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 patients 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 ECG. 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.

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?

REFERENCES
