fibrinolytic therapy, so there is a fine balance between allowing sufficient time for fibrinolytic therapy to work and not delaying to the extent that there is little myocardium to salvage. b) Although it seems logical that a 60-min ECG protocol would result in inclusion of lower-risk patients than would a 90-min (or 120-min) ECG protocol, there is evidence that a 60-min ECG identifies high-risk patients just as well as an ECG performed at 180 min (3). The MERLIN trial results support this. The 60-min ECG identified a population at significantly higher risk of adverse events than did previous studies (4). In addition, over 40% of patents in the rescue arm had Thrombolysis In Myocardial Infarction (TIMI) flow grade 3 in the infarct-related vessel, and therefore *may* not have been taken to the catheter laboratory on the basis of a 90-min ECG. One might expect these patients to be a low-risk subgroup, but we observed a 36% incidence of the composite secondary end point at 30 days. This high event rate cannot be attributed to the procedure itself, because less than one-third of patients with TIMI flow grade 3 had an immediate angioplasty, and all of these were technically successful. c) Relying on a later ECG for the diagnosis of failed fibrinolysis would result in further delays in the rescue angioplasty process. In particular, we were concerned that transferred patients could be disadvantaged by a longer period between the onset of fibrinolytic therapy and the diagnosis of failed fibrinolysis, given the inevitable further delay associated with transfer. In the end, 40% of patients in the rescue arm were transferred, and the time delay was almost 2 h (105 ± 32 min). d) Neither a 60-min nor 90-min ECG addresses the problem of using paired static ECGs to make a diagnosis of failed fibrinolysis. This method provides no information on whether persistent ST-segment elevation on the second ECG is the result of failure to reperfuse or reperfusion injury, nor does it provide any information on subsequent stability of the ST segments. Nonetheless, it remains the most simple and practical noninvasive test currently available.

Third, the investigators have missed our remarks on the presence or absence of ongoing chest pain. We agree that chest pain should be evaluated following administration of fibrinolytic therapy, and it is possible that this could reduce the number of patients who are exposed unnecessarily to a rescue procedure. However, our large experience of infarct angioplasty, before and including the MERLIN trial, is that the presence of chest pain is not a reliable predictor of persistent arterial occlusion and that the absence of chest pain is a very poor indicator of arterial patency. This is consistent with the observations of other investigators (5). It is unclear whether the assessment of symptoms is more useful for predicting perfusion at the *cellular* level. We recorded the presence or absence of chest pain in all MERLIN trial patients at the time of randomization. In the article discussion, we report that, in the rescue arm, 53% of those who were pain free at randomization had inadequate antegrade flow in the infarct-related vessel and 38% of those in pain at randomization had normal antegrade flow in the infarct-related vessel.

Furthermore, 43% of rescue-arm patients who were pain free immediately before the coronary angiogram had inadequate antegrade flow in the infarct-related vessel, and 26% of those in pain immediately before the coronary angiogram had normal antegrade flow in the infarct-related vessel. Therefore, reliance on the presence or absence of ongoing symptoms would have resulted in a large number of incorrect diagnoses and, in particular, a large number of incorrect diagnoses of successful fibrinolysis.

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# Influence of Pretreatment Systolic Blood Pressure on Benefit From Carvedilol in Severe Chronic Heart Failure Patients

According to the important report by Rouleau et al. (1) and the thoughtful accompanying editorial by Cohn (2), one would expect heart-failure patients with the lowest systolic blood pressure, and who are at the highest risk because of the severity of heart failure, to enjoy a higher relative benefit from carvedilol compared to patients with higher systolic blood pressure. Would the investigators speculate on why the relative benefit was similar across all pretreatment systolic blood pressure levels?

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