European Heart Journal (2007) **28**, 2313–2319
doi:10.1093/eurheartj/ehm306

Field triage to primary angioplasty combined with emergency department bypass reduces treatment delays and is associated with improved outcome

Steen Carstensen[1,*], Greg C.I. Nelson[1], Peter S. Hansen[1], Lewis Macken[2], Stephen Irons[3], Michael Flynn[3], Pramesh Kovoor[4], Soon Y. Soo Hoo[1], Michael R. Ward[1], and Helge H. Rasmussen[1]

[1]*Department of Cardiology, Royal North Shore Hospital, University of Sydney, Australia;* [2]*Emergency Department, Royal North Shore Hospital, University of Sydney, Australia;* [3]*Ambulance Service of New South Wales, Sydney, Australia; and* [4]*Department of Cardiology, Westmead Hospital, University of Sydney, Australia*

Received 26 September 2006; revised 1 June 2007; accepted 21 June 2007; online publish-ahead-of-print 1 August 2007

KEYWORDS
Myocardial infarction;
Treatment pathway;
Primary angioplasty;
Mortality

Aims We investigated the net benefit in the outcome of reducing treatment delay through field triage and emergency department (ED) bypass in patients with ST-elevation myocardial infarction (STEMI) treated with primary angioplasty.
Methods and results In a prospective registry study, consecutive patients with suspected STEMI were assigned to: (i) pre-hospital ECG and triage or (ii) ECG and triage at the closest ED, solely based on ambulance availability. Four district hospitals and one regional heart centre serviced the 890 000 population metropolitan area and primary angioplasty was the only reperfusion strategy employed. Baseline characteristics were similar in STEMI patients triaged in the field (108) and the EDs (193). Symptom onset to balloon times: 154 [inter-quartile range (IQR) 120–233) vs. 249 (IQR 184–405) min ($P < 0.001$) and peak creatine kinase in early presenters (<2 h): 1435 (95 %CI: 904–1966) U/L vs. 2320 (95% CI: 1881–2762) U/L ($P = 0.009$) were lower in field- than in ED-triaged patients. Mortality in the PCI treated were 1.1 and 8.2% [$P = 0.025$, RR 0.14 (95% CI: 0.01–1.08)] and overall mortality were 1.9 and 7.3% [$P = 0.046$, RR 0.26 (95% CI: 0.05–1.11)].
Conclusion Field-triage and ED bypass were feasible means of reducing treatment delay in patients with suspected STEMI and resulted in smaller infarct size in early presenters and a trend towards a reduction in mortality.

Introduction

It is generally accepted that a close relationship exists between ischaemia time (time from symptom onset to reperfusion), infarct size, and mortality in the early hours after coronary occlusion.[1] While this paradigm is supported by clinical data on fibrinolysis in ST-elevation myocardial infarction (STEMI),[2] data on primary percutaneous coronary intervention (PCI) are conflicting with studies showing no relation between ischaemia time and infarct size or mortality,[3–6] and others demonstrating a close relationship.[7–10] Nevertheless, the dogma that 'time is muscle' renders randomization of STEMI patients to excess treatment delays ethically problematic and the net benefit in outcome achieved from reducing treatment delays in primary PCI is not likely to be assessed in randomized controlled trials. We therefore utilized the partial introduction of a new, fast treatment pathway to primary PCI to obtain controlled data on the net benefit in the outcome associated with reducing treatment delay to primary PCI in unselected patients with suspected STEMI. The objective of this study was to test the hypothesis that fast-tracking STEMI patients from the field directly to the catheter laboratory, bypassing district and local emergency departments, is feasible and safe and that overall treatment delays and infarct size in early presenters are reduced significantly with this triage strategy compared with conventional triage.

Methods

Emergency Triage of Acute Myocardial Infarction registry

The ETAMI (Emergency Triage of Acute Myocardial Infarction) registry was prospectively collected to monitor the quality of care and outcome during the implementation of a new treatment pathway combining pre-hospital ECG recording and field triage of patients with suspected STEMI directly to the catheter laboratory in the Northern Sydney Area Health Service (NSAHS). The population base was 890 000 people, and the NSAHS covered an area of 1600 km^2 and included four district hospitals that referred STEMI

* Corresponding author: Department of Cardiology, Roskilde Hospital, University of Copenhagen, DK-4000 Roskilde, Denmark. Tel: +45 30502589; fax: +45 46362797.
E-mail address: sc@dadlnet.dk

patients to a regional heart centre (RHC) for primary PCI. The longest distance from within the area to the RHC was 36 km and the corresponding longest transport time was estimated to be 54 min. The RHC was a high volume centre with approximately 550 emergency angioplasties performed annually by four interventional cardiologists.

Treatment pathways

Throughout the study period from 1 April 2004 to 30 September 2005, one-third of the ambulances at stations in the NSAHS were equipped for field triage. All emergency calls for an ambulance were answered by nurses at the call centre of the Ambulance Service of New South Wales and the callers were taken through structured interviews enabling computer-based suggestions for diagnoses and priority. If acute myocardial infarction were suspected, a field triage capable ambulance would be sent to the scene and if such an ambulance were not available, an ambulance with standard equipment attended the patient. While the latter took the patient to the ED of the closest hospital for diagnosis and management, the field triage ambulances had 12-lead ECG recording and transmission capabilities (Lifepack 12, Medtronic, Minneapolis, MN, USA) and were staffed with specifically trained paramedic ambulance officers. ECGs were transmitted to a dedicated computer (Lifenet RS, Medtronic, Minneapolis, MN, USA) in the ED of the RHC where emergency physicians analysed the ECG, called the ambulance, and triaged the patient: directly to the catheter laboratory when ECG and presentation indicated a STEMI or to (re-)assessment at the ED of the closest hospital if this were not the case (*Figure 1*). The catheter laboratory was activated immediately when patients were triaged to primary PCI and upon arrival at the RHC patients were taken through the ED on the ambulance stretcher on their way to the catheterization laboratory. In case of loss of consciousness, haemodynamic or respiratory instability during transport the ED physician would quickly review the patient, re-confirm STEMI suspicion, and try to stabilize the condition before rushing the patient to the catheter laboratory. In the majority of uncomplicated cases, the patient went straight to the catheter laboratory where the interventional cardiologist reviewed the patient before proceeding with an emergency angiogram.

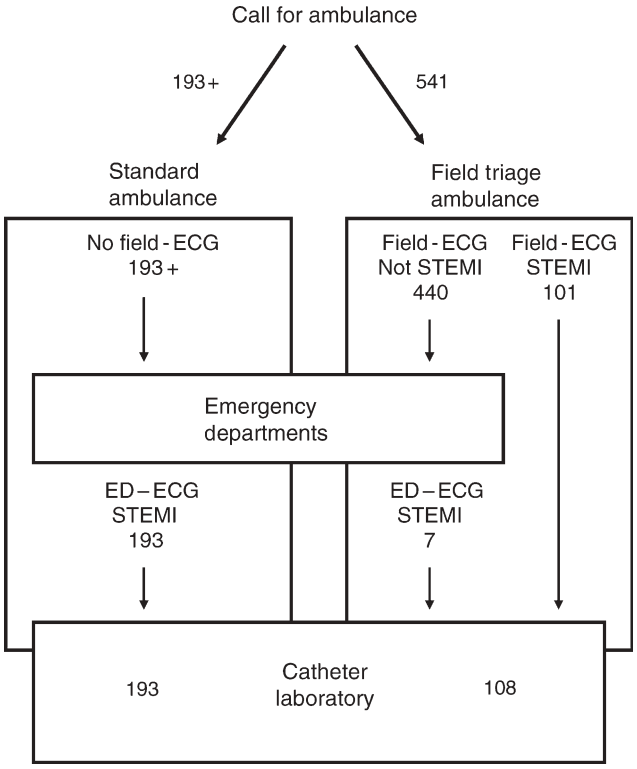


Figure 1 Diagram illustrating the two treatment pathways for patients with suspected acute myocardial infarction including the number of patients at each stage.

Study population

Patients brought in by ambulance to one of the five NSAHS hospitals, presenting with a clinical suspicion of acute myocardial infarction, symptom onset within the past 24 h, ECG indicating STEMI, and who were subsequently triaged to an emergency angiogram were included in the study. ECG criteria for STEMI were $\geq$0.1 mV of ST-elevation in more than two contiguous leads or presumed new left bundle branch block. Patients suffering cardiac arrest before the ambulance reached them were excluded from analysis. Clinical characteristics of the study population are listed in *Table 1*. The proportion of patients with hypertension was higher in the ED- than in the field-triaged group.

Treatment and monitoring

All patients received sublingual nitroglycerin and oral aspirin and had intravenous access established by ambulance officers. In addition, the ED-triaged patients were given 5000–10 000 IU of heparin intravenously and oral clopidogrel loading with 300 or 600 mg before transfer to the catheter laboratory. Angiography was performed according to standard procedure and included a ventriculogram usually recorded after PCI. Assessment of left ventricular ejection fraction and coronary stenoses was visual and the treatment strategy thereafter was at the discretion of the interventional cardiologist. In case of primary PCI, heparin up to a dose of 75–100 IU/kg was given intravenously, the use of glycoprotein IIIb/IIa receptor blockers was optional, stents were used if deemed appropriate and the policy was only to treat the infarct-related artery. Cardiac surgery was available on-site. Monitoring included blood samples for Troponin-T (TnT) and creatine kinase (CK) on admission, 8 and 24 h after the procedure and on clinical indication as well as serial 12-lead ECGs.

Outcome measures and statistical analyses

Statistical analyses were performed in a JMP IN statistical package version 5.1.2 (SAS Institute Inc.) on an intention to treat basis,

Table 1 Clinical characteristics in 301 STEMI patients undergoing emergency angiography

	ED triage ($n = 193$)	Field triage ($n = 108$)	*P*-value
Age, mean (SD)	67 (14.6)	64 (13.7)	0.12
Female gender, *n* (%)	65 (34)	28 (26)	0.17
Hypertension, *n* (%)	117 (60)	44 (41)	<0.001
Hypercholesterolaemia, *n* (%)	103 (53)	52 (48)	0.39
Diabetes, *n* (%)	33 (17)	18 (17)	0.93
Smoker, *n* (%)	52 (27)	33 (31)	0.51
Previous AMI, *n* (%)	44 (23)	16 (15)	0.10
Previous CABG, *n* (%)	14 (7)	10 (9)	0.54
Previous PCI, *n* (%)	20 (10)	14 (13)	0.50
Previous TIA/stroke, *n* (%)	15 (8)	5 (5)	0.30
Renal impairment, *n* (%)	12 (6)	5 (5)	0.57

SD, standard deviation; ED, emergency department; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention.

i.e. group assignment was based on ambulance allocation (standard or field-triage capable). Pre-defined outcome measures were ischaemia time, i.e. time elapsed from symptom onset to first balloon inflation, in-hospital mortality, and peak values of CK and TnT within 24 h following primary PCI. The markers of infarct size were analysed in the pre-specified subgroup of patients presenting to hospital within 2 h after symptom onset[1,2,5,11,12] in whom myocardial salvage is likely to depend on time to reperfusion.[13–15] Categorical data are presented as number with percentage in parentheses and continuous data as mean and 95% confidence intervals (95% CI) or as median value and inter-quartile range (IQR). Categorical data were analysed with a χ^2 test or Fisher's test whenever appropriate[16] and the relative risk of death with 95% CI is given but should be interpreted with caution as this method has not been validated for limited sample size datasets. Continuous data were analysed using Student's *t*-test or ANOVA if normally distributed and with the Mann–Whitney or Kruskall–Wallis test if this were not the case (delay and biomarker data). All tests were two-sided and a *P*-value of 0.05 was considered significant. No correction was made for multiple testing.

Results

Triage and transport

During the study period, 541 patients had an ECG recorded in the field with 534 (99%) interpretable ECGs being read by the ED physicians. ECG criteria for STEMI were fulfilled in 108 (20%) patients, 101 of which were triaged directly to the catheter laboratory while seven patients were re-assessed in an ED and subsequently triaged to the catheter laboratory via the same pathway as the 193 patients who were attended by standard equipped ambulances and had their first ECG recorded in the ED of a district hospital (120) or the ED of the RHC (73). The quality and location of ischaemic ECG abnormalities in the triage ECGs were well-matched between triage groups (*Table 2*). Cardiac arrest occurred at the scene[3] or during transport[2] in 5 (5%) patients in the field triage group, whereas 12 (6%) patients in the conventional triage group suffered a cardiac arrest before reaching the catheter laboratory, five at the scene, one during transport, and six in the ED ($P = 0.57$). They all reached the catheter laboratory alive. Four patients (4%) triaged in the field and seven ED-triaged patients (4%) were hypotensive on admission, did not respond to volume and pressor therapy within 30 min and were considered to be in a state of cardiogenic shock on admission ($P = 0.97$). Two of these patients died in the catheter laboratory before an angiogram could be performed (one in each triage group).

Emergency angiography and reperfusion therapy

The ECG changes, haemodynamics, and the angiographic results are presented in *Table 2*. These variables were obtained at different points in time in relation to symptom onset and the heart rate differed significantly between triage groups with higher values observed in the (delayed) ED-triaged patients. There were no significant between group differences in the number of diseased vessels or the location of coronary artery disease, however, the proportion of patients with an occluded infarct-related artery was significantly higher in the field- than in the ED-triaged group. The reperfusion strategy employed after the emergency angiogram and the angiographic outcome after primary PCI are listed in *Table 3*. A higher proportion of patients in the field-triage group underwent primary PCI and a trend was noted towards fewer undergoing CABG during the admission when compared with the ED-triaged patients. The use of glycoprotein IIb/IIIa receptor blockers was lower in the clopidogrel loaded ED-triaged patients and this difference was present and of similar magnitude in early and late presenters. Of the 108 patients in the field-triage group significant coronary artery disease was detected in 99 (92%) and 93 (86%) underwent primary PCI.

Table 2 ECG and angiographic findings by triage group in 301 STEMI patients undergoing emergency angiography (obtained at different points in time in relation to symptom onset)

	ED triage	Field triage	*P*-value
ECG abnormalities, triage ECG			
ST-elevation, *n* (%)	185 (96)	105 (97)	0.55
Anterior, *n* (%)	93 (48)	45 (42)	0.28
Left bundle branch block, *n* (%)	8 (4)	3 (3)	0.55
Heart rate, mean (SD)	77 (21.4)	72 (21.0)	0.07
Systolic blood pressure, mean (SD)	130 (27.2)	130 (27.9)	0.84
Angiographic results			
Normal or insignificant disease, *n* (%)	18 (9)	8 (7)	0.58
Single vessel disease, *n* (%)	72 (37)	38 (35)	0.72
Multi-vessel disease, *n* (%)	102 (53)	61 (56)	0.55
LVEF, mean (SD)	44 (12.0)	46 (11.5)	0.15
Infarct-related artery, occluded, *n* (%)	103 (53)	73 (68)	0.02
Infarct-related artery identified, *n* (%)	156 (81)	94 (87)	0.17
Left main artery, *n* (%)	2 (1)	1 (1)	0.88
Left anterior descending artery, *n* (%)	68 (44)	41 (44)	1.00
Left circumflex artery, *n* (%)	16 (10)	7 (7)	0.46
Right coronary artery, *n* (%)	69 (44)	42 (45)	0.95
Saphenous vein graft, *n* (%)	1 (1)	3 (3)	0.13

ED, emergency department; LVEF, left ventricular ejection fraction; SD, standard deviation.

Time to reperfusion

Table 4 shows the time delays in the two treatment pathways. The time from symptom onset to hospital arrival was similar, whereas all other delays were significantly longer for ED- compared with field-triaged patients. In the latter group, symptom onset to balloon time was below 3 h in 58 of 93 patients (62%), while the corresponding number for ED-triage was 32 of 145 (22%), $P < 0.001$. The median time from symptom onset to TIMI-3 flow in the infarct-related artery was 103 min longer for ED- than for field-triage. While there was no significant difference in symptom onset to balloon time between patients triaged in the EDs at the RHC or district hospitals [256 (IQR: 152–392) min vs. 247 (IQR: 199–418) min, $P = 0.36$] the door to balloon times at the RHC differed significantly and were

Table 3 Reperfusion therapy by triage group

	ED triage ($n = 193$)	Field triage ($n = 108$)	*P*-value
Primary PCI, *n* (%)	145 (75)	93 (86)	0.025
TIMI-3 pre-procedure, *n* (%)	15 (10)	8 (9)	0.66
TIMI-3 post-procedure, *n* (%)	132 (91)	89 (96)	0.18
No reflow phenomenon, *n* (%)	12 (8)	5 (5)	0.40
GP-IIb/IIIa receptor blocker, *n* (%)	90 (62)	74 (80)	0.005
Stenting, *n* (%)	126 (87)	84 (90)	0.43
Non-infarct artery PCI, *n* (%)	7 (5)	8 (9)	0.25
Bridge to CABG, *n* (%)	9 (6)	3 (3)	0.31
Emergency CABG, *n* (%)	1 (1)	1 (1)	0.68
CABG during admission, *n* (%)	18 (12)	4 (4)	0.08

ED, emergency department; TIMI-3: TIMI-flow grade 3 in the infarct-related coronary artery; CABG, coronary artery bypass grafting; PCI, percutaneous coronary intervention; GP, glycoprotein.

Table 4 Delay from symptom onset to reperfusion and components thereof by triage group

	ED triage ($n = 193$)	Field triage ($n = 108$)	*P*-value
Symptom onset to			
Door at first hospital[a]	110 (65–226)	110 (85–176)	0.32
First ECG[a]	130 (81–210)	75 (47–150)	<0.001
Door at RHC[a]	195 (130–330)	110 (85–197)	<0.001
First balloon inflation[a]	249 (184–405)	154 (120–233)	<0.001
TIMI-3 (initial occlusion)[a]	253 (186–361)	150 (122–238)	<0.001
Door (first hospital) to balloon[a]	116 (87–161)	39 (29–67)	<0.001
Door (RHC) to balloon[a]	50 (34–85)	34 (27–48)	<0.001
After hours presentation, *n* (%)[b]	121 (63)	63 (58)	0.46

ED, emergency department; RHC, Regional Heart Centre.
[a]Median (IQR).
[b]Normal working hours: 7:00 am to 5:59 pm Monday to Friday.

91 (IQR: 69–115) and 37 (IQR: 27–47) min in these groups, respectively, $P < 0.001$.

Infarct size

Peak levels of CK and TnT in patients who underwent primary PCI are presented in *Figure 2*. In early presenters, these differed significantly between ED- and field-triaged patients: peak CK 2320 (95% CI: 1881–2762) U/L vs. 1435 (95% CI: 904–1966) U/L; $P = 0.009$ and peak TnT 9.07 (95% CI: 6.75–11.40) vs. 4.32 (95% CI: 1.56–7.11); $P = 0.011$, whereas no between-group differences were found in late presenters, in the entire group undergoing primary PCI or in the entire study population.

Mortality

Sixteen patients (5.3%) died during hospitalization, two (1.9%) in the field-triaged, and 14 (7.3%) in the ED-triaged group; $P = 0.046$. The corresponding relative risk of in-hospital death was 0.26 (95% CI: 0.05–1.11) for field- compared with ED-triaged patients. For patients treated with primary PCI mortality was 1/93 (1.1%) and 11/145 (8.2%) in the two triage groups ($P = 0.025$) and the relative risk of death 0.14 (95% CI: 0.01–1.08). While the proportions of patients with cardiogenic shock were similar; 18 of 193 (9.3%) vs. 10 of 108 (9.3%), a trend was noted towards lower mortality for cardiogenic shock in the field- than in the ED-triaged group of two of 10 (20%) vs. 10 of 18 (56%); $P = 0.074$, relative risk of death 0.36 (95% CI: 0.10–1.33). In five (1.7%) patients, myocardial ruptures were documented: three free-wall ruptures (one by ventriculography and two by autopsy) and two ventricular septal defects (echocardiography). Surgery was attempted in three and one patient was discharged alive. Intra-aortic balloon pump therapy for cardiogenic shock was used in four of 10 patients (40%) in the field-triage and in three of 18 patients (17%) in the ED-triage group; $P = 0.180$.

There were no differences between ED- and field-triaged patients in rate of re-infarction: five of 193 (2.6%) vs. none in 108 (0%); cardiac arrest: 20 of 193 (10.4%) vs. 10 of 108 (9.3%); or stroke: four of 193 (2.1%) vs. none in 108 (0%).

Discussion

In the current study, a feasible and safe means of reducing treatment delay in patients with STEMI was described. When this approach was applied to an all-comer population with suspected STEMI, outcome was improved exactly as predicted by the reperfusion paradigm. The indication of an in-hospital mortality benefit associated with the fast treatment pathway underscores the benefits of very early reperfusion and the clinical relevance of the reperfusion paradigm in primary PCI.

Treatment delay

The introduction of pre-hospital ECG and field-triage combined with ED bypass reduced the median treatment delay 95 min primarily through a reduction in door-to-balloon time. We detected no indication of an excess risk or of inappropriate referrals with this strategy although the majority of patients presented in the on-call period as expected.[17,18] Short door-to-balloon times at the RHC have been achieved with inter-hospital transfers,[13–15] and the time gain associated with bypassing the ED after field-triage by a physician-staffed ambulance has also been reported upon,[19,20] but the reduction in treatment delay, the safety, and the clinical outcome of an approach where first physician contact is in the catheter laboratory has not been described previously. The treatment delays in our conventional treatment pathway are well within the range covered by clinical practice and trials documenting the advantage of transfer for primary PCI over on-site fibrinolysis,[13–15] but they are still

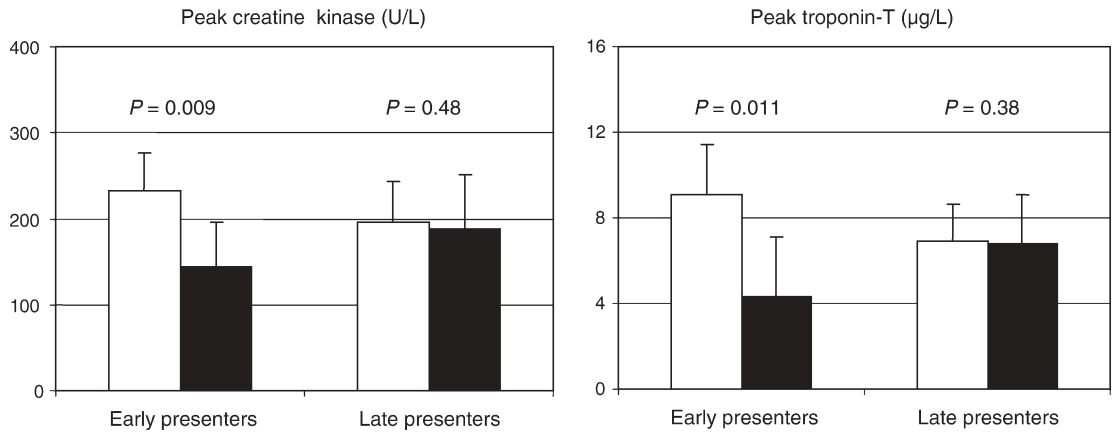


Figure 2 Mean values of peak creatine kinase and troponine-T in patients treated with primary PCI by time to hospital presentation; patients presenting within (early presenters) or beyond (late presenters) 120 min of symptom onset. Open bars, emergency department triage; filled bars, field triage. Error bars indicate upper 95% confidence limit.

suboptimal judging from current guidelines[21] and highlight the need for new and faster treatment pathways for STEMI patients who do not call an ambulance but self-present at an ED.

Outcomes

Gersh and Anderson[1] initially described a two-phased relationship between ischaemia time and outcome after coronary occlusion characterized by an early phase (up to 3 h) where outcome is highly dependent on ischaemia time and the potential for reducing infarct size and mortality with reperfusion therapy is large, and a second later phase where outcome is less-dependent on ischaemia time and other factors determine outcome, presumably because most of the acutely jeopardized myocardium has already infarcted. While the clinical relevance of this reperfusion paradigm is supported by the literature on fibrinolysis-mediated reperfusion,[2] the reports relating to primary PCI are conflicting.[3–10]

Although a rough measure of infarct size peak CK values correlate closely with MRI assessment of infarct size in patients undergoing primary PCI,[22] and TnT exhibits similar early release kinetics in this setting.[22,23] In the present study both variables indicate that infarct size was reduced in early, but not in late presenters when treatment delay was reduced, entirely in accord with the reperfusion paradigm and previous reports of greater extent of myocardial salvage, smaller infarct size, and better preservation of left ventricular ejection fraction in cohorts of patients with shorter treatment delays,[9,10] but in conflict with the study of Schömig *et al.*[3] who found no relation between ischaemia time and infarct size in cohort data from two randomized trials. The lack of control for the j-shaped nature of the time-to-presentation vs. mortality (and presumably infarct size) curve in the latter study may explain the diverging results in early presenters.[1]

The apparent in-hospital survival benefit associated with reducing treatment delays in the early hours after symptom onset also concur with the reperfusion paradigm and with several cohort studies demonstrating lower mortality in patients with shorter treatment delays.[1,4–8] That mortality was more closely related to door-to-balloon time than the total ischaemia time in some of these studies probably reflects that many patients were treated late and that the natural history of STEMI (the j-shaped time-to-presentation vs. mortality curve) was not controlled for.[1,4–6] The results of this study parallel previous observations of favourable results obtained with the field triage strategy in STEMI and suggesting a particular benefit in patients developing cardiogenic shock.[20] This is in keeping with reports indicating beneficial effects on new onset cardiogenic shock and mortality in patients treated very early (pre-hospital) with fibrinolysis[11] and with the observed benefit of early PCI-mediated reperfusion in patients developing cardiogenic shock.[24,25] Even with primary PCI, a substantial proportion of the potential mortality benefit appears to be lost in the early hours after symptom onset.

Study limitations

This was an observational study with the inherent possibility of bias in patient selection and management. A trend towards higher risk was apparent in the baseline characteristics of the ED-triage group (age, gender, prior myocardial infarction), but a significantly larger proportion of patients in the field-triage group had an occluded infarct-related artery at the time of angiography indicating higher risk. Whether the latter finding reflects selection, play of chance, intrinsic fibrinolysis, or effects of the upstream aspirin, clopidogrel, and heparin administered to the ED-triaged patients remains speculative, but this observation may offer an explanation to the lower rate of primary PCI and higher rate of CABG during admission in the ED-triage group. Data were collected at the RHC and while all patients in the field-triage group were accounted for any STEMI patients in the ED-triage group that died at the scene, in the ambulance, or in the local hospital before the RHC was notified were missed by our registry. Glycoprotein IIb/IIIa inhibitors were used more frequently in the field- than in the ED-triaged group (80 vs. 62%) and may reduce mortality in primary PCI.[26] However, this would account for $<0.2\%$ mortality difference between triage groups.[26] Furthermore, subgroups of early and late presenters received similar glycoprotein IIb/IIIa inhibitor therapy, yet the outcomes in terms of infarct size differed significantly. Therefore, the observed difference in

mortality is not explained by differences in glycoprotein IIb/IIIa inhibitor therapy.

We report our single centre experience with a limited number of patients appreciating the risk of type 1 error and that feasibility, safety, and benefits of new management strategies are better documented by multi-centre experiences and larger sample sizes.

Conclusions and clinical implications

Primary PCI can safely be delivered very fast to a substantial proportion of patients presenting with STEMI in a large metropolitan area. The benefit of fast-tracking these patients to the catheter laboratory appears to be a reduction in infarct size and mortality driven by a particular benefit in patients developing cardiogenic shock. The reperfusion paradigm is highly clinically relevant for primary PCI and a substantial proportion of the potential mortality benefit from this treatment modality appears to be lost with current practice. Every effort should be made to organize treatment pathways that can fast-track STEMI patients to a catheter laboratory.

Acknowledgements

Field-triage was set up in collaboration with Western Sydney Area Health Service and the Ambulance Service of New South Wales. We highly appreciate the dedicated effort from the ambulance officers, the emergency physicians, and the catheter laboratory staff. Medtronic Australasia Pvt Ltd kindly sponsored 40 portable defibrillator/monitor units and The North Shore Heart Research Foundation sponsored data collection for the ETAMI registry.

Conflict of interest: none declared.

References

1. Gersh BJ, Anderson JL. Thrombolysis and myocardial salvage. Results of clinical trials and the animal paradigm–paradoxic or predictable? *Circulation* 1993;**88**:296–306.
2. Boersma E, Maas AC, Deckers JW, Simoons ML. Early thrombolytic treatment in acute myocardial infarction: reappraisal of the golden hour. *Lancet* 1996;**348**:771–775.
3. Schömig A, Ndrepepa G, Mehilli J, Schwaiger M, Schuhlen H, Nekolla S, Pache J, Martinoff S, Bollwein H, Kastrati A. Therapy-dependent influence of time-to-treatment interval on myocardial salvage in patients with acute myocardial infarction treated with coronary artery stenting or thrombolysis. *Circulation* 2003;**108**:1084–1088.
4. Cannon CP, Gibson CM, Lambrew CT, Shoultz DA, Levy D, French WJ, Gore JM, Weaver WD, Rogers WJ, Tiefenbrunn AJ. Relationship of symptom-onset-to-balloon time and door-to-balloon time with mortality in patients undergoing angioplasty for acute myocardial infarction. *JAMA* 2000;**283**:2941–2947.
5. Zijlstra F, Patel A, Jones M, Grines CL, Ellis S, Garcia E, Grinfeld L, Gibbons RJ, Ribeiro EE, Ribichini F, Granger C, Akhras F, Weaver WD, Simes RJ. Clinical characteristics and outcome of patients with early (<2 h), intermediate (2–4 h) and late (>4 h) presentation treated by primary coronary angioplasty or thrombolytic therapy for acute myocardial infarction. *Eur Heart J* 2002;**23**:550–557.
6. Berger PB, Ellis SG, Holmes DR Jr, Granger CB, Criger DA, Betriu A, Topol EJ, Califf RM. Relationship between delay in performing direct coronary angioplasty and early clinical outcome in patients with acute myocardial infarction: results from the global use of strategies to open occluded arteries in Acute Coronary Syndromes (GUSTO-IIb) trial. *Circulation* 1999;**100**:14–20.
7. De Luca G, Suryapranata H, Ottervanger JP, Antman EM. Time delay to treatment and mortality in primary angioplasty for acute myocardial infarction: every minute of delay counts. *Circulation* 2004;**109**:1223–1225.
8. Brodie BR, Stuckey TD, Wall TC, Kissling G, Hansen CJ, Muncy DB, Weintraub RA, Kelly TA. Importance of time to reperfusion for 30-day and late survival and recovery of left ventricular function after primary angioplasty for acute myocardial infarction. *J Am Coll Cardiol* 1998;**32**:1312–1319.
9. Milavetz JJ, Giebel DW, Christian TF, Schwartz RS, Holmes DR Jr, Gibbons RJ. Time to therapy and salvage in myocardial infarction. *J Am Coll Cardiol* 1998;**31**:1246–1251.
10. Liem AL, van't Hof AW, Hoorntje JC, de Boer MJ, Suryapranata H, Zijlstra F. Influence of treatment delay on infarct size and clinical outcome in patients with acute myocardial infarction treated with primary angioplasty. *J Am Coll Cardiol* 1998;**32**:629–633.
11. Steg PG, Bonnefoy E, Chabaud S, Lapostolle F, Dubien PY, Cristofini P, Leizorovicz A, Touboul P. Impact of time to treatment on mortality after prehospital fibrinolysis or primary angioplasty: data from the CAPTIM randomized clinical trial. *Circulation* 2003;**108**:2851–2856.
12. Boersma E. The Primary Coronary Angioplasty vs. Thrombolysis Group. Does time matter? A pooled analysis of randomized clinical trials comparing primary percutaneous coronary intervention and in-hospital fibrinolysis in acute myocardial infarction patients. *Eur Heart J* 2006;**27**:779–788.
13. Widimsky P, Groch L, Zelizko M, Aschermann M, Bednar F, Suryapranata H. Multicentre randomized trial comparing transport to primary angioplasty vs. immediate thrombolysis vs. combined strategy for patients with acute myocardial infarction presenting to a community hospital without a catheterization laboratory. The PRAGUE study. *Eur Heart J* 2000;**21**:823–831.
14. Widimsky P, Budesinsky T, Vorac D, Groch L, Zelizko M, Aschermann M, Branny M, St'asek J, Formanek P. Long distance transport for primary angioplasty vs. immediate thrombolysis in acute myocardial infarction. Final results of the randomized national multicentre trial PRAGUE-2. *Eur Heart J* 2003;**24**:94–104.
15. Andersen HR, Nielsen TT, Rasmussen K, Thuesen L, Kelbaek H, Thayssen P, Abildgaard U, Pedersen F, Madsen JK, Grande P, Villadsen AB, Krusell LR, Haghfelt T, Lomholt P, Husted SE, Vigholt E, Kjaergard HK, Mortensen LS. A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction. *N Engl J Med* 2003;**349**:733–742.
16. Campbell I. Chi-squared and Fisher-Irwin tests of two-by-two tables with small sample recommendations. *Stat Med* 2007; Published online ahead of print 21 February.
17. Koyama Y, Hansen PS, Hanratty CG, Nelson GI, Rasmussen HH. Prevalence of coronary occlusion and outcome of an immediate invasive strategy in suspected acute myocardial infarction with and without ST-segment elevation. *Am J Cardiol* 2002;**90**:579–578.
18. Magid DJ, Wang Y, Herrin J, McNamara RL, Bradley EH, Curtis JP, Pollack CV Jr, French WJ, Blaney ME, Krumholz HM. Relationship between time of day, day of week, timeliness of reperfusion, and in-hospital mortality for patients with acute ST-segment elevation myocardial infarction. *JAMA* 2005;**294**:803–812.
19. Terkelsen CJ, Lassen JF, Norgaard BL, Gerdes JC, Poulsen SH, Bendix K, Ankersen JP, Gotzsche LB, Romer FK, Nielsen TT, Andersen HR. Reduction of treatment delay in patients with ST-elevation myocardial infarction: impact of pre-hospital diagnosis and direct referral to primary percutaneous coronary intervention. *Eur Heart J* 2005;**26**:770–777.
20. Ortolani P, Marzocchi A, Marrozzini C, Palmerini T, Saia F, Serantoni C, Aquilina M, Silenzi S, Baldazzi F, Grosseto D, Taglieri N, Cooke RM, Bacchi-Reggiani ML, Branzi A. Clinical impact of direct referral to primary percutaneous coronary intervention following pre-hospital diagnosis of ST-elevation myocardial infarction. *Eur Heart J* 2006;**27**:1550–1557.
21. Van de Werf F, Ardissino D, Betriu A, Cokkinos DV, Falk E, Fox KA, Julian D, Lengyel M, Neumann FJ, Ruzyllo W, Thygesen C, Underwood SR, Vahanian A, Verheugt FW, Wijns W. Management of acute myocardial infarction in patients presenting with ST-segment elevation. The Task Force on the Management of Acute Myocardial Infarction of the European Society of Cardiology. *Eur Heart J* 2003;**24**:28–66.
22. Haase J, Bayar R, Hackenbroch M, Storger H, Hofmann M, Schwarz CE, Reinemer H, Schwarz F, Ruef J, Sommer T. Relationship between size of myocardial infarctions assessed by delayed contrast-enhanced MRI after primary PCI, biochemical markers, and time to intervention. *J Interv Cardiol* 2004;**17**:367–373.
23. Gibbons RJ, Valeti US, Araoz PA, Jaffe AS. The quantification of infarct size. *J Am Coll Cardiol* 2004;**44**:1533–1542.
24. Webb JG, Lowe AM, Sanborn TA, White HD, Sleeper LA, Carere RG, Buller CE, Wong SC, Boland J, Dzavik V, Porway M, Pate G, Bergman G, Hochman JS. Percutaneous coronary intervention for cardiogenic shock in the SHOCK trial. *J Am Coll Cardiol* 2003;**42**:1380–1386.

25. Brodie BR, Stuckey TD, Muncy DB, Hansen CJ, Wall TC, Pulsipher M, Gupta N. Importance of time-to-reperfusion in patients with acute myocardial infarction with and without cardiogenic shock treated with primary percutaneous coronary intervention. *Am Heart J* 2003;**145**: 708–715.

26. De Luca G, Suryapranata H, Stone GW, Antoniucci D, Tcheng JE, Neumann FJ, Van de Werf F, Antman EM, Topol EJ. Abciximab as adjunctive therapy to reperfusion in acute ST-segment elevation myocardial infarction: a meta-analysis of randomized trials. *JAMA* 2005;**293**: 1759–1765.

Clinical vignette

doi:10.1093/eurheartj/ehm119
Online publish-ahead-of-print 7 May 2007

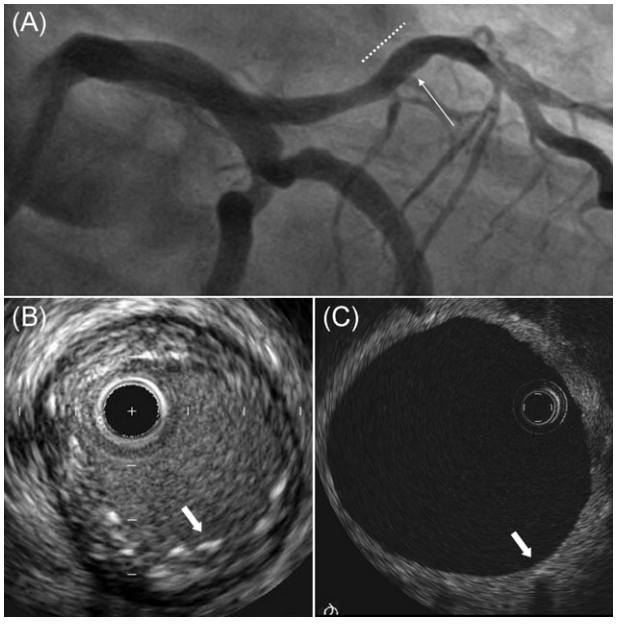
Coronary bioabsorbable magnesium stent: 15-month intravascular ultrasound and optical coherence tomography findings

Peter Barlis, Jun Tanigawa, and Carlo Di Mario*

Department of Cardiology and the National Heart and Lung Institute, Royal Brompton Hospital, Imperial College, Sydney Street, London SW3 6NP, UK

* Corresponding author. Tel: +44 20 7352 8121; fax: +44 20 7351 8473. *E-mail address*: c.dimario@rbht.nhs.uk

A 65-year-old man underwent elective percutaneous coronary intervention for a stenosis in the proximal left anterior descending artery (LAD). As part of the PROGRESS study (designed as a first-in-man coronary study in 65 patients in seven European centres), a 3.5 × 15 mm magnesium-based bioabsorbable stent (Lekton Magic Stent, Biotronik, Bulach, Switzerland) was implanted achieving an excellent angiographic result. Fifteen months following the initial intervention, an exercise treadmill test to evaluate exertional dyspnoea was positive. Angiography revealed a new and separate lesion in the mid-LAD/diagonal artery that was treated successfully with two sirolimus-eluting stents (Cypher Select, Cordis, J&J) in a culotte fashion. In the previously treated proximal segment, the vessel lumen was patent with no signs of narrowing or edge effect (Panel A). Imaging using intravascular ultrasound (Panel B) and optical coherence tomography (Panel C) showed the absence of circumferential stent struts with shadowing. Small, scattered, and circumscribed zones of high intensity (arrows) indicated the previous stent strut position (Panels B and C). All struts were covered by a thin neointimal layer with a thickness between 80 and 140 μm (Panel C).



The bioabsorbable stent is constructed from a magnesium alloy containing also zirconium (<5%), yttrium (<5%), and rare earth elements (<5%). The struts disappear over time, but their position can still be identified because of the fact that the strut material is absorbed and the space filled in by calcium apatite complex, accompanied by a phosphorous compound. These stents are compatible with cardiac magnetic resonance imaging and multi-slice computed tomography and can be used as vehicles for possible drug and gene delivery. Such a novel technology may prove useful in negating some of the untoward complications of current permanent metallic stents, namely, stent thrombosis and the need for prolonged dual anti-platelet therapy.

Panel A. Coronary angiography in the right caudal view showing the proximal left anterior descending artery 15 months after implantation of a magnesium bioabsorbable stent. The stented area is indicated by the dashed line. The arrow indicates the region imaged by intravascular ultrasound and optical coherence tomography.

Panels B and C. Intravascular ultrasound and optical coherence tomography imaging of the previously stented segment at 15 months of follow-up. The vessel wall is without stent struts after absorption, but small, well-defined zones of high intensity (arrowheads) are scattered indicating the previous stent strut position. There is a thin, concentric layer of neointima with thickness between 80 and 140 μm (Panel C).

See online supplementary material available at *European Heart Journal* online for a colour version of this figure.