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The impact of prehospital thrombolytic treatment on re-infarction rates

To the editor: We congratulate Horne and colleagues2 for their paper drawing attention to the high risk of re-infarction for patients receiving prehospital thrombolytic therapy for ST-segment elevation myocardial infarction. We disagree with their main conclusion that the major cause of this is underdosing with heparin and delay in commencing a heparin infusion. We suspect that the main factor behind the high rate of re-infarction is the very effectiveness of early prehospital thrombolytic therapy in preserving jeopardised myocardium. As the authors acknowledge in their discussion, higher rates of initial reperfusion inevitably provide a greater opportunity for re-infarction. The rate of re-infarction for thrombolytic therapy delivered late is likely to approach zero.

We suggest that their data should be viewed as augmenting the already very strong evidence from trials such as CARESS-in-AMI1 and CAPITAL-AMI1 that patients treated with thrombolytic therapy should be taken to an angioplasty centre and undergo prompt angiography with a view to angioplasty. The precise timing of this intervention remains controversial; immediate (so-called facilitated) angioplasty appears to result in adverse effects but early intervention is highly effective. The lytic effects of modern thrombolytic drugs are brief, platelet activation can be minimised by GpIIb/IIIa inhibitor use and bleeding complications by radial artery access. Such an approach yields results that are probably equivalent to primary percutaneous coronary intervention as evidenced by the CAPTIM1 and GRACIA 22 studies and the FAST–MI2 registry. The STREAM trial will compare the two strategies directly in a randomised study. We now know how to combine lytic therapy with angioplasty successively. This knowledge should be applied without delay to the majority of patients with ST-segment elevation myocardial infarction in the UK who are currently receiving thrombolytic therapy.

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The author’s reply: Drs Townend and Routledge make the point that a blocked coronary artery needs to have been re-opened before it can re-occlude, and that the excess re-infarction rate seen with prehospital thrombolytic treatment is simply a marker of more effective (and earlier) restoration of patency. There are data in our study7 that support this view—re-infarction rates are highest in those with the shortest delay from onset of symptoms to start of treatment, whether that treatment is given before or after arrival at hospital.

Among the reasons we put forward in support of our hypothesis that inadequate anticoagulation also may be responsible were:

▸ The association between increased re-infarction and prolonged journey time to hospital after prehospital administration of thrombolytic drugs, as a marker of delay to heparin infusion.

▸ The lower rate of bleeding within 24 h of prehospital treatment compared with in-hospital treatment, as a marker of less intensive anticoagulation in the former group.

▸ The association between increased re-infarction and greater body weight, considering that early bolus dose heparin was not “weight-adjusted.”

Short of a randomised trial of immediate versus delayed heparin following thrombolytic treatment, it is impossible to prove our suggestion regarding the role of anticoagulation. However, we continue to advocate optimal and prompt adjunctive antiplatelet and anticoagulant therapy in patients receiving prehospital thrombolytic treatment before, as Townend and Routledge suggest, they are transported to “heart attack centres” for assessment for immediate (rescue), or later, percutaneous intervention. Such a “pharmacoinvasive” approach is already successfully applied in some centres.3

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Management of heart failure with preserved ejection fraction: back to the drawing board?

To the editor: We read with interest the editorial on heart failure with preserved ejection fraction by Yip and colleagues.1 As they state, there remains a great deal of uncertainty and debate regarding its management. This is likely to be due to the fact that there are no agreed universal criteria for the diagnosis of this condition. The current guidelines of the American College of Cardiology/American Heart Association2–4 reflect the presence of symptoms and signs in keeping with a diagnosis of heart failure with evidence of a preserved ejection fraction.2 However, the guidelines of the European Society of Cardiology stipulate an additional objective demonstration of abnormal diastolic function.3 The two large randomised trials of pharmacological therapy referred to by the authors recruited patients on the former criteria.4 The lack of universally accepted diagnostic criteria casts doubt over the selection of appropriate patients into studies to date and, therefore, the validity of their findings. This is evidenced by the findings of Persson et al5 who retrospectively studied 312 of the patients enrolled in the Candesartan in Heart Failure–Assessment of Reduction in Mortality and Morbidity (CHARM)–Preserved trial and noted that 53% of this subset did not have demonstrable diastolic dysfunction on echocardiography. These patients therefore do not meet the criteria for heart failure with preserved ejection fraction under the current European