Mobilization of bone marrow-derived stem cells after myocardial infarction and left ventricular function: simply effects of optimized drug treatment?: reply

We thank Drs Thum and Bauersachs for their interest in our article.\(^1\) They correctly note that although we found transient mobilization of bone marrow-derived stem cells (BMSCs) after acute myocardial infarction (AMI) in man, Nygren et al.\(^2\) failed to find mobilization of BMSCs after AMI in mice. Interestingly, two previous studies in the ‘human’ model have confirmed our observation of a transient increase in CD34\(^+\) cell count following AMI.\(^3,4\) This discrepancy between clinical and experimental data might well be explained by medical treatment given to patients; in particular, statins have been shown to mobilize BMSCs.\(^5\) Accordingly, in our study, statins were independent predictors of BMSCs release after MI at the multi-variable analysis. However, the model could explain only 27% of variability, thus suggesting that other, perhaps genetically determined, factors are likely to play an important role in determining the degree of BMSCs mobilization after AMI in man. In addition, among patients with AMI on statins during hospitalization, CD34\(^+\) cell count was higher than that observed in patients with stable angina and in patients with AMI reassessed at follow-up (all on statins) (8.80±7.26 vs. 3.80±2.12 cells/L; \(P<0.001\)).

On the basis of our observation that CD34\(^+\) cell count was an independent predictor of global and regional improvement of LV function at 1 year after AMI, Drs Thum and Bauersachs propose that CD34\(^+\) cells could be used as a diagnostic tool for the identification of patients at higher risk of developing heart failure after AMI. We believe that this conclusion is still premature as the results of our study need to be confirmed in much larger populations. The main goals of our study were to confirm BMSCs mobilization after AMI and to establish whether mobilization was associated to changes in left ventricular function after AMI. Our study shows that ‘good mobilizers’ of BMSCs after AMI exhibit a better evolution of post-AMI dysfunction than that observed in ‘poor mobilizers’. These findings represent a strong stimulus to investigate pharmacological strategies which are able to transform ‘poor mobilizers’ into ‘good mobilizers’. Indeed, this approach might turn out to be more effective at reducing the risk of developing heart failure after AMI.