slowing dramatically from 2006 to 2008. JointPoint regression analysis of different age groups demonstrates that the slower rate of decline from 2006 may be due to stubbornly high numbers of deaths in the 35–44 age group. Lastly the National figures on mortality from CHD are shown to be misleading as many people are still dying from CHD just when they have crossed the 75-year old exclusion criteria; as a result a delay in mortality is presented as prevention of mortality from CHD.

**Discussion** There is a danger that previous successes are being offset by high rates in the younger cohorts, and that the overall trend may be eventually be reversed. There is still work to be done in reducing risk factors and also applying treatments that have had a proven positive impact (such as revascularisation) more effectively. Statistically significant changes in declining CHD mortality rates.

**Future work** This 10 000 word report formed the basis of a funding application to the British Heart Foundation for a follow-up to the United Kingdom Heart Attack Study.

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Abstract 18 Figure 1 All cause mortality after PCI for STEMI.

**Conclusion** Patients presenting with anaemia undergoing primary PCI appear at significantly higher risk of an adverse outcome. This risk increases further in population receiving RBC transfusions during index hospitalisation.

**Abstract 18**

**Patients presenting with anaemia undergoing primary PCI appear at significantly higher risk of an adverse outcome**


**Background** Previous studies have demonstrated a relationship between pre-existing anaemia and inpatient mortality after percutaneous coronary intervention (PCI). There is limited data looking at the impact of baseline Haemoglobin and long term outcome after primary PCI.

**Methods** Clinical information was analysed from a prospective database on 2357 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. Anaemia was defined according to WHO definition of Hb greater than or equal to 12 g/dl for females and 15 g/dl for males.

**Results** 471 (20%) patients were anaemic at presentation. The anaemic cohort, were older (72.2 vs 62.4, p<0.0001), had higher incidence of diabetes (27% vs 15%, p<0.0001), hypertension (42 vs 35%, p=0.01), hypercholesterolaemia (40 vs 30%, p=0.007), previous PCI (15 vs 7%, p=0.01), and previous MI (23% vs 12%, p<0.0001). There were similar incidences of three-vessel disease and cardiogenic shock. Over a 3-year follow-up period there was significantly higher all cause mortality in the anaemic group compared to the normal Hb group (20.4% vs 13.5%, p<0.0001). See Abstract 18 figure 1. After adjusting for comorbidities, anaemia remained an independent predictor of long-term adverse outcome (OR=2.4, 95% CI=1.1 to 3.7, p<0.001). Patients with baseline anaemia who received a blood transfusion were significantly more likely to suffer an adverse outcome than those that did not receive a transfusion (21% vs 6%, p<0.0001).

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Abstract 17 Figure 2

**Patients presenting with anaemia undergoing primary PCI appear at significantly higher risk of an adverse outcome**


**Background** Previous CABG were excluded. 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. Anaemia was defined according to WHO definition of Hb greater than or equal to 12 g/dl for females and 15 g/dl for males.

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up to 1-year of follow-up with the lowest rates of events in the SR group. However after 3 years MACE rates are significantly increased in the COR group (24%) but were similar in the CR (18%) and SR (17%) groups. See Abstract figure 1. MACE rates were driven mainly by death in the CR with high rates of TVR in the COR and SR groups. See Abstract figure 2.

Abstract 19 Table 1

<table>
<thead>
<tr>
<th></th>
<th>COR N = 638</th>
<th>SR N = 180</th>
<th>CR N = 263</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>64.77</td>
<td>64.32</td>
<td>64.32</td>
<td>0.144</td>
</tr>
<tr>
<td>Gender (female)</td>
<td>156 (23.7%)</td>
<td>13 (13.0%)</td>
<td>74 (27.9%)</td>
<td>0.0114</td>
</tr>
<tr>
<td>Ethnicity (Caucasian)</td>
<td>441 (67.0%)</td>
<td>79 (79.0%)</td>
<td>185 (69.8%)</td>
<td>0.0511</td>
</tr>
<tr>
<td>Previous MI</td>
<td>109 (16.6%)</td>
<td>11 (11.0%)</td>
<td>36 (13.6%)</td>
<td>0.2414</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>15 (2.3%)</td>
<td>2 (2.0%)</td>
<td>3 (1.1%)</td>
<td>0.5231</td>
</tr>
<tr>
<td>Previous PCI</td>
<td>83 (12.6%)</td>
<td>5 (5.0%)</td>
<td>23 (8.7%)</td>
<td>0.031</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>129 (19.6%)</td>
<td>16 (16.0%)</td>
<td>55 (20.8%)</td>
<td>0.5932</td>
</tr>
<tr>
<td>Hypertension</td>
<td>312 (48.1%)</td>
<td>40 (40.0%)</td>
<td>91 (41.2%)</td>
<td>0.1205</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>269 (41.5%)</td>
<td>37 (37.0%)</td>
<td>92 (41.8%)</td>
<td>0.7751</td>
</tr>
<tr>
<td>GPIIb/IIIa Inhibitor</td>
<td>572 (87.7%)</td>
<td>93 (93.0%)</td>
<td>231 (89.5%)</td>
<td>p &lt; 0.0001</td>
</tr>
</tbody>
</table>

Abstract 19 Figure 1 Comparison of MACE between multivessel disease.

Abstract 19 Figure 2 Breakdown of MACE at 5 years.

Conclusions Culprit vessel-only angioplasty was associated with the highest rate of long-term MACE compared with multivessel treatment. Patients scheduled for staged revascularisation experienced a similar rate of MACE to patients undergoing complete simultaneous treatment of non-IRA.

Abstract 20 Figure 1

Conclusion We have described for the first time an aspirin-independent increase in AA-induced platelet aggregation, and (ii) an unexpected and significant rise in AA-induced platelet aggregation. TXB2 was consistently suppressed confirming inhibition of COX by aspirin.

Abstract 20 Figure 1

Conclusion A clustering of adverse events, in particular stent thrombosis (ST) has been observed following clopidogrel cessation 1-year after drug-eluting stenting (DES), the aetiology of which is poorly understood. We investigated the effect of withdrawing clopidogrel in DES patients using a simple, rapid, reproducible near-patient platelet function test known as short Thrombelastography (s-TEG) that has been developed and validated by this group.

Methods 33 patients on aspirin and due to stop clopidogrel at 1 year following DES were investigated. Venesection was performed at (i) 4 weeks and 24 h pre clopidogrel cessation (ii) 24 h, 48 h, 1, 2 and 4 weeks post clopidogrel cessation. At all time-points, platelet reactivity was determined using s-TEG and thromboxane (TX) B2, IL-6, CD40 ligand and high sensitivity CRP were measured.

Results Clopidogrel cessation produced (i) a predictable increase in ADP-induced platelet aggregation, and (ii) an unexpected and significant rise in AA-induced platelet aggregation. TXB2 was consistently suppressed confirming inhibition of COX by aspirin.

Abstract 20 Figure 1

21 INFLUENCE OF FRACTIONAL FLOW RESERVE MEASUREMENT ON TREATMENT-DECISIONS IN PATIENTS WITH RECENT ACUTE NON-ST ELEVATION MYOCARDIAL INFARCTION

doi:10.1136/heartjnl-2011-300198.21

Introduction Non-ST elevation acute myocardial infarction (NSTEMI) is the most common form of acute coronary syndrome and has a relatively poor prognosis. Visual interpretation of the coronary angiogram is the standard approach to guide treatment decisions in patients with recent acute NSTEMI. The aim of our study was to determine whether measurement of coronary pressure derived fractional flow reserve (FFR), compared to coronary angiography alone, might influence treatment decisions.