TIMI flow pre- and post-procedure, smoking history, cardiogenic shock, previous MI, CABG or PCI) were compared between groups. Statistical comparison was made using χ^2 test for categorical data and t test for continuous data.

Results 829 patients underwent Primary PCI between April 2008 and November 2011. 530 had single vessel disease. 299 patients had MVD; 193 underwent single vessel angioplasty to the infarct related artery, 71 patients underwent multivessel PCI at time of primary PCI and 35 had a further PCI either during that admission or post-discharge. Mean follow-up was 524±347 days. Overall in-hospital mortality was 3.5% and mortality to follow-up was 8.5%. Baseline characteristics were similar between groups with the exception of previous MI and Previous CABG which was significantly higher among patients with MVD & single vessel PCI. Cardiogenic shock was also higher among patients with MVD & multivessel PCI (19% vs SVD 3.8%, p<0.05). In-hospital mortality between the three groups was similar; SVD=3%, MVD& single vessel PCI=4.9%, MVD & multivessel PCI=2.9%, p=0.203. Overall mortality to follow-up between the three groups was also similar (Abstract 131 table 1, p=0.20). Exclusion of cardiogenic shock demonstrated a trend towards improved overall mortality in patients undergoing multivessel PCI (Abstract 131 table 2, p=0.17). Looking exclusively at patients post-discharge, a similar trend towards improved mortality with multivessel PCI was seen (SVD=5.1%, MVD with single vessel PCI=4.97%, MVD disease with multivessel PCI=1.94%; p=0.372).

Abstract 131 Table 1

	Total number	Number of deaths to follow-up	Mortality (%)
Single vessel disease	530	49	9.2
Multivessel disease: single vessel PCI	196	20	10.4
Multivessel disease: multivessel PCI	103	11	10.4

Abstract 131 Table 2

	Total number excl. cardiogenic shock	Number of deaths to follow-up	Mortality (%)
Single vessel disease	509	44	8.6
Multivessel disease: single vessel PCI	182	17	9.3
Multivessel disease: multivessel PCI	97	5	5.2

Conclusions These findings show a trend towards lower mortality post-discharge with mutlivessel PCI carried out in STEMI patients with MVD, suggesting consideration should be given to complete revascularisation in haemodynamically stable STEMI patients with multivessel disease, either during acute admission or post-discharge. The results of the ongoing UK multicentre randomised trial CVLPRIT will address this important aspect of managing patients with STEMI and multivessel disease.

132 COMBINED PRASUGREL AND BIVALIRUDIN TREATMENT DURING PRIMARY PCI OFFERS A SAFE AND EFFECTIVE STRATEGY IN ST-ELEVATION MI

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Introduction Successful treatment of STEMI requires early diagnosis and urgent passivation of the culprit lesion by antithrombotic therapy and mechanical revascularisation. A delicate balance exists between the risk of thrombosis and bleeding, consequently the choice of antithrombotic therapy is critical. Bivalirudin, a direct thrombin inhibitor, has been shown to offer a superior safety profile over heparin and GP2b3a inhibition in STEMI (Horizons-AMI). Similarly, the novel P2Y12-receptor inhibitor, Prasugrel, achieves faster and more consistent platelet inhibition than clopidogrel with improved clinical outcomes in STEMI (TRITON-TIMI 38). Although untested in a randomised clinical trial, the combination of bivalirudin and prasugrel for the treatment of STEMI appears to offer fast and effective inhibition of thrombosis with an acceptable bleeding profile. A randomised trial is in progress (BRAVE 4) but data will not be available until 2013.

Abstract 132 Table 1

	All patients, n=533	Group A, n=345	Group B, n=188
Age (median)	63.6	58.8	72.3
Male sex (%)	78.8	82.9	71.3
Diabetes (%)	11.1	7.8	17.0
Hypertension (%)	30.6	26.1	38.8
Hypercholesterolaemia (%)	30.6	30.1	31.4
Current smoking (%)	33.2	39.4	21.8
Previous MI (%)	8.6	7.5	10.6
Previous CABG (%)	2.3	1.2	4.3
Previous PCI (%)	6.4	5.5	8.0

Methods Since June 2010 our preferred treatment strategy for patients presenting with STEMI has been pre-loading with prasugrel 60 mg and peri-procedural bivalirudin (0.75 mg/kg bolus followed by an infusion of 1.75 mg/kg/h). Patients >75 yrs, <65 kg, with a history of cerebrovascular accident, pre-treated with clopidogrel or intubated without NG tube access were excluded and received treatment according to operator preference. The preferred access site was the radial artery and all operators were experienced in performing percutaneous coronary intervention from this route. Patients were discharged on lifelong aspirin and prasugrel 10 mg for 12 months. Additionally, patients routinely received statin, ACE inhibitor and β-blocker therapy. Consecutive patients were enrolled over a 12-month period commencing in July 2010.

Results In total 533 patients presented with STEMI and underwent treatment with PPCI in the study period. Of these 345 received a prasugrel loading dose and bivalirudin treatment (Group A). The remaining 188 patients received alternative therapy (Group B). Baseline demographics (Abstract 132 table 1) differ significantly between groups due to the criteria used for drug regimen selection. Procedural outcomes are summarised in Abstract 132 table 2. Procedural success was achieved in 97.6% of patients, using radial access in 79.9%. Bleeding occurred in 1.9% of all patients (Group A 1.2%). Stent thrombosis occurred in 1.1% of patients and no definite

Abstract 132 Table 2

	All patients n=533	Group A n=345	Group B n=188
All Bleeding (%)	1.9	1.2	3.2
Stent Thrombosis (%)	1.1	0.9	1.6
Definite ST (%)	0.6	0.6	0.5
Probable ST (%)	0.6	0.3	1
Reinfarction (%)	0.6	0.6	0.5
Reintervention (%)	3.9	3.8	4.3
Stroke (%)	0.8	0.3	1.6
In-hospital mortality (%)	5.3	2.6	10.1
30-day mortality (%)	6	2.6	12.2

early stent thrombosis was observed in Group A. The 30-day death rate was 6% with 2.6% in Group A.

Conclusions Use of bivalirudin and prasugrel in the acute treatment of STEMI demonstrates excellent efficacy and safety. The Horizons-AMI data suggested a risk of early stent thrombosis in the absence of heparin, however, our heparin-naive cohort were free of this complication. Furthermore, our combined strategy of anti-thrombotic therapy and preferred radial access maintains very low access-site complication rates. Randomised trial data confirming this strategy is awaited.

133 CORONARY WAVE INTENSITY: A NOVEL INVASIVE TOOL FOR PREDICTING MYOCARDIAL VIABILITY FOLLOWING ACUTE CORONARY SYNDROMES

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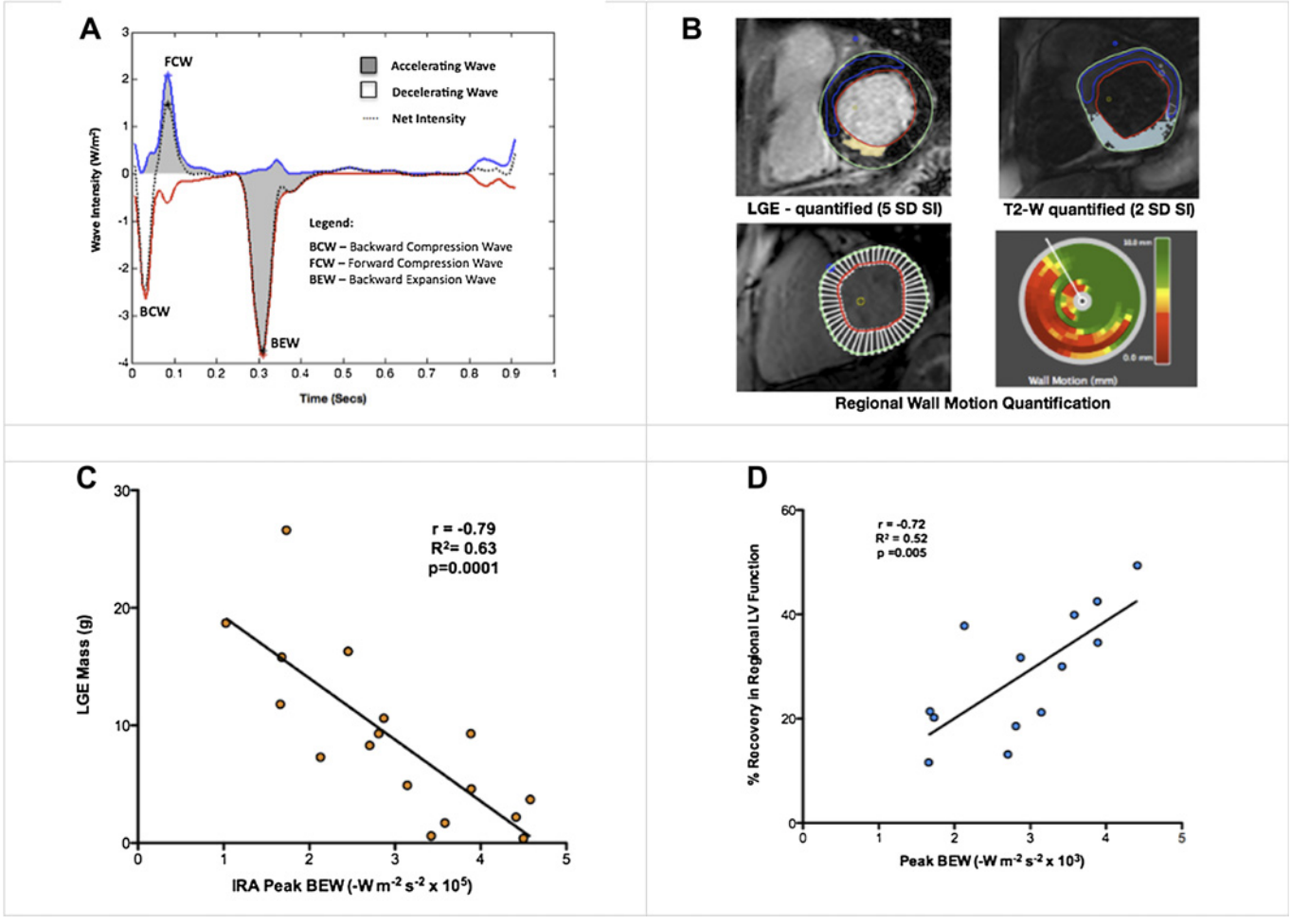
Introduction Wave intensity analysis (WIA) uses simultaneous changes in intracoronary pressure and flow to characterise energy transfer within the coronary circulation. In normal hearts, flow is predominantly driven by a microcirculatory-derived, diastolic phase, backward expansion wave (BEW) and aortic-derived, systolic phase, forward compression wave (FCW) (Abstract 133 figure 1A). Regional changes in contraction and microvascular function following acute coronary syndromes (ACS) may affect these waves, but the utility of WIA in this setting remains unknown.

Methods Patients were included 2–7 days after presenting with Non-ST elevation myocardial infarction (NSTEMI). Those with prior MI, haemodynamic instability or coronary artery disease unsuitable for PCI were excluded. Left ventricular ejection fraction (LVEF) and late-gadolinium enhancement (LGE) were assessed by cardiac MRI. Subsequently, intra-coronary (IC) pressure and Doppler measurements were taken in the infarct-related artery (IRA) and a remote reference vessel (REF), during IC adenosine-induced hyperaemia. Blinded WIA was performed offline. Regional left ventricular recovery following percutaneous coronary intervention (PCI) was quantitatively assessed by MRI, at 3 months (Abstract 133 figure 1B). Pearson Regression analysis was performed to assess the statistical relationship between WIA and size of infarction and recovery in function, following PCI.

Results 18 patients (57±11 yrs) 88±51 h post-myocardial infarction were enrolled. 12-h Troponin T, LVEF and % left ventricular infarct (LGE) mass were 1.53±1.40 μg/l, 56%±11.1% and 8.9%±6.0% respectively. BEW and FCW energies predominated with the mean peak WI being −3.17 and +2.80 ($W/m^2/s^2 \times 10^5$) respectively. IRA BEW energy strongly correlated with regional left ventricular recovery (R^2=0.53, p=0.005) and was inversely correlated to infarct mass (R^2=0.63, p<0.0001) (Abstract 133 figure 1C). REF BEW correlated weakly with LGE (R^2=0.34, p=0.03).

Conclusion The microcirculatory backward expansion wave energy correlates with the magnitude and location of infarction and is



Abstract 133 Figure 1 (A) Typical Coronary Wave Intensity Profile. (B) Quantitative LGE Mass and Regional Wall Motion Assessment. (C) Peak IRA BEW Wave Intensity vs LGE Mass. (D) BEW Correlation with Regional LV Recovery.