Long-term benefit of primary angioplasty compared to thrombolytic therapy for acute myocardial infarction

The treatment of myocardial infarction has evolved considerably over the past decades. Reported mortality rates have fallen, due to a variety of factors, including earlier diagnosis and treatment of the acute event, improved management of complications such as recurrent ischaemia and heart failure, and widespread availability of pharmacological therapies such as aspirin, beta-blockers and ACE inhibitors[1]. Most attention, however, has been focused on therapies that restore antegrade coronary blood flow in the culprit vessel of the patient with evolving acute myocardial infarction. The two methods to achieve this goal are intravenous thrombolytic therapy most often followed by a conservative approach of watchful waiting, or immediate coronary angiography followed by primary angioplasty if appropriate. An overview of short term results of 10 randomized comparisons[2] between these two approaches has shown that compared to thrombolysis, primary angioplasty results in a lower mortality (4·4% vs 6·5%, relative risk 0·66, 95% confidence intervals 0·46–0·94), translating into an absolute benefit of two lives saved per 100 patients treated with angioplasty rather than thrombolysis. The relative reduction in death or non-fatal reinfarction after angioplasty compared to thrombolysis is even more striking (7·2% vs 11·9%, relative risk 0·58, 95% confidence intervals 0·44–0·76). With respect to safety, stroke was reduced from 2·0% with thrombolysis to 0·7% with angioplasty (relative risk 0·27, 95% confidence intervals 0·15–0·52). The combined incidence of death and non-fatal reinfarction was lower for early events, within the first 30 days, with a relative risk of 0·13 (95% confidence interval 0·05–0·37), as well as for late events, after 30 days, with a relative risk of 0·62 (95% confidence interval 0·43–0·91). The rates of readmissions for heart failure and ischaemia were lower among patients in the angioplasty group than among patients in the streptokinase group. Total medical charges per patient were lower in the angioplasty group (U.S.$ 16,090) than in the streptokinase group (U.S.$ 16,813).

That costs are not higher, and in fact even may be lower, after primary angioplasty compared to thrombolysis, has now been confirmed in many settings[4–6]. The impact of stenting is in particular pertinent to the cost issue[7]. By reducing the rate of repeat procedures, stent-eligible patients benefit from stenting compared to balloon angioplasty as a consequence of a lower restenosis rate, probably without an effect on major clinical events such as death and reinfarction[8]. Further research on the role of stenting will certainly be necessary. Given the superior safety and efficacy of primary angioplasty, this treatment is now preferred when logistics allow this approach. As has been shown for angioplasty for stable and unstable angina[9], it is likely that the results of primary angioplasty will be, in part, dependent on the setting in which it is performed, and therefore the results from various hospitals may differ considerably[2,10]. Establishing and maintaining a proficient primary angioplasty programme takes considerable institutional will and effort[11,12]. Many patients with acute myocardial infarction are admitted to hospitals without angioplasty facilities, necessitating additional transportation to an interventional catheterization laboratory. Although this can be organized safely[13] the time delay may offset some of the benefits. Although time to therapy may be less important for clinical outcome in angioplasty compared to
thrombolytic patients\cite{14}, shortening the time delay can be beneficial, in particular in the first 1 or 2 h\cite{15}. The main issues pertinent to the quality of care should be monitored continuously. In addition to performance of the angioplasty procedure by experienced operators, other aspects of care are at least as important, such as communication with the pre-hospital providers of care for patients with acute myocardial infarction, organization of emergency room and coronary care unit, concomitant pharmacological therapy, and after the procedure, risk stratification, rehabilitation and secondary prevention.

Pre-hospital diagnosis of acute myocardial infarction by 12-lead electrocardiography allows time for preparation before arrival of the patient. As shown in Fig. 1, patients transferred from another hospital have a shorter admission to first balloon inflation time, compared to patients with infarct diagnosis in the emergency room of our hospital, due to the ability to make preparations before arrival and to avoid the emergency room and coronary care unit, going directly to the catheterization laboratory. We recently performed a pilot study on computer-assisted infarct diagnosis by 12-lead electrocardiography in the ambulances in our area. This resulted in an important improvement in the delivery of reperfusion therapy (Fig. 1). Pre-hospital diagnosis offers an additional advantage: the possibility to consider pharmacological pre-treatment on the way to the catheterization laboratory\cite{16-18}. This is an important new opportunity to further improve clinical outcome in patients with acute myocardial infarction.

**Figure 1** Time delays in the treatment of patients with acute myocardial infarction.

(A) Time delays in 894 patients with acute myocardial infarction, who were presented to the emergency room of our hospital.
A1=symptom-onset to emergency room.
A2=emergency room to 1. balloon inflation.

(B) Time delays in 402 patients with acute myocardial infarction, who were presented to the emergency room of another hospital and were referred for primary angioplasty.
B1=symptom-onset to emergency room.
B2=emergency room of another hospital to catheterization laboratory of our hospital.
B3=admission in our hospital to 1. balloon inflation.

(C) Time delays in 106 patients in the pilot phase of pre-hospital infarct angioplasty triage with computer-assisted myocardial infarct diagnosis by 12-lead electrocardiograms made in the ambulance.
C1=symptom-onset to arrival of the ambulance equipped with 12-lead electrocardiography.
C2=pre-hospital electrocardiography and transportation directly to the catheterization laboratory in our hospital.
C3=arrival in our hospital to 1. balloon inflation.

<table>
<thead>
<tr>
<th>Symptom onset</th>
<th>A1 138 ± 69</th>
<th>B1 108 ± 70</th>
<th>C1 95 ± 52</th>
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<tr>
<td></td>
<td>A2 67 ± 28</td>
<td>B2 73 ± 27</td>
<td>C2 45 ± 49</td>
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<td>1. balloon inflation</td>
<td>B3 39 ± 27</td>
<td>C3 32 ± 22</td>
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<td>Total ischaemic time</td>
<td>205 ± 74</td>
<td>220 ± 82</td>
<td>172 ± 67</td>
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Time delays in minutes (mean ± SD)
References


