percutaneous coronary interventions (PCI). The incidence is increasing and to date outcomes are not well characterised, though there is a suggestion that there is a worse clinical outcome. We therefore sought to compare STEMI caused by ST vs de novo coronary thrombosis to evaluate procedural risk and clinical outcome.

Methods Clinical information was analysed from a prospective database on 2421 patients who underwent Primary PCI following STEMI between October 2003 and May 2010 at a London centre. Information was entered at the time of procedure, diagnosis of stent thrombosis made at the time by primary operator and outcome assessed by all-cause mortality information provided by the Office of National Statistics via the BCIS CCAD national audit.

Results Stent thrombosis (ST) accounted for 7.4% (180/2421) of all STEMI with a frequency that has increased over time (5.4% in 2005 to 9.8% 2009). ST occurred early (0–30 days) in 36% (65/180), late (30 days–1 year) in 22% (40/180) and very late (>1 year) in 42% (75/180) of pts. Drug-eluting stents (DES) accounted for 48% of ST overall and 70% over the past 3 years. Proposed mechanisms included premature discontinuation of anti-platelets (11%), under-deployment of previous stent insertion (22%) and underlying prothrombotic conditions (eg, SLE) (6%). Pts with ST compared to native artery occlusion had higher rates of previous MI (53.9% vs 11%, $p<0.0001$) and incidence of multi-vessel disease (59.8% vs 51.7%, $p=0.04$). There was no difference in age, diabetes or cardiogenic shock. See Abstract 16 table 1. Infarct size based on peak enzyme markers was similar (2.5 vs 2.2, $p=0.45$). In patients with ST, angiographic success (postprocedural Thrombolysis In Myocardial Infarction grade III flow) was worse than in patients with de novo STEMI (87.2% vs 93.7%, $p=0.02$). Pts with STEMI due to ST had higher in-hospital MACE (11% vs 3%, $p=0.0001$), MACE at 30 days (19% vs 6%, $p<0.0001$), this persisted up to 3 years (41% vs 12%, $p<0.0001$). See Abstract 16 figure 1. MACE was driven by higher rates of MI (7% vs 2%, $p<0.0001$), TVR (14% vs 3%, $p<0.0001$) and death (18% vs 6%, $p=0.0001$). After adjusting for comorbidities, stent thrombosis was an independent predictor of long-term adverse outcome (OR=2.1, 95% CI=1.3 to 2.8, $p<0.001$).

Abstract 16 Table 1

	AST (n=180)	No AST (n=2241)	Significance (p value)
Age	63.9±18	62.9±4	0.406
Multi-vessel disease	101 (59.76%)	1011 (51.74%)	0.045
Previous MI	96 (53.93%)	246 (10.96%)	<0.0001
Previous CABG	9 (5.06%)	60 (2.67%)	0.020
Diabetes mellitus	46 (25.84%)	407 (18.14%)	0.061
Hypertension	101 (56.74%)	975 (43.45%)	0.002
Hypercholesterolaemia	108 (60.67%)	768 (34.30%)	<0.0001
Cardiogenic shock	108 (60.67%)	768 (34.30%)	<0.0001

Abstract 16 Figure 1

Conclusion Primary PCI for treatment of ST is less effective, and these patients are at increased risk for in-hospital and long-term mortality compared with patients undergoing primary PCI due to de novo STEMI.

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SUDDEN CARDIAC DEATH AND ACUTE MYOCARDIAL INFARCTION: HOW HAS THE PICTURE CHANGED?

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¹G Mole, ²D Watson, ³C Davidson. ¹Brighton & Sussex Medical School, Brighton, UK; ²Department of Informatics, University of Sussex, Brighton, UK; ³Brighton & Sussex University Hospital, Brighton, UK

Introduction Coronary heart disease (CHD) is a major burden worldwide, particularly in economically developed countries such as the UK. Between 1980 and 2000, deaths from CHD fell by over 50%, and have continued to fall. The cost of CHD manifests itself in mortality, disability and economic impact: this should be looked at in the context of a disease that is preventable.

Methods Data from death certificates and studies with strict clinico-pathological criteria on mortality from CHD were accessed. These were analysed in terms of hospital admissions, revascularisation rates and index of multiple deprivation (IMD). Trends in mortality overall and for different age groups were analysed over time to determine how the picture has changed and predict what may happen going into the future. The mortality rates from the catchment area of the Royal Sussex County Hospital and York Hospital were analysed to assess reliability of official figures against strict clinico-pathological inclusion criteria. A range of statistical tests including, linear regression, ANOVA and JoinPoint regression were employed.

Results Between 1993 and 2008 deaths from CHD have fallen by over 50%. The decline has been greater in older age groups particularly the 65–74 age group (the oldest age group analysed). Comparison with data from studies with strict clinico-pathological criteria showed this to be the age group in which official statistics were least accurate. Regression analysis demonstrated that a higher IMD is associated with increased mortality from CHD ($r^2=0.69$, $p<0.001$). Increased admission rates was not significantly associated with decreased mortality from CHD ($r^2=0.004$, $p=0.239$). Increased revascularisation was significantly associated with decreased mortality from CHD ($r^2=0.99$, $p<0.001$). JoinPoint regression analysis shows a constant rate of decline in mortality from 1993 to 2001 which then decreases faster between 2001 and 2006 before

Abstract 17 Figure 1

slowing dramatically from 2006 to 2008. JoinPoint 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.

Abstract 17 Figure 2

18 PATIENTS PRESENTING WITH ANAEMIA UNDERGOING PRIMARY PCI APPEAR AT SIGNIFICANTLY HIGHER RISK OF AN ADVERSE OUTCOME

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K R Rathod, D A Jones, B Rathod, A Graham, E Sammut, S Gallagher, J Behar, A K Jain, C Knight, A Mathur, A Wragg, R Amersey. *Barts and the London NHS trust, London, UK*

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 13 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 (13 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%, $p<0.0001$).

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.

19 TREATMENT OF MULTIVESSEL CORONARY ARTERY DISEASE IN PRIMARY PCI FOR ST ELEVATION MYOCARDIAL INFARCTION: CULPRIT ONLY REVASCUARISATION IS ASSOCIATED WITH HIGHER MACE RATES

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K S Rathod, L A McGill, E Sammut, V S Rathod, D A Jones, R Weerackody, A Jain, C Knight, A Mathur, A Wragg. *London Chest Hospital NHS Trust, London, UK*

Background Multi-vessel disease occurs in 40%–65% of patients undergoing Primary PCI for STEMI and is associated with adverse prognosis. Contemporary guidelines recommend treating the infarct related artery alone (culprit) during the urgent procedure. There is limited data comparing outcomes of complete with infarct-related artery (IRA)-only revascularisation in primary PCI for STEMI with few studies including the option of later date elective procedures for the other lesions (staged revascularisation). We therefore sought to clarify the outcome of patients with multi-vessel disease undergoing primary PCI dependent on management strategy.

Methods Clinical information was analysed from a prospective data base on 2131 STEMI patients who underwent Primary PCI between January 2004 and May 2010 at a London centre. Patients with 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. Patients were split into three different treatment groups: culprit vessel angioplasty-only (COR group); staged revascularisation (SR group) and simultaneous treatment of non-IRA (CR group). The primary end point used was major adverse cardiac events (MACE), defined as death, myocardial infarction (MI), stroke and target vessel revascularisation (TVR).

Results There were 963 (45%) consecutive patients with STEMI and multivessel CAD undergoing primary angioplasty. There were similar baseline characteristics between the 3 groups, aside from cardiogenic shock, which was significantly higher in the complete revascularisation group. See Abstract 19 table 1. At 30-days of follow-up, 23/263 (9%) patients in the CR group experienced at least one major adverse cardiac event (MACE), 1 (1%) in the SR group and 35 (5%) in the COR group, $p=0.01$. This trend continued