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pressure monitoring and echocardiographic assessment of hemodynamically unstable patients, could critical care clinicians be persuaded to choose the least invasive technique. With increasing numbers of echocardiographers (cardiologists or other specialists with adequate training in echocardiography) involved in critical care management, this choice could start a new trend, eventually leading to reduced costs. Furthermore, as smaller and less expensive ultrasound units in conjunction with smaller probes become available, continuous monitoring (e.g., of the short axis of the left and right ventricles) should be possible. Thorough, lengthy training in echocardiography for all critical care providers would further improve critical patient care.

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Reply

We thank Poelaert for his interest in our report. We agree that transesophageal echocardiography frequently provides important information that is not evident from pulmonary artery catheterization. We are interested and encouraged to learn that his study demonstrated a similar management change rate after transesophageal echocardiography in patients with pulmonary artery catheters.

We also agree with his suggestion that transesophageal echocardiography may obviate the need for pulmonary artery catheterization in a significant number of patients with hypotension. His recommendation that hemodynamically unstable patients receive the less invasive procedure before right heart catheterization is reasonable if the transesophageal echocardiogram is readily available. However, demonstrating conclusively that this strategy is optimal may prove difficult. To prove that there is no clinically significant difference in outcome with the noninvasive strategy would require a very large randomized trial.

We agree that cost of therapy may prove the deciding factor. In today's cost-conscious market, cost-effectiveness will have to be demonstrated before either a one-time transesophageal echocardiogram or continuous monitoring can be widely recommended.

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Q Wave and Non-Q Wave Myocardial Infarction After Thrombolysis

Matetzky et al. (1) recently reported the results of a study of 150 patients with acute myocardial infarction who received thrombolytic therapy. The authors found that 80% of patients had a Q wave and 20% a non-Q wave infarction on the 24-h electrocardiogram (ECG); no significant differences were noted between these groups with regard to either in-hospital clinical course or long-term prognosis. In contrast, predischarge ECG analysis revealed that 72% of patients had a Q wave and 28% a non-Q wave infarction. This predischarge ECG stratification was a more useful prognostic descriptor in that a trend toward lower 2-year mortality was seen among the non-Q wave group, despite a higher incidence of reinfarction and revascularization during this time period.

The differences in prognostic information conveyed by a predischarge rather than a 24-h ECG in this study appears to relate to a "crossover" from one group to the other, with the disappearance of pathologic Q waves in 18 patients and the subsequent development of Q waves in 7 patients during the hospital period. Surprisingly, the authors do not comment on a previously published study (2) that also describes the evolution and prognostic importance of Q waves after thrombolytic therapy. In contrast, this latter study concluded that the development of Q waves beyond the 24-h window after thrombolytic therapy to the time of hospital discharge was infrequent (1.5% of 201 patients). Further, the early and 1-year prognosis among the non-Q and Q wave groups was similar (2).

Clearly, the timing of ECG stratification after thrombolytic therapy is an important factor in the interpretation of the prognostic utility of Q waves versus non-Q waves. Indeed, it is difficult to reconcile the apparent differences in Q wave evolution and subsequent prognosis after thrombolysis seen in the few contemporary published reports. The incidence of non-Q waves ranges from as low as 13% to 15% (2,3) to as high as 43% (4,5), with a few of the large international thrombolytic trials suggesting that the occurrence of non-Q wave myocardial infarction after thrombolysis is between 20% and 30% (6–8). Even the subsequent prognosis of non-Q waves appears to vary dramatically. Aguirre et al. (8) described a trend toward a higher 1-year reinfarction rate but a similar mortality rate among the non-Q wave group compared with the Q wave group in a secondary analysis of the Thrombolysis in Myocardial Infarction (TIMI) II trial. Tajer et al. (6) described a significantly higher 6-month reinfarction and mortality rates among non-Q wave myocardial infarction hospital survivors in the Tissue Plasminogen Activator Versus Streptokinase Trial (TPASK). Barbagelata et al. (7) described a significantly lower 30-day and 1-year mortality rate among the non-Q wave group in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Arteries (GUSTO) trial.

I would appreciate some comment by Matetzky et al. on these issues, particularly because four of the coauthors of this recent publication were also coauthors of the earlier and apparently conflicting analysis (2) of postthrombolytic Q wave evolution and prognosis, and they failed to mention this latter study in their current discussion and conclusions regarding the timing and value of Q wave/non-Q wave dichotomization in the thrombolytic era.

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