). Of these, 45 (7.4%) required TTE (35 (5.7%) due to ECG abnormalities; 5 (0.08%) due to family history of sudden death; 5 (0.08%) due to symptoms). ECG abnormalities warranting TTE included right axis deviation (n=4), left axis deviation (n=17), right bundle branch block (n=3), right ventricular hypertrophy (n=1), abnormal T wave inversion (n=5) and prolonged QT (n=1). Six of the 45 subjects demonstrated mild changes on TTE (markedly dilated LV cavity (n=3), mitral regurgitation (n=1), pulmonary stenosis (n=1), dilated aortic root (n=1)), requiring serial surveillance. Five demonstrated abnormalities on TTE and/or ECG that warranted referral for further evaluation including exercise stress test (n=5), 24-h ECG (n=5) and cardiac MRI (n=3). The reasons for these tests included possible arrhythmogenic right ventricular cardiomyopathy (n=3), suspicion of hypertrophic cardiomyopathy (n=1) and QT prolongation on ECG (n=1). None of the players exhibited a cardiac disorder that warranted disqualification from sport. Overall 7.4% of athletes required further investigation following initial ECG, and 1.8% required further tests following TTE. False positive rate was 5.6%.

Conclusion Cardiovascular evaluation of British rugby players with a structured questionnaire and ECG resulted in clearance of 92.6% following initial tests, and 5.6% were reassured after TTE. Only 1% players required surveillance echocardiograms and 0.8% were referred for further diagnostic evaluation. False positive rate was 5.6%. The results indicate that PPCE carried out in an expert setting results in a relatively small number of athletes requiring further tests, and a low false positive rate.

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DARBEPOETIN ENHANCES ENDOTHELIAL-DEPENDENT VASOMOTOR FUNCTION IN PATIENTS WITH STABLE CORONARY ARTERY DISEASE ONLY AFTER PRECEDING ISCHAEMIA-REPERFUSION

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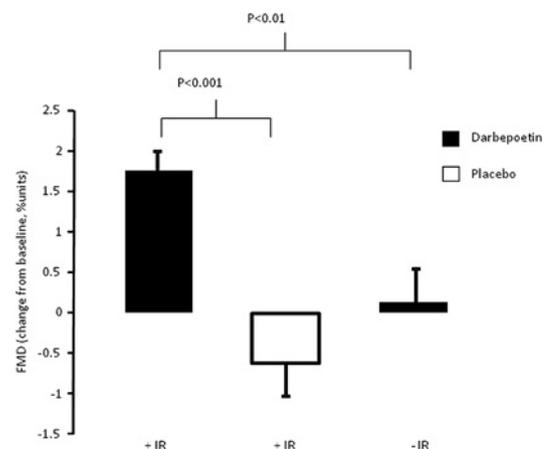
Background Vasoprotective effects of erythropoietin in animal models are mediated by endothelium-derived nitric oxide (NO) and/or mobilisation of endothelial progenitor cells (EPC) and may be

enhanced by ischaemia; whether they are present in humans is unknown. We examined whether the erythropoietin analogue darbepoetin improves flow mediated dilatation (FMD), a measure of endothelium-derived NO, and whether this is influenced by preceding ischaemia-reperfusion.

Methods 36 patients (50–75 years) with stable coronary artery disease were randomised to receive a single dose of darbepoetin 300 µg or saline placebo. Immunoreactive erythropoietin was measured by an enzyme linked immunospecific assay. FMD was measured at the brachial artery using high resolution ultrasound. CD34+/VEGFR2+/133+ circulating EPC were enumerated by flow cytometry. Measurements were made immediately before darbepoetin/placebo and at 24 h, 72 h and 7 days. At 24 h FMD was repeated after 20 min ischaemia-reperfusion of the upper limb. A further group of 11 patients were studied according to the same protocol, all receiving darbepoetin, with omission of forearm ischaemia-reperfusion at 24 h.

Results Immunoreactive erythropoietin peaked at 24 h in the darbepoetin group (median value of 724 U/l (IQR 576–733 U/l), compared to 12 U/l (IQR 9–21 U/l) in the placebo group) and remained elevated at approximately 500 fold baseline at 72 h. FMD did not differ significantly between groups at 24 h (before ischaemia-reperfusion). At 72 h, (48 h after ischaemia-reperfusion) FMD increased from baseline in the darbepoetin group but not in the placebo group so that FMD (and change in FMD from baseline) was significantly greater in the darbepoetin group (change from baseline 1.7±0.3% and -0.6±0.4% in darbepoetin and placebo groups respectively, p<0.001). The increase in FMD at 72 h after darbepoetin and ischaemia-reperfusion at 24 h was significantly greater than that without preceding ischaemia-reperfusion (p<0.01). A ~20% increase in CD133+/VEGFR2+ cells after darbepoetin was temporally dissociated with the increase in FMD.

Conclusions Preceding ischaemia-reperfusion is required for darbepoetin to enhance endothelial function, possibly by increasing expression of the erythropoietin receptor and by a mechanism likely to involve Akt/NO rather than circulating EPC.



Abstract 48 Figure 1 Change from baseline in FMD at 72 h, 48 h after ischaemia-reperfusion (+IR), after placebo and darbepoetin (study 1) and after darbepoetin without preceding ischaemia-reperfusion (-IR, study 2).

Abstract 48 Table 1 Endothelial function and EPC

Endothelial function	Placebo Baseline	Placebo 24 h	Placebo 72 h	Placebo 7d	Darbepoetin Baseline	Darbepoetin 24 h	Darbepoetin 72 h	Darbepoetin 7d
FMD%	3.5±0.80	3.4±0.73	2.9±0.63	3.4±0.75	3.5±0.92	4.4±0.97	5.2±0.9***	3.7±0.61
Progenitor cells								
CD133+/VEGFR2+	110±17.6	117±19	101±19	123±21	146±13	180±13*	180±11*	182±16*
CD34+/VEGFR2+	4.1±0.6	3.9±0.5	3.1±0.6	4.7±0.8	8.7±3.07	11.1±3.9	7.1±1.4	13.3±4.5
CD133+/CD34+	17.3±2.9	17.0±1.9	17.7±3.6	20.4±3.2	23.6±2.4	29.3±4*	31.6±4.6*	29.9±4.8*

Values are means±SE.

* p<0.05, ***p<0.001